THE ARCHAEOLOGY OF MIND

Neuroevolutionary Origins of Human Emotion

JAAK PANKSEPP AND LUCY BIVEN

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A NORTON PROFESSIONAL BOOK

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Neuroevolutionary Origins of Human Emotions

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Lucy Biven

Foreword by Daniel J. Siegel

W. W. NORTON & COMPANY

New York • London

Dedicated to Tiina Alexandra Panksepp (1975–1991)

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Preface and Acknowledgments

ALL OF US GET ANGRY at times, especially when our interests are ignored or thwarted. Has traditional brain science told us how this emotion is created? Not yet. We all get lonely and sad at times. Has modern neuroscience sought to clarify those aspects of our nature? We have barely begun to talk about such things, even though great progress has been made in some quarters. Most of us get great joy from interacting playfully with others; some do not, especially if they are depressed. Neuroscience has remained largely silent about the nature of joy, while psychology has seen a revolution in the study and discussion of its cognitive derivative, happiness, with few insights into the neural nature of joy.

Just like the many other emotional powers of our minds, all of which emerge from the functions of the brain, traditional neuroscience has had relatively little to tell us about how the intense emotional feelings that we call *affects* can arise from brain activities. This is because feelings are subjectively experienced, and some say the traditional third-person measurements of science (i.e., external observation of phenomena) cannot deal effectively with first-person experiences. We disagree, to the extent that other mammals have evolutionarily related brain systems. Modern

neuroscience is well poised to finally clarify the ways that the mammalian brain generates affective valuations of world events in the form of nonverbal feeling states—or the passions of the mind, as some Renaissance scholars would describe them.

This book describes a new scientific discipline called affective neuroscience, which seeks to illuminate how our most powerful emotional feelings—the primal emotional affects—arise from ancient neural networks situated in brain regions below the neocortical "thinking-cap." The neocortex is an organ that generates complex cognitive abilities as well as culture, and it is definitively important for complex perceptions, learning, and cognitions. The neocortex is responsible for almost all of the cultural milestones that human beings have been able to achieve. And neuroscience has also provided an important message—practically all of the psychological specializations within the cortex are learned. None has yet been empirically demonstrated to be an intrinsic, evolutionarily dictated "module." However, the cortex could achieve nothing without an evolved foundational mind deeper in the brain. Those ancient neural territories below the neocortex constitute our ancestral mind—the affective mind, which is evolutionarily specialized and that we share with many other animals. It is "archaeological treasure," for it contains the sources of some of our most powerful feelings. Those ancient subcortical brain systems are precious, multihued "jewels" for anyone wishing to understand the roots of all the basic values we have ever known and will experience in our lives. The affects are the foundations upon which the beauty and ugliness of life has been constructed. And affects also change with experience, but more quantitatively rather than qualitatively.

This book is an updating and an attempt at popularizing an earlier textbook, *Affective Neuroscience: The Foundations of Human and Animal Emotions* (Panksepp, 1998a). This text has garnered wide attention as a major new approach to the science of the emotional mind and has become a source book for clinicians who wish to understand the basic emotions of their clients. Even though work on kindred animals has been so crucial to the development of affective neuroscience, Jaak Panksepp started his work with an interest primarily in human emotions, especially their disturbances in clinical disorders. He soon realized that deep neuroscientific understanding could not be achieved without appropriate animal models. This position has changed somewhat with the emergence of modern brain

imaging, but not much if one wants to really understand the evolved functional networks of the brain. It is rather difficult to have intense emotions while lying still within brain scanners that make measurements that cannot tolerate movements. Still the new evidence obtained with those spectacular human brain-imaging technologies has clarified much about the cognitive aspects of emotion but rather little about the sources of such feelings in the brain. The primary-process emotions are all connected to movements, and the evidence now indicates that raw emotional feelings arise from the same ancient brain networks that control our instinctual emotional life. Despite many theories in the field, the facts indicate that these raw emotional feelings arise from the emotional action networks of the brain.

Overall, the topic of emotions is of great interest to practically everyone —from psychiatrists who have to deal with human feelings that have become extreme, to anyone who is curious about those powerful states that govern so much of what we do and who we are in the world. We hope that what will be discovered between these covers will be of considerable use to many in their quest to understand themselves and others, including fellow animals, and to recognize how much all mammals share in the ways that they emotionally respond to the world. We suspect that many diverse groups of people will find these perspectives to be especially useful.

WHY PSYCHIATRISTS, PHYSICIANS, AND PSYCHOTHERAPISTS SHOULD UNDERSTAND THE SEVEN BASIC AFFECTIVE SYSTEMS

We have found that the ancient subcortical regions of mammalian brains contain at least seven basic affective systems: Here, we refer to these systems as SEEKING (expectancy), FEAR (anxiety), RAGE (anger), LUST (sexual excitement), CARE (nurturance), PANIC/GRIEF (sadness), and PLAY (social joy). (We will explain later why we use capitalization to label these systems; for now, suffice it to say that they designate specific functional networks of evolutionarily very ancient regions of our brains.)

This book should be of special interest to psychiatrists and other mental health professionals as well as students of the affective, behavioral, and cognitive neurosciences (each of which takes a rather different approach to the study and discussion of emotions). Our focus here will be on the

primary-process nature of these systems, but we will not neglect the levels that most other investigators are studying—the *secondary process* (inbuilt emotional learning mechanisms) and the *tertiary process* (emotional thoughts and deliberations that are so evident in human experience).

The failure of neuroscientists to deal empirically with the primary-process (evolved) level of emotional organization is impeding as coherent a synthesis of different approaches as is currently possible in emotion studies. As one ascends through the evolutionary layers of the brain and mind, there are more and more diverse ways to envision emotional life. In contrast, there is abundant evidence indicating that the basic affective systems of mammalian *brains* are ancient universal value structures of mammalian *minds* that provide evaluations of the world in the form of categories of individual affective experiences. The further up one goes in BrainMind complexity—from primary to tertiary levels—the more variable and complex the overall equation becomes. Multiple emotional streams may cross in the thinking mind, creating an enormous variety of higher emotions that are often the focus of psychologists—pride, shame, confidence, guilt, jealousy, trust, disgust, dominance, and so forth with hundreds of possible variants. However, without a clear vision of the primary processes the important work on higher processes remains profoundly incomplete. We cannot have a credible *theory of mind* without a credible understanding of the basic emotional feelings we inherit as evolutionary tools for living. It is possible that the higher (socially constructed) feelings all require certain permutations of our evolved capacities to feel certain ways. All aspects of mental life can be influenced by our primary-process feelings, and the overall affective spectrum of the lower MindBrain is foundational for higher mental health issues. The extent to which the lower powers of the mind eventually come to be molded by the emerging higher functions will be of great interest in future work. We already know that higher brain processes can arouse emotions, as dramatically as they reduce emotions. All this will remain a most interesting aspect of affective neuroscience for a long time to come.

Physicians, especially psychiatrists, must know about these affective systems, because they afford new insights into mind-body interactions. Some such interactions are already well known. Consider, for example, the misery of sustained anxiety, an expression of the FEAR system. Arousal of the FEAR system eventually leads to excessive production of cortisol.

Under optimal conditions when an animal is afraid, the secretion of cortisol mobilizes glucose as an energy supply for the skeletal muscles in case the animal decides to flee. In this way, cortisol secretion is beneficial. However, excessive secretion can begin to damage the body if elevations are sustained for too long. Normally when cortisol has circulated through the blood back up to the brain, the paraventricular nucleus (PVN) of the hypothalamus exerts an inhibitory effect that stops further release of cortisol. If, however, a person or animal is subjected to an excessive amount of stress—when they are chronically frightened or anxious—the PVN may not be able to stop the production of cortisol.

Although the intensities and time patterns of the emotional effects of cortisol can vary dramatically from one person to another, all visceral organs and many areas of the brain, as well as the immune system, can be adversely affected by a prolonged excess of cortisol. Many resulting stress-induced cascades in the brain and body can contribute to these adverse effects as well. Prolonged high cortisol levels are common in a number of psychiatric syndromes, most especially in depression. It is not known exactly how excessive secretion of cortisol can promote clinical depression. However, disruptions in the normal production of a variety of growth factors, such as BDNF (Brain-Derived Neurotrophic Factor) have been implicated. Play tends to promote positive affect partly through such chemistries (see Chapter 10), providing evidence for the common-sense principle that positive and negative feelings counteract each other in the affective economy of the mind.

In addition, when people are severely depressed they often suffer from hippocampal damage because an excess of cortisol can cause hippocampal cells to shrivel and at times even die off. Perhaps surprisingly to some, simply tickling rats and provoking the rats to "laugh" can promote the sprouting of new neurons in the hippocampus (see Chapter 10). The hippocampus is a brain structure that is essential for the creation of declarative and episodic memories—conscious memories of knowledge and experiences (see Chapter 6). Without this brain region, one would live in a perpetual present, with no memory of events that have passed. Thus, excessive cortisol release can participate in a number of serious mental disorders, including memory deficits.

Similarly, in small doses, opiates will elevate mood and promote social solidarity. In large doses, they promote intoxication. In fact, appropriate

amounts of endogenous opioids can have medically beneficial effects. For example, the placebo effect, whereby patients respond favorably to fake medications, can be explained in terms of this emotional chemistry. If a patient feels that his needs are being considered and tended to, then the positive feelings of being cared for are accompanied by the release, in the brain, of calming endogenous opioids, which diminish the feelings associated with the GRIEF/PANIC system.

In addition to producing good emotional feelings, opioids also reduce stressful arousal, reduce feelings of physical as well as psychological pain, and produce various immune benefits. So these patients will feel comforted and be much better off medically than they would be if they thought that no one seemed to care. We now know that the placebo effect is real medicine that operates mainly through the activation of brain opioid systems. These healing tendencies can thus be reduced, and even eliminated, by drugs like naloxone and naltrexone, which block the effects of opioids.

In the past, when an apparently healthy patient appeared emotionally agitated and complained of physical symptoms, doctors tended to believe that the symptoms were psychosomatic, "all in the mind," and therefore not physical or "real." This is no longer an accepted view of psychosomatic illness. As soon as we recognize that affects emerge from emotional systems that are fueled by brain chemicals that can also exert an eventual effect on the functioning of the brain and the body, then the division between emotional and physical disorders narrows to the point of extinction. Although it may appear that the mind and the brain are different entities, the mind being incorporeal, and the brain being physical, they are really one and the same thing. The MindBrain (or BrainMind) is a unified entity lacking any boundary with the body—it is integral to the physical system as a whole.

An understanding of brain emotional systems, and the psychological and bodily symptoms that they can generate, is not only important for medicine in general; it also offers a totally new perspective for contemporary psychiatry. Affective neuroscience points the way to treating the real and specific symptoms of emotional imbalances, the natural *endophenotypes* of the BrainMind, rather than vague nosological abstractions such as autism, depression, and schizophrenia, which were handed down to us with pre-neuroscientific classifications of mental disorders. These diagnostic concepts have been inferred from average clinical presentations. But we

now know that all of them are highly nebulous—each diagnostic category is a conceptual umbrella for a host of overlapping MindBrain problems.

For example, rats are inherently afraid of the smell of a predator. They also have an inherent fear of well-lit open spaces and thus prefer to be in dark and hidden areas. They often also exhibit symptoms of fear (commonly measured by freezing behaviors, elevations in blood pressure, and increased frequency of defecation) when placed in an unfamiliar cage. Common antianxiety drugs such as benzodiazepines quell the fear of open spaces and of a new cage. Rats still remain afraid of predator smell, however, suggesting that this is a somewhat different kind of fear. Surprisingly, morphine, which is so effective in reducing separation distress, is able to reduce a rat's fearful responses to the smell of predators. Ordinarily we lump different kinds of fear into a single category, but affective brain research suggests that there are neural models for distinct types of fear and anxiety. If this is so, then we should be able to develop specific drugs to treat each type. As we will explore in detail in a later chapter, there are convincing distinctions to be made between trepidation of the kind associated with physical danger (the FEAR system) and the panicky type of fear associated with separation anxiety (the GRIEF/PANIC system).

For quite a while, the development of psychiatric medicine has been stifled by man-made concepts, gleaned from complex symptomatology rather than from brain research. If psychiatric research were linked more to the actual emotional symptoms of the MindBrain and more productively linked to functional neuroscience, we might make much faster progress. For instance, we might easily develop specific drugs for irritability and anger. This is presently difficult to achieve because no official diagnostic categories have been designed for excessive anger (except perhaps for Intermittent Explosive Disorder). Yet society as a whole, and children in particular, are frequently victims of excessive RAGE. We already have medications such as Substance P receptor antagonists, and the drug aprepitant (a medication currently used to treat nausea), which should, if one can generalize from the animal data, reduce angry irritability (see Chapter 4). There is presently considerable excitement in pursuing a better understanding of such emotional endophenotypes, so that our diagnostic tools can be radically revised and so that better medicines can be developed.

Knowledge of the seven basic emotional systems has begun to revolutionize the practice of psychotherapy because it offers the most comprehensive, data-based brain taxonomy of primary-process emotions that is currently available. Knowledge of these systems also entails a more comprehensive view of how human emotions operate. We help to provide a data-based taxonomy for discussing the foundations of emotional life, and we provide many examples of the importance of specific brain functions in affective life—for instance, the powerful role of endogenous opioids and oxytocin in the positive affect of supportive social relationships. This provides neurobiological support for the view that healthy emotional development relies heavily on maintenance of supportive human interactions. In dire circumstances, the prescription of safe medications that support such brain chemistries can promote and solidify psychotherapeutic practice.

Just to highlight our approach to key conceptual issues in psychotherapy, let us consider how the present view contrasts with some of the tenets of classical psychoanalytic thought. We do this with intellectual admiration for the theoretical subtleties of that field, but here we focus mainly on how we would view primary affective processes differently than psychoanalytic theorists, whose views were based on clinical insights rather than on neuroscientific research.

Although psychotherapy has evolved in many different directions in the past half century, many therapists are continuing to rely on psychoanalytic theories to inform them about basic affects. Moreover, currently popular views of emotion, which envision some variation on a simple polar schematic of positive and negative affective valence, modulated by high and low arousal, have really not fallen all that far from the psychoanalytic tree. Freud maintained that human drives are rooted in our physiological needs, and he grouped these together into only two categories of drive: *libido* and *aggression*. Drives find psychic expression in wishful thoughts—in thoughts that are imbued with affective color. According to Freudian theory, the two main affects concerned wishes about sexual desires and aggressive urges.

Freud argued for several types of drive expression, each rooted in different stages of libidinal development: oral, anal, phallic, and oedipal. Aggressive drive was similarly partitioned along these developmental stages. This gave broader scope to the two interacting drives and their

consequent affective wishes. Nevertheless, the range of discrete affects was considerably more limited than those produced by the seven affective systems that have since been revealed by neuroscientific research. We are happy to note that the SEEKING system provides an interesting parallel to Freud's libidinal drive (insofar as he saw libido as a generic appetitive force, rather than in narrowly sexual terms). It is difficult to reconcile Freud's views on anxiety, however, as well as his views on lust in relation to attachment and affectionate bonds, and much else besides, with the knowledge we have derived from rigorous neuroscientific investigation.

Most modern psychoanalytic and cognitive-behavioral approaches to therapy fail to clearly identify SEEKING as a basic emotional urge. Some researchers also tend to confuse FEAR and PANIC/GRIEF, seeing anxiety as a single manifestation. The importance of social interaction is also insufficiently highlighted in many psychoanalytic theories. Freudians see social interaction as a derivative means of gratifying sexual and aggressive impulses. Social needs are not seen as basic urges that might, at times, supersede sex or aggression in importance, even at the level of primary instinctual impulses. Although object-relations theorists stress the importance of interpersonal needs, they tend to focus on early relationships within the family, particularly the mother/child bond. Today we have more information about the importance of PLAY, for example, and the associated basic psychology of social dominance.

At the same time, what we have to offer here says little about the unique, idiographic aspects of human mental life with which each psychotherapist must contend. There are higher, tertiary-process cognitive functions with which emotions will interact in real life. But by clarifying the primal mental energies that need to be considered as we try to help people in emotional distress, it may simplify the tertiary-process tasks of the psychotherapist. How? That would require another book. But perhaps one insight may suffice for now: The lower brain seems to be organized in such a way that one primal affective state prevails at any one time. This "monomania," for lack of a better word, also coaxes the cognitive apparatus "to follow" with obsessive self-serving ruminations. The goal of therapy is to facilitate a more complex perspective taking in the higher mental apparatus—what Aristotle called *phronesis*, becoming master of one's passions by understanding "low-minded" ways.

Perhaps this central problem in the clinical practice of classic psychoanalysis can be addressed by affective neuroscience. As we see it, a key reason that classic psychoanalysis may have been less effective than it could have been lay in the fact that *interpretation*—the crux of the talking cure—was long deemed to be the main psychotherapeutic tool. Psychoanalysts tended to concentrate on the relationship between affective states and their corresponding cognitive manifestations (wishes). They have long assumed that by interpreting relevant thoughts and ideas, by uncovering their origins in childhood and explaining their primitive emotional meaning, a patient will be cured. But how do we know this can untangle the emotional "knots" of most people's lives?

Suppose that in childhood a boy had endured physical and emotional abuse at the hands of his father. In adulthood, this man himself tended to bully those who were weak. A psychotherapist would help the patient to identify problematic areas in his adult personality, namely his tendency to bully or even abuse others, and would then trace these traits back to childhood. The therapist would perhaps interpret that this man bullied the weak and abused the vulnerable in order to vent his rage at his father in a way that would not result in retaliation. Other interpretations might highlight the possibility that he bullied others in order to restore his masculine self-esteem. As a result of these and still other interpretations, the patient would presumably be cured or at least proceed to have a happier life. In this vision cognitive issues were seen as a gateway to emotional ones.

The psychoanalytic tradition was followed, during the behaviorist era, with highly focused "behavior modification therapies," where both the cognitive and emotional issues were put aside and therapists sought to mold maladaptive behavior patterns by adjusting reinforcement contingencies. With the cognitive revolution, the focus shifted to "cognitive behavioral therapies" (CBT) that were remarkably effective for some disorders such as specific phobias (Beck, 1976). Now, with the recognition that emotional tides lie at the core of psychiatric disorders, the winds are shifting again.

The primacy of affect in BrainMind evolution suggests that therapies must have clear visions of human affective life, so that therapists can provide optimal understanding of and help for psychiatric problems. Indeed, such bottom-up views may turn the cognitive "interpretive" type of emotion theorizing in psychology and philosophy on its head. Clearly, even though

cognitive issues loom large in tertiary-process emotions, primary-process emotions have to be dealt with on their own terms. When traditional modes of therapy (psychoanalysis or CBT) fail to quell emotional storms, then probably medication is warranted. At present, most of these medications do not exist because psychiatrists do not know enough about the anatomy and chemistry of the emotional brain. We hope that this book may stimulate more research that will result in the creation of such medications. In a sense, what is needed is a fuller integration of all the therapeutic traditions, from dynamic-psychoanalytic to the new generations of affective balance therapies that will be the major focus in this book (see Chapter 12).

For instance, considering the case discussed above, suppose that the abuse suffered in childhood had fatefully sensitized the FEAR and RAGE systems in ways that made commensurate affects difficult or impossible to quell. Even if the therapist succeeded in convincing the patient about the origins of his problems and even if the patient was well aware that he was unfair and unjust to others, this might not be enough to effect any cure because he would still suffer from an overwhelming irritability, which may present itself as an apparent wish to bully.

Neuroscience supports this supposition. Two millennia ago, Plutarch noted that "the continuance and frequent fits of anger produce in the soul a propensity to be angry: which oft-times ends in choler, bitterness, and moroseness, when the mind becomes ulcerated, peevish and querulous and is wounded by the least occurrence." Plutarch, it seems, was correct. We now know that the RAGE circuits of the brain can be sensitized and become hyper-responsive. Thus, even if the patient fully understood the origins of his rage, and made an extreme effort of will to curb his rage, he might not be able to stop feeling chronically irritated, and he would remain emotionally ill. Perhaps others might be spared the deleterious effects of his anger, but the patient himself might continue to suffer as much as he did prior to therapy, perhaps even more, when he at least had an outlet for the feelings that he could not control.

The point is that thoughts are not always stronger than affects, which is why cognitive interpretations often do not work well with serious psychopathologies. Indeed, clients can be confused by complexities that the therapist sees "clearly." When affects maintain the upper hand, the talking cure is apt to fail because the interpretive method, the cardinal psychotherapeutic tool, can frequently be ineffective in the face of our

primal passions. Perhaps this is why even Freud himself looked forward to the day when it would be possible to exercise a direct chemical influence on the drives, as he saw them. But this does not mean that psychotherapy should simply be replaced by pharmacotherapy. Affective neuroscience research highlights that clinicians should not treat human beings as if they were bags of neurochemicals or "brains in vats." Affective feelings are part of the full equation, and they should not be ignored when psychiatrists seek new treatments for problems. Also, the mammalian brain is fundamentally a social brain, and it needs to be treated as such. The basic emotion systems do not operate in a social vacuum, even at the primary-process level. Thus, almost all mind-medicine interventions need to be complemented by appropriate psychosocial help, not only to trace and unravel the secondary- and tertiary-process derivatives of (perhaps lifelong) basic emotional imbalances, but also to guide, facilitate, and activate the desired primary-process affects. Positive affects can promote resilience, which can have lasting beneficial effects for many emotional problems. Affective neuroscience highlights that the role of social emotions in all future therapeutic schools of thought must remain in focus in order for lasting improvements to be maximized.

OTHER AUDIENCES

All people who wish to be well informed about human emotion—from parents to educators—will want to understand how feelings are created from within the brain. These affective systems have important implications for most academic disciplines that deal with human beings, from philosophy to economics and from the arts to the social sciences.

Parents

Parents will want to know about these systems in order to assess normal development in their children. If one sees a felicitous balance of all systems, this indicates that children are developing in emotionally healthy ways. But if a particular system is over—or under—aroused, this may indicate a problem. For example, an excessively studious or serious child may have an underactive PLAY system. The PLAY system allows children to learn about social rules of conduct—for example, when to cooperate and

when to compete, and at times to retreat in good-humored ways and let someone else win. When animals engage in rough-and-tumble play and one animal wins more than 70% of the time, the losing animal no longer enjoys the game and may drop out of such interactions entirely. So when children play, they learn valuable social skills, such as the necessity of reciprocity and giving way on occasion. Children will learn these skills because, if they do not, their playmates may begin to reject them.

Parents should understand the importance of maintaining an optimal balance of positive affects in their children, especially when they are very young. Subcortical emotional systems can become sensitized by experience. Neuroscientists are beginning to learn how emotional brain systems are molded, often permanently, through life experiences, just like the muscles and bones that carry our bodies dynamically into the world develop and strengthen over time. These changes can extend to the level at which genes become activated, sometimes leading to lifelong patterns of affective strengths and weaknesses. Understanding these *epigenetic* (environmentally induced) long-term changes in gene expressions and hence often the lifelong strengths and weaknesses of the BrainMind will be a most exciting forthcoming chapter in emotion research.

Therefore, children are blessed if they have received a great deal of nurturing CARE, leading to the formation of secure social bonds, with positive attachment facilitated by low activity of the PANIC/GRIEF system. If the child has had the opportunity to engage in abundant joyful play, and if the child's curiosity has been stimulated, then the neural circuits that support these capacities will be more robust throughout life. If, on the other hand, the child has been subjected to untoward frustrations that engender her RAGE system, or if the child has endured high levels of FEAR or PANIC/GRIEF, then her capacity for these negative feelings will be enlarged. However, this does not mean that parents need to protect their children from negative emotions. All children must learn to cope with them because they are a natural part of living. It is reasonable to believe that all the negative emotions, in small manageable doses, facilitate long-term psychological resilience that may help ward off longer-lasting future disappointments that could lead to depression.

Teachers

Teachers will surely benefit from knowing about the seven basic affective systems. All good teachers stimulate the SEEKING system when they make learning an exciting experience rather than purely a matter of rote memorization. However, given that much learning involves some measure of drudgery, teachers also need to impose social sanctions. The conscientious child is rewarded with praise, engendering satisfying feelings emanating from the positive social bonding arms of the CARE and GRIEF/PANIC systems. The recalcitrant child, however, must often endure the threat of disapproval with accompanying activation of the negative arm of the above social-affect systems, not to mention the throes of RAGE and FEAR. If so, that child's life will be ruled by negative affect and worries, rather than the positive affects that can spur children on to greater accomplishments. A second chance, offered gracefully to children with excessive negative affect, can be a wonderful life-sustaining experience. In any event, well-ministered social constraints can fortify children's ability to tolerate frustration and prepare them to deal with inevitable setbacks in adult life.

We will even emphasize how abundant physical play may reduce the incidence of impulsivity and problems such as Attention-Deficit Hyperactivity Disorder (ADHD). When children have fulfilled their natural urge to play physically, they are better prepared to sit still and pay attention in the classroom. The re-introduction of play might work best if we make recess the first class of each day. In effect, this need used to be met when children walked to school and arrived early enough to meet up with and engage playmates before classes started.

Managers and Supervisors

Certain emotional types seem to work best in specific roles and environments. Every manager needs to win the trust and respect of employees. Employees should feel that managers will help them with their problems at work, and managers should be confident that employees will meet their responsibilities. This implicit social contract is built on the mutuality of the CARE system. They must give each other what *they* need to feel secure and to excel. Managers also know the importance of team cohesion. Team days can support this process by fostering a spirit of PLAY, whereby members of a large working group share the opportunity to

interact in more intimate and relaxed environments. This kind of playful interaction cements social bonds that are important for the solidarity of the workforce.

Animal Behaviorists

People who work with animals will find much important information here about the emotions that control animal behavior. Indeed, one of the most sensitive and hence foremost animal behaviorists in the United States, Temple Grandin—a highly accomplished person with autism—has brought forward such information in her compelling book *Animals Make Us Human* (2009). This work also helps affirm long-held beliefs that animals do, in fact, have emotional feelings. Indeed, there is a rapidly growing movement, outside the academic disciplines, to recognize and value the emotions of other animals, but much of that is based on well-reasoned beliefs and fascinating anecdotes rather than on well-collected scientific facts.

The evidence summarized in our book aims to provide an empirical rather than an opinion-based view of what emotional minds are really like in mammalian species. The current evidence-based view is that all other mammals are full of emotional passion—they are quite full of affects. As we shall see, this is now a conclusion supported by vast amounts of experimental evidence (massively detailed in Panksepp, 1998a, and more modestly here). Those who remain in denial are adhering to a time-honored skepticism. In so doing, they typically fail to integrate modern affective neuroscientific research into their thinking. Perhaps other mammals cannot think about their affective lives in the ways that we do (their tertiary processes may be very different), but robust evidence indicates that they do experience a full range of primary-process affects.

We could go on about those who could benefit from understanding affective neuroscience: philosophers, politicians, artists, and other cultural leaders who want to make a better world. But most of all, we think that every person, to some extent, would want to become conversant with these basic tools for living that Mother Nature has endowed within our brains.

ACKNOWLEDGMENTS

We both have much to be thankful for.

Jaak is especially grateful for all the support and advice he received from his wife, Anesa Miller, who read and edited the entire manuscript. She completed this hard work while undergoing medical treatments for lymphoma. At the same time, Jaak was struggling with a different kind of lymphoma (thankfully, they are both in full remission at this time). Jaak is a member of the Center for the Study of Animal Well-Being within the Department of VCAPP (Veterinary Comparative Anatomy, Pharmacology, and Physiology) with the College of Veterinary Medicine at Washington State University. He thanks all of the fine colleagues who have helped make scientific pursuits a pleasure again. Sheri Six, Jaak's lab manager, has provided invaluable attention to the many details of keeping his lab going during these times, which in these days of modern science can be a daunting task. She also read the manuscript with her fine eye for detail and with a mind devoted to the sensitive use of animals in research. During the past year, Mark Solms, a beloved and respected colleague, also provided useful and enthusiastic input for every chapter. At the very end of this protracted journey to publication, Tim Lyons, a former student, who had become much more than a student, returned for a few weeks at the end of the summer of 2010 to assist with the final polishing, and he smoothed many remaining wrinkles in the text. His energy and devotion, especially based on this training for a second career in clinical/counseling (after being a lawyer most of his professional life), improved the book substantially. Thanks to all who helped out along the way.

Jaak thanks all his fine colleagues at the related science departments of Washington State and in the humanities department of the University of Idaho for the cordial support and camaraderie they have offered throughout the half dozen years of his third academic career. After receiving his Ph.D. at the University of Massachusetts in 1969, Jaak pursued postdoctoral work at the University of Sussex and the Worcester Foundation for Experimental Biology. Jaak's vision of primary-process emotionality in the mammalian brain matured as he progressed from being Assistant Professor to Distinguished Professor of Psychobiology at Bowling Green State University (BGSU) across 30 years of work that might not have been possible elsewhere. Following his early retirement, precipitated partly by medical issues and partly by the premature death of his daughter Tiina, Jaak joined the Falk Center for Molecular Therapeutics at Northwestern University, pursuing the genetics of the affective mind with the camaraderie

and intellectual and research support of Joe Moskal, Roger Kroes, and Jeff Burgdorf. He continues to collaborate with many former colleagues, especially on research on the genetics of the emotional brain, with the aspiration to identify new neurochemical pathways that control mammalian emotionality. He thanks his many colleagues at BGSU, especially Vern Bingman and Casey Cromwell, who organized a Festschrift to celebrate his work in May 2010, much of which appears as a special issue of *Neuroscience and Biobehavioral Reviews*.

Jaak also thanks Audrey Gruss and friends and colleagues at the Hope for Depression Research Foundation (HDRF) for their intellectual engagement with the problem of depression and for their fruitful interactions during the past few years. Jaak is currently the research codirector of HDRF, and his ongoing research is devoted largely to developing new animal models for understanding and treating depression. He has been recognized as a revolutionary (a radical by some) in his field, with many prizes and recognitions. His work is summarized in well over 400 scientific publications, half of which are listed in biological archives, and the other half in those serving the social sciences.

Lucy Biven is the former Head of the Department of Psychotherapy at the Child and Adolescent Mental Health Service, part of the National Health Service in Leicestershire, England. She became interested in neuroscience about 20 years ago when she was appointed by the Michigan Supreme Court to devise and implement a protocol for the transfer of custody of a 2½ year old girl from the home of a couple whom the child regarded as her parents, to the home of her biological parents. Like most of her colleagues, Lucy worried about the little girl's psychological development, yet the child progressed well and today is an emotionally healthy young woman. Where did it all go right? Only neuroscience provided the answers.

Thus began an abiding interest in neuroscience. Yet even after reading extensively for a number of years, she was dissatisfied because most research focused on perception, learning and memory rather than emotion. When neuroscience did touch on emotion it was usually fear and its role in conditioned learning. Neuroscience did not focus on a full range of emotions or on emotion itself.

Then in the year 2000 she attended a symposium in London arranged by The International Neuropsychoanalysis Society, chaired by Mark Solms.

Jaak Panksepp was a keynote speaker. Jaak was the first and only neuroscientist who focused squarely on the emotional brain. There followed a lengthy and instructive series of e-mails between Jaak and Lucy that ultimately resulted in the publication of this book.

Jaak's thoughtful research has enhanced her clinical work, but there are others to whom she is grateful for their instruction and advice. First is her father, Charles Brenner, a psychoanalyst, whose clear thinking and accessible written exposition always provided an exemplary goal. Anna Freud was still intellectually vigorous when she directed London's Hampstead Clinical where Lucy trained, and to this day, she has not met a more gifted clinician. While still a student, Lucy met Vann Spruiell, whose clinical and emotional honesty allowed her to see that psychoanalysis could and should be an invigorating pursuit as well as an intellectual endeavor. Along the way there have been other wonderful and influential colleagues, amongst them Josephine Klein, Anne Alvarez, and Thelma Hillaby.

Lucy was Senior Research Associate at the University of Michigan, under the inspired direction of Dr. Humberto Nagera, another brilliant clinician. She was a faculty member of the Michigan Psychoanalytic Institute, and in 1985, she received the Ira Miller memorial award for a clinical paper. She was an editorial reader for the *International Journal of Psychoanalysis* and also for the *Psychoanalytic Quarterly*.

She has written several papers about neuroscience and its relevance to psychotherapy and psychiatry and she has lectured widely in the United States, England, South Africa and Mexico. Finally, the most important person in her professional and personal life is her husband Barrie, whom she thanks with all her heart.

We both thank the fine staff at W. W. Norton who brought this work to fruition, especially Deborah Malmud, our acquisitions editor, who provided guidance and encouragement in the writing of this book.

Foreword

DANIEL J. SIEGEL, MD

AN UNDERSTANDING OF OUR INNER subjective lives and our interconnections with others is illuminated in a deep and helpful way in the in-depth journey into *The Archaeology of Mind*. By exploring our neural architecture, our social relationships, and our mental worlds and how they intertwine, neuroscientist Jaak Panksepp and psychotherapist Lucy Biven have created a detailed view into the ancient origins of human life. At the heart of this important synthesis is the notion that our subcortical circuits are the foundational substrate of "primary" experience—of emotions and motivations that shape our subjective lives, influence our behaviors, and mold our relationships. Panksepp and Biven propose that higher neocortical regions play an important—but distinctly "secondary"—role in how we learn to generate emotional responses, while the deeper, subcortical recesses that still exist within our older mammalian and reptilian circuits shape the innate textures of our everyday mental experience.

Jaak Panksepp has spent his academic life exploring the nature of these circuits, and his views serve as the essential core of this work. After a professional career devoted to advocating for the idea that non-human animals have an inner emotional world that needs to be both respected and understood, this important leader in the field of affective neuroscience has

turned his focus to helping human beings using these new insights into old circuits. Panksepp is an outspoken advocate for compassionate understanding of all members of the animal kingdom. With his work, we come to see the importance of honoring the inner core of subjective life and applying this knowledge to helping all lives.

Whether you are a clinician, educator, researcher, or interested general reader, you will find in these pages useful and detailed information within the fascinating discussions of seven major primary circuits that form our feelings and mold our motivations: SEEKING, RAGE, FEAR, LUST, CARE, PANIC/GRIEF, and PLAY. While the interplay of these subcortical systems with the higher neocortex is naturally essential in our experience of being human, in this book we are offered a chance to dive deeply into these more ancient sources of our affective core. We know that many aspects of psychotherapy and of mental training serve as important ways the neocortex learns over time and can change various aspects of our emotional brains (see Davidson & Begley, 2012, for a helpful discussion). Mindfulness meditation, for example, has been shown to alter cortical connections in important regions that regulate emotion, attention, empathy, and self-understanding. Attachment relationships (see Schore, 2012; Cozolino, 2010) may also shape prefrontal cortical regions that link our widely separated higher and lower neural areas (see Siegel, 2012a, 2012b). And so the neocortex learns from experience.

Naturally, a therapist, teacher, parent, or others interested in how learning shapes our minds and brains will see this neuroplasticity as an important dimension of how we change across the lifespan (see Doidge, 2007 for an overview of cortical neuroplasticity). So then why should we take the time to learn about more "basic" or "primary" neural areas that may be well formed before we are born—before extra-uterine learning begins? The answer is quite simple: These regions below the cortex serve as the substrate for both how the cortex grows in differentiated ways (see Trevarthen, 1990; McGilchrist, 2009) and how we come to experience mental life—our core, inner subjective texture of living moment by moment. Furthermore, a scientific view of these deep structures will only serve to expand our self-understanding and can offer empowering insights that may improve our lives.

In this book you'll find in-depth discussions of depression, anxiety, grief, and fear that may illuminate something about your own personal life. There

are also helpful explorations of how experience shapes the circuitry of memory and emotion, forming the neural foundations of our inner lives and altering our capacity to regulate our affective responses. These discussions offer the clinician important vistas into the nature of their client/patient's experience and how they can use this new knowledge to improve their capacity for empathic understanding and clinical intervention. The challenges people experience with social difficulties such as autism, learning issues such as attention deficit conditions, and emotion regulation problems such as disorders of mood, each take on a new light with the perspectives revealed in this work. This book also offers teachers a unique opportunity to understand the deep circuitries of motivation, emotion, and learning at the heart of the educational experience. When we realize that teacher–student relationships are based on trust, we come to see that these subcortical circuits set the stage for an effective learning relationship. If you are an academic researcher, this book provides a vast and detailed review of the subcortical aspects of affective neuroscience in one flowing narrative that may trigger some new ideas for understanding the field and perhaps may directly inform your own projects.

As someone trained both as a researcher and as a clinician, I have found this book to be a fascinating exploration of an often-ignored area of science and its application to therapeutic understanding. As an educator and the founding editor of the Norton Series on Interpersonal Neurobiology, I feel that knowing this material can help us bring more effective treatments and educational insights into our work and our world.

If I may, let me offer one suggestion here that may be helpful in the process of soaking in the pages that follow. If you are a scientist, you likely will be very interested in the ample details and abundance of academic references that are offered throughout the text. If, however, you are a clinician, educator, or general reader, you may find that a different approach to your reading will make this work more enjoyable. There is a lot of material here—written in an accessible and fascinating way—and there is no shortage of detailed discussions of neural circuits, transmitters, and the studies that illuminate what we know about them. Here is my suggestion to you: Read this work like a fascinating nonfiction story. Just like you wouldn't memorize a novel, do not worry about remembering all the details about research studies. You won't be tested on how well you've memorized what you've read! As you read in this more at-ease manner, you may find

that your mind will detect patterns of information that naturally emerge over time. Initially unfamiliar terms may begin to feel familiar, unusual names more comfortable to see and say, so that you'll start to become more at home with these less common terms as you go along. The old subcortical favorites that are in the popular press—such as the amygdala and hippocampus—are all here. But you'll also meet less well-known subcortical neural regions such as the periaqueductal gray (PAG) and nucleus accumbens, which also play important roles in this archaeological narrative of our emotional lives. You may be quite familiar with dopamine and serotonin, but you'll also find detailed discussions of prolactin and oxytocin here too. Relax and just listen in to this fascinating story as it unfolds. Let go of those ancient responses of FEAR and PANIC (from childhood and school) that you may have if you try to memorize everything you read. Instead, be PLAYFUL and SEEK out just what feels relevant for you as you go along. You are about to experience Jaak Panksepp's passionate mind and his way of thinking about our neural origins. Enjoy the journey with Jaak and let yourself take in a lifetime's labor of love and learning!

The Archaeology of Mind

CHAPTER 1
Ancestral Passions

. . . certain actions, which we recognize as expressive of certain states of mind, are the direct result of the constitution of the nervous system, and have been from the first independent of the will, and, to a large extent, of habit. . . . Our present subject is very obscure, but, from its importance, must be discussed at some length; and it always is advisable to perceive clearly our ignorance.

—Charles Darwin (1872)

THIS BOOK TAKES US ON an archaeological dig deep into the recesses of the mammalian brain, to the ancestral sources of our emotional minds. To the best of our knowledge, the basic biological values of all mammalian brains were built upon the same basic plan, laid out in consciousness-creating affective circuits that are concentrated in subcortical regions, far below the neocortical "thinking cap" that is so highly developed in humans. Mental life would be impossible without this foundation. There, among the ancestral brain networks that we share with other mammals, a few ounces of brain tissue constitute the bedrock of our emotional lives, generating the many primal ways in which we can feel emotionally good or bad within ourselves. As we mature and learn about ourselves, and the world in which we live, these systems provide a solid foundation for further mental developments. These subcortical brain networks are quite similar in all mammals, but they are not identical in all details. This similarity extends even to certain species of birds that, for instance, also have separation-distress PANIC networks—a GRIEF system, as we will often label it here—one of the main sources of psychological pain within their brains and ours (see Chapter 9).

We mammals and birds share many other basic emotional systems, and some even seem to exist in cold-blooded reptiles, but less is known about them. Thus, across many species of warm-blooded vertebrates, a variety of basic emotional networks are anatomically situated in similar brain regions, and these networks serve remarkably similar functions. We will discuss the nature of these brain systems that are being revealed by research on *other animals* (henceforth just "animals"). This knowledge is beginning to inform us about the deeper aspects of human nature. It provides a scientifically based vision about the origins of mind.

As briefly mentioned in the preface, the ancient subcortical regions of mammalian brains contain at least seven emotional, or affective, systems: SEEKING (expectancy), FEAR (anxiety), RAGE (anger), LUST (sexual excitement), CARE (nurturance), PANIC/GRIEF (sadness), and PLAY (social joy). Each of these systems controls distinct but specific types of behaviors associated with many overlapping physiological changes. To the best of our knowledge, these systems also generate distinct types of affective consciousness, and some of the most compelling data for that come from humans (Panksepp, 1985). As we will see, when these systems are stimulated in humans, people always experience intense emotional feelings, and presumably when the systems are normally activated by life events, they generate abundant memories and thoughts for people about what is happening to them.

The triangulation approach of affective neuroscience (discussed later in this chapter) provides an opportunity to assemble the needed evidence for these systems' effects. But to proceed effectively we need a new language to describe the emotional systems of the brain in order to match our emerging understanding of these primary-process psychological powers. This is why we capitalize the names of the affective systems. Vernacular usages handed down from folk psychology can create misunderstanding of these primary-process powerhouses of the mind. The capitalizations indicate that real physical and distinct networks for various emotions do exist in mammalian brains.

As highlighted in a medial view of the right cerebral hemisphere (Figure 1.1), these emotion-generating brain regions are concentrated in the most ancient medial (midline) and ventral (belly-side) brain areas, ranging from (i) the midbrain, especially a region known as the periaqueductal gray (PAG), or "central gray" as it used to be called; (ii) the hypothalamus and

medial thalamus, connected massively to (iii) higher brain regions, traditionally known as "the limbic system," which include the amygdala, basal ganglia, cingulate cortex, insular cortex, hippocampus, and septal regions (see Figure 1.2, which depicts the circuits hidden inside the left hemisphere adjacent to the one in Figure 1.1); as well as (iv) various medial frontal cortical and ventral forebrain regions (e.g., orbitofrontal cortex) that provide higher controls for emotional reactivity. Although the concept of the subcortical "limbic system" has been under assault for some time, all would have to admit that it was a great advance over some earlier views (e.g., the James-Lange theory) that situated emotions in higher brain regions.

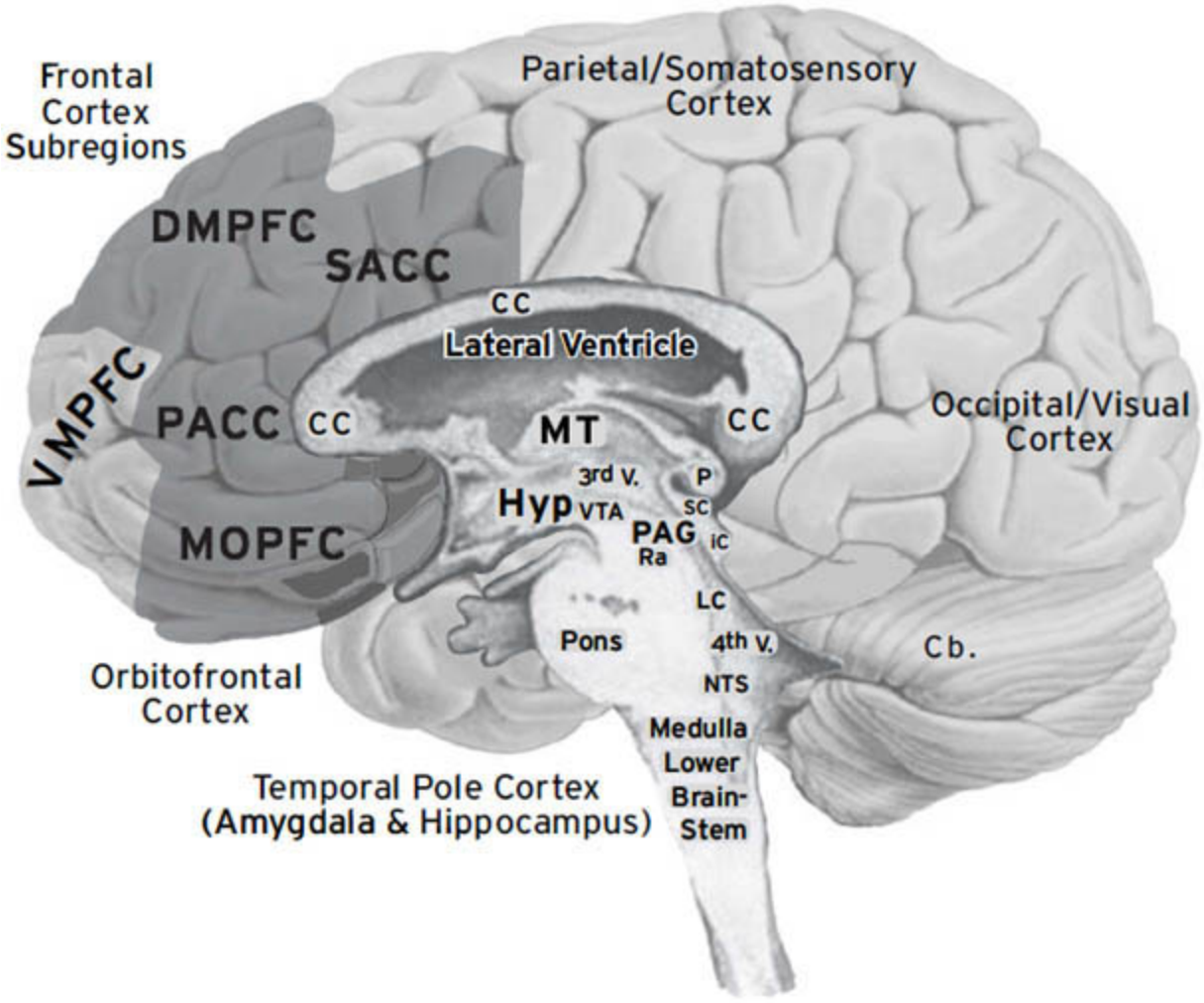

Figure 1.1. A medial view of the human brain (right hemisphere) that is highlighting some major regions of the brain. Going from front to back are the following abbreviations: DMPFC: dorsomedial prefrontal cortex; SACC: superior anterior cingulate cortex; VMPFC: ventromedial prefrontal cortex; PACC: perigenual anterior cingulate cortex; MOPFC: medial orbito-prefrontal cortex; CC: corpus callosum; MT: medial thalamus; Hyp: hypothalamus; VTA: ventral tegmental area (source of the mesolimbic dopamine system that innervates basal ganglia and medial prefrontal regions; see Chapter 3); P: pineal gland; sc: superior colliculus; ic: inferior colliculus; PAG: periaqueductal gray; Ra: Raphe dorsalis (the source of the major serotonin system innervating the limbic system); LC: Locus Ceruleus (the major source of the ascending dorsal norepinephrine pathway that feeds the whole forebrain); NTS: nucleus of the Tractus Solitarius (the location of the major internal receptor system coming from viscera via the vagus nerve); Cb: cerebellum. (We thank Georg Northoff for the use of this view of the brain.)

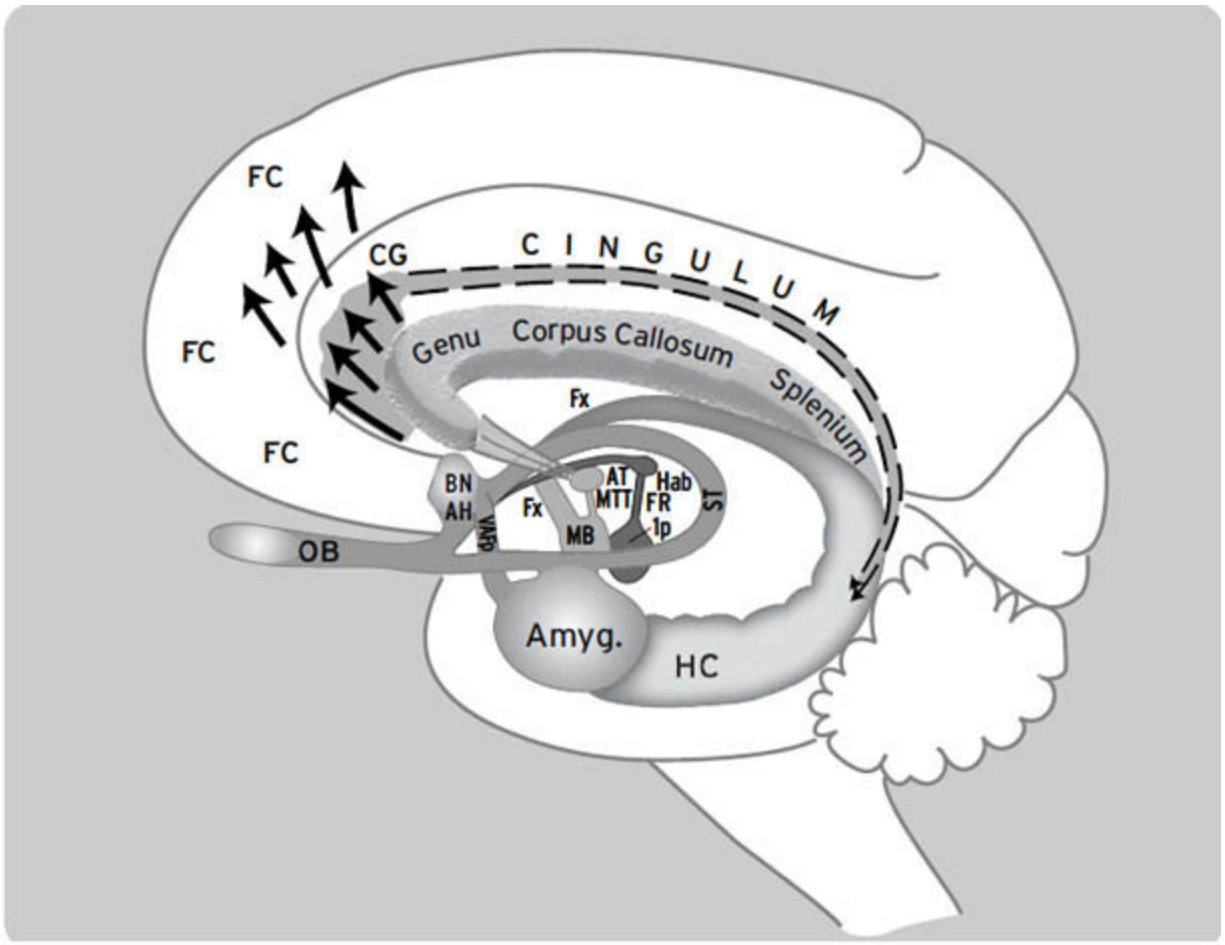

Figure 1.2. Schematic of the limbic system with the Papez circuit highlighted in stippling. FC: frontal cortex; CG: cingulate gyrus; OB: olfactory bulbs; BN: bed nucleus of the stria terminalis; AH: anterior hypothalamus; VAFp: ventral amygdalofugal pathway; Amyg.: amygdala; HC: hippocampus; Fx: fornix; AT: anterior thalamus; MB: mammillary bodies; MTT: mamillo-thalmic tract; Hab: habenula; FR: fasciculus retroflexus; ip: interpeduncular nucleus; ST: Stria Terminalis (from Panksepp, 1998a; republished with the permission of Oxford University Press).

As far as we know right now, primal emotional systems are made up of neuroanatomies and neurochemistries that are remarkably similar across all mammalian species. This suggests that these systems evolved a very long time ago and that at a *basic* emotional and motivational level, all mammals are more similar than they are different. Deep in the ancient affective

recesses of our brains, we remain evolutionarily kin. This has long been evident in our body structures and biochemistries. The same types of neural paths and brain chemicals that arouse each of these seven emotion-mediating systems are found within the various mammals. And according to current evidence, both humans and other mammals experience similar feelings when these systems are activated. Of course, these feelings cannot be identical, and we should not expect them to be. Evolution always adds diversity to shared general principles that, despite evolutionary diversification, provide the bridge for translating key issues from one species to many others. Many discoveries in modern medicine have been based on animal-models by using the same reasoning.

As we noted in the preface, these affective substrates are "archaeological treasures"—multi-faceted "jewels" of mind that embody our capacity for affective experience, a capacity that we still share with our animal cousins. However, as humans, we have higher brain expansions that allow us to think deeply about our nature as well as about our options to live more cerebrally, culturally, and creatively. We can bite our tongues when we are angry and not say things that make matters worse. But many "choose" not to. We used scare quotes in the previous sentence, because for many people their emotions are not under the willful control of their higher mind. Indeed, there are reasons to believe that our neocortical functions were substantially programmed by our lower mind, in conjunction with our early rearing, leading to blessed lives (Narvaez et al., 2012; Szalavitz & Perry, 2010) or to those full of misery.

Because of our higher brain expansions, we experience life at cognitive levels that other animals cannot imagine. We can reflect on our options in subtle ways, leading to ever more subtle feelings, constructed largely through learning. Our unique minds, in this world and the cosmos, arise from the cognitive riches of our higher neocortical expansions. But all the while, our higher minds remain rooted in our ancestral past. It is understandable that many wish to envision our affective lives as being completely intertwined with our cognitive abilities, but from a neuro-evolutionary perspective, that is not correct. Although many cognitive scientists and philosophers prefer to only think about our unique cerebral abilities, that does not serve our understanding of the origins of mind at all. But it is fascinating to think about those tertiary aspects of our minds. At that level, we have the full complexity of all the levels interacting, allowing

us to even dwell on our mortality, with existential dread, or to have feelings sublime (Hoffman, 2011). It is unlikely that other animals experience their minds with such neuro-affective angst and appreciative depth. But they surely experience their primal emotions, and surely some other levels that are much harder to understand. Here our concern is to go to the deepest roots of the human mind, through an appreciation of the minds of other creatures.

Although neuroscientists have long known much about the ancient emotional circuits of our brains, these circuits have only recently been definitively linked to our emotional feelings. This allows neuroscientists to delve deeply into the neural substrates of affects—the menagerie of our basic internally generated feelings. Which brain systems bring us joy? Why are we sometimes sad? Why, at times, are some people always sad? How do we experience enthusiasm? What fills us with lust, anger, fear, and tenderness? The traditional behavioral and cognitive sciences cannot provide satisfactory answers to such profound issues (and not simply because researchers have failed to ask such questions).

Affective neuroscience has made a fresh start by proceeding from the bottom up, without denigrating our unique human abilities, and it is offering both a new vision of mental origins and new data to back up such assertions. Affective neuroscience seeks to link the affective mind to animal brains—to triangulate among (i) subjective mental states (most easily studied in humans), (ii) brain functions (more easily studied in animals), and (iii) the natural (instinctual) emotional behaviors that all young mammals must exhibit early in life in order to survive. This triangulation allows us to envision the ancient ground plan for human mental life and the deep neural sources of our values—our primal emotional feelings.

This knowledge points us toward the brain functions we must study in order to understand emotional disorder—the various psychiatric syndromes that cause mental chaos in both human and animal lives. But maturational experiences soon supplement those evolved tools with abundant thoughts and learning, making the overall picture very complex. However, we plan to remain, as much as possible, at the primary-process level of analysis. This is not only because that level has been neglected by those who study psychology, philosophy, and the humanities. The analysis of the unconscious secondary processes is already a robust well-established branch of behavioral neuroscience (just think of fear-conditioning, which

we will dwell upon in Chapters 5 and especially 6). We will neglect the many higher-order (tertiary-process) aspects of the human mind, but we will argue that all those mental luxuries must be grounded on a most thorough understanding of the foundational issues. The reason we have not achieved that understanding is because these issues can only be well clarified through animal brain research. And for a century now, there has been very little discussion and research on how mind emerges in animal brains. Many researchers still claim that animals are mindless zombies with no comparable BrainMind organization that clearly leads, in humans, to a sense of self (Chapter 2).

There are surely many scholars who might disagree with the above strategy. We will try to avoid convoluted scholarly debate here (that would be endless), but we do need to give readers a flavor of the way in which many scientists with vested interests in this field might respond to our position. We will do this generically, usually without pointing at anyone specific who is still alive. Readers who are interested in pursuing the details of the diverse visions in this field may consult other publications by Jaak Panksepp, who has engaged with these issues many times. An excellent additional reading, highlighting the many views out there, is contained in *The Nature of Emotion* (edited by Ekman & Davidson, 1994).

There is currently a battle in psychology between those who believe we have "*basic*" emotions and those who prefer a "*dimensional*" view of emotional life. For a clear vision of that debate, a forthcoming collection edited by Zachar & Ellis (2012) may be especially useful: Within the volume is a full-length treatment of the views of Panksepp and those of Professor James Russell of Boston College, who has championed the *dimensional* view of emotional life. The dimensional view envisions that a unitary bivalent (positive to negative valence, and high and low arousal dimensions) arising from a brain process called the Core Affect is the fundamental grounding of our emotional nature. The debate was supplemented by additional perspectives taken by diverse commentators. This dimensional view has engendered abundant fine research, including recently, subtle animal emotion studies that have evaluated how animals make complex affect-related cognitive choices (Mendl et al. 2010). That approach can now be supplemented by affective neuroscience strategies, by linking findings to neuro-evolutionary levels of control within the BrainMind (see commentary to Mendl and colleagues by Panksepp, 2010a).

Such a hybrid approach is essential for making progress in understanding the fuller complexities of the MindBrain.

We use these two terms, mind and brain, double capitalized and in both sequences, to highlight that affective neuroscience is thoroughly monistic, with no remaining dualistic perspectives. The term "BrainMind" is used more often when we take the bottom-up view, and "MindBrain" when we take the top-down view, both being essential for understanding the "circular causalities" within the evolutionary strata of the brain. The double capitalization, without a space, also highlights the necessity of viewing the brain—"mind-meat" as some enjoy calling it—as a unified organ with no residue of the dualistic perspective that envisions mind and brain as separate entities, an intellectual tradition that has only hindered our understanding (see Chapter 2). At the same time the two versions of this term highlight (i) that certain aspects of the brain are intrinsic to the types of mental contents we have (BrainMind), while (ii) the other emphasizes that in upper regions of this organ, abundant learning and thought, commonly guided by societal and cultural influences, generate complexities that may not be clarified by animal research.

Thus, we have higher brain functions—commonly envisioned, these days, as a computational-cognitive mind—that need to be distinguished from a more universal affective mind. This distinction between affective and cognitive aspects of mind, although not popular, can be supported in many ways (Figure 1.3). It is important to ground psychotherapies on a knowledge of affective processes and thereby to understand how to most effectively recruit beneficial cognitive perspectives (Panksepp, 2010b).

The position that brain and mind are separate entities was Rene Descartes' greatest error, to borrow Antonio Damasio's (1994) famous turn of phrase. Another of Descartes' big errors was the idea that animals are without consciousness, without experiences, because they lack the subtle nonmaterial stuff from which the human mind is made. This notion lingers on today in the belief that animals do not think about nor even feel their emotional responses. Most who study animal brains have not yet learned how to discuss and study animal minds, especially their emotional feelings, as systematically and superbly as they study learned behaviors. Animals' primal feelings are best studied ethologically—by monitoring their natural emotional tendencies. Our view is that it is time for us to begin that difficult

journey, since it may tell us more about the ancient foundations of our own minds than any other approach that has been tried.

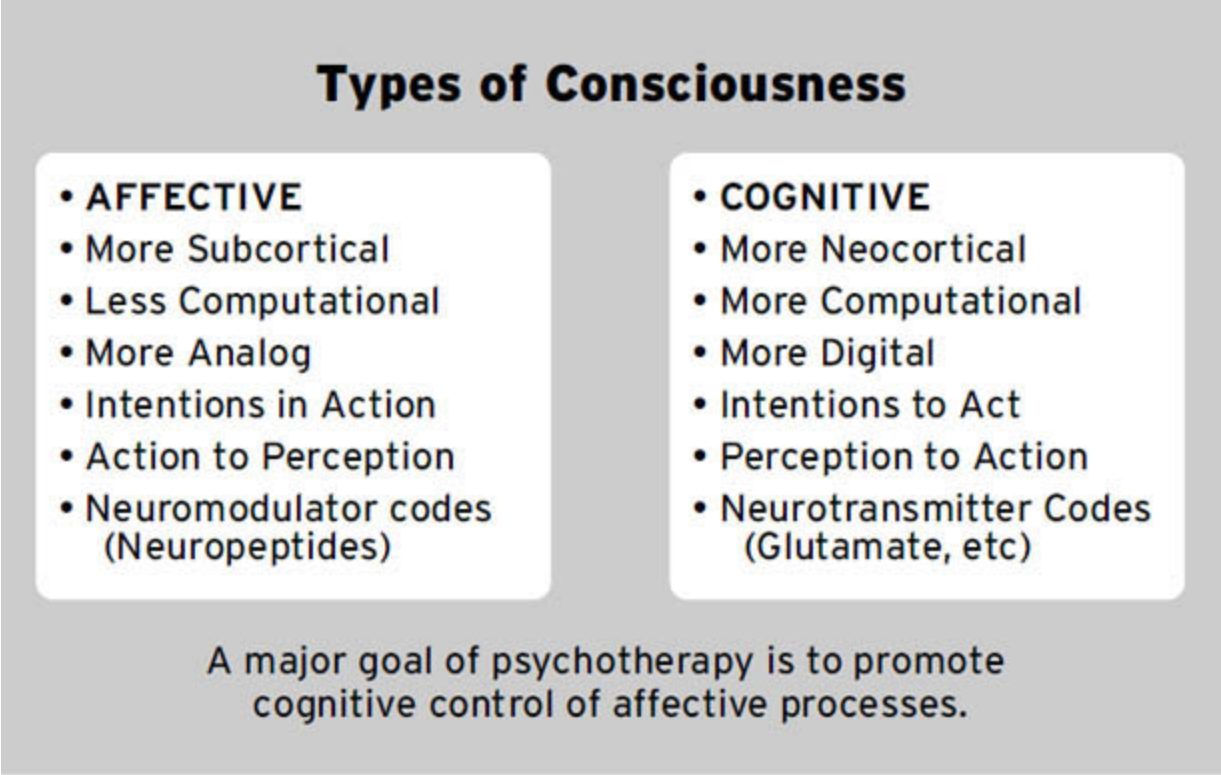

Figure 1.3. A summary of the major differences between brain systems that mediate affective and cognitive processes in the brain. Overall, the affective system controls global states of the brain, while cognitions process incoming information from the external senses.

Thus, the detailed knowledge of modern neuroscience, gleaned largely from animal research, has revealed that it is no longer useful to distinguish between the mind and the brain, although we surely must distinguish types of minds and types of brains: Affective feelings, which psychologists and philosophers try to understand largely in terms of ideas are, in fact, functions of the brain. But brain research that can get at neural "mechanisms" (i.e., the details of how a neural system actually works) is quite impossible to do in humans, ethically. Whether it can be done ethically in animals remains a matter of debate. In any event, we believe the evidence is definitive that other animals do have affective experiences, and

understanding these systems is very important for biological psychiatry as well as psychotherapeutic practices. Thus, we will feel free to refer to the MindBrain or the BrainMind, depending on which facet of the brain we wish to emphasize, whether it is in humans or animals. But our concern here is largely with the primary-process emotions of the MindBrain, as clarified by animal brain research.

Please consider the following additional terminological clarification before we proceed: In this book we are most concerned with, first, the instinctual emotional responses that generate raw affective feelings that Mother Nature built into our brains; we call them *primary-process* psychological experiences (they are among the evolutionary "givens" of the BrainMind). Second, upon this "instinctual" foundation we have a variety of learning and memory mechanisms, which we here envision as the *secondary processes* of the brain; these have been especially well studied by those who work on fear-conditioning (see Chapters 5 and 6); we believe these intermediate brain processes are deeply unconscious. Third, at the top of the brain, we find a diversity of higher mental processes—the diverse cognitions and thoughts that allow us to reflect on what we have learned from our experiences—and we call them *tertiary processes*. Recognizing such levels of control helps enormously in understanding the fuller complexities of the BrainMind (Figure 1.4).

Once we begin to seriously consider the evidence that already exists, we believe there can be little question abut the existence of many *basic* emotional feelings in the basement of the mind (Panksepp, 1998a). This "basic" vision of emotional life has also long been advocated by those who study the expressions of the human face (Darwin, 1872; Ekman & Davidson, 1994; Izard, 2007). Indeed, the most recent "meta-analysis" of human brain imaging, combining evidence from most of the relevant studies, has recently reached the same conclusion (i.e., Vytal & Hamann, 2010).

Many debates have arisen (e.g., Ekman, 1994; Russell, 1994) because human research really cannot clearly delineate the primary emotional processes of the human mind, since practically all the work with human beings proceeds at the tertiary and secondary levels of analysis. But because of the psychological power of primary-process emotions, those who study our facial expressions have seen the glimmers of basic emotions with sufficient clarity to convince most people that there is something

fundamental about our emotional nature. But they have not had the tools to tell us what that is. However, because of animal research, we can be confident that all mammals have many primary-process emotional systems, and other affective ones as well (sensory and homeostatic—Figure 1.4). And the systems are not concentrated in the neocortex, even though they have reciprocal relationships with our higher brain functions (Figure 1.5).

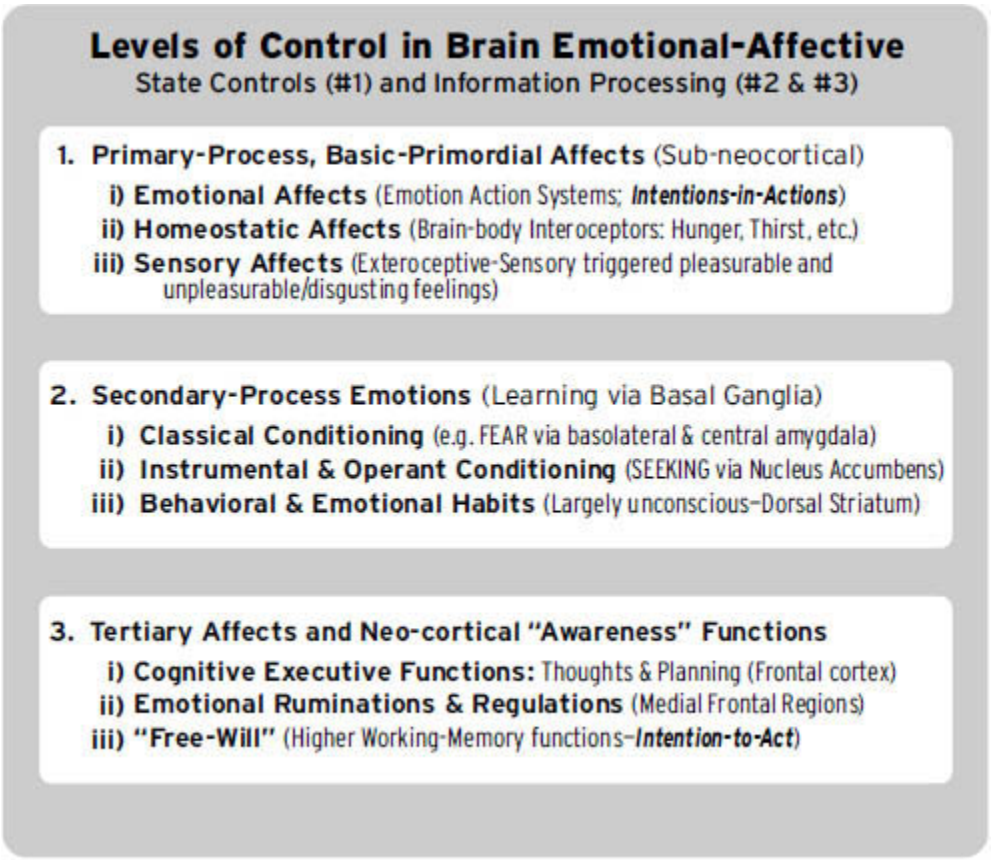

Figure 1.4. A summary of the global levels of control within the brain: (1) Three general types of affects, (2) three types of basic learning mechanisms, and (3) three representative awareness functions of the neocortex (which relies completely on loops down through the basal ganglia to the thalamus, looping back to the neocortex before it can fully elaborate both thoughts and behavior).

There are few neuroscientists and even fewer psychologists who are working on how primary-process emotional mechanisms, shared by all

mammals, are constituted in the brain. Almost none are working on the feeling (affective) aspects. This helps explain the century-long silence about how affects are actually created within brains. In contrast, many, many scientists are working on perceptual functions such as hearing and vision (for a fine summary of lower-brain perceptual abilities, see Merker, 2007). The almost universal neglect of the primary-process affective networks of the brain leads many scholars of human psychology, not to mention social scientists and philosophers, to neglect issues that their closest interdisciplinary colleagues do not talk about.

In recognizing the evolutionary levels within the BrainMind, one issue regarding brain specializations is of critical importance: At birth, the neocortical "thinking cap" of our MindBrain is largely a blank slate, and experience imprints many abilities and skills up there "naturally." These imprints include what seem to be "hard-wired" brain functions like our sophisticated hearing and visual abilities. At the neocortical level, those abilities are constructed by the process of living in the world and not by any stringent genetic dictates. Among the many critical lines of evidence, the most compelling is as follows: If we eliminate the cortical regions that are "destined" to become visual processing areas *before birth*, perfectly fine visual functions emerge in adjacent areas of the cortex (Sur & Rubinstein, 2005). The subcortical (e.g., thalamic) influences, perhaps directly from the visual projections of the lateral geniculate nucleus (LGN) or perhaps chemical gradients in the cortex itself, are sufficient for the cerebral surface to develop visual competence. Parenthetically, we can be confident that sophisticated hearing is a more ancient process in BrainMind evolution than vision. This is because at the midbrain level, the Grand Central Station of auditory processing—the inferior colliculi that project to the medial geniculate nuclei (MGN) in the thalamus—is lower down (more caudal, implying more ancient) than the hub for midbrain visual processing (the superior colliculi), which project to the LGN. This also may help explain why hearing, which evolutionarily emerged from touch, is a much more emotional sense than vision.

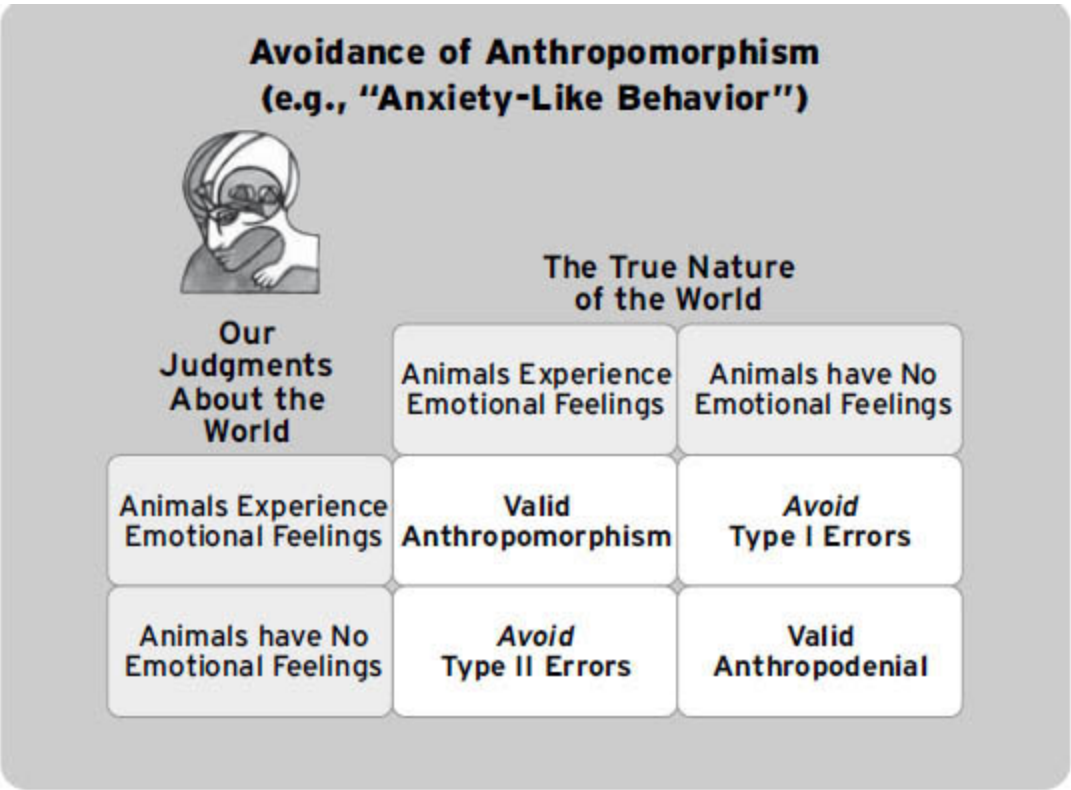

Figure 1.5. A truth diagram relating how we need to think about the possible affective nature of animals. Most of the twentieth century was spent believing that the right lower corner was the correct place to be philosophically, so one could avoid Type I errors, namely concluding something that is not true to be scientifically correct. This led to discussions of "anxiety-like" behaviors in animals as opposed to actual fear in animals. This book is premised on the data-based conclusion that scientists are wise to situate themselves in the upper left quadrant, because that way we can avoid Type II errors, which is missing the detection of a real phenomenon because we have false beliefs, or inadequate methods to evaluate the presence of a phenomenon.

This principle by which we can roughly "date" brain systems is at present just a rule of thumb, and there are exceptions. For instance, more modern downward influences from the neocortex do penetrate through many old layers of the brain. Perhaps the most dramatic example is the longest pathway in the brain, the cortico-thalamic tract. This tract courses all the way from the motor cortex in the frontal regions of the brain far down into

the spinal cord, allowing us voluntary control over our fingers and toes, as is needed to play pianos and all other musical instruments with full sophistication, to perform dance routines, and to write books.

Many emotion researchers as well as neuroscience colleagues make a sharp distinction between affect and emotion, seeing emotion as purely behavioral and physiological responses that are devoid of affective experience. They see emotional arousal as merely a set of physiological responses that include emotion-associated behaviors and a variety of visceral (hormonal/autonomic) responses. In their scientific view, animals may show intense behavioral emotional responses, without actually experiencing anything—many researchers believe that other animals may not feel their emotional arousals. We disagree. Some claim that the systems we will talk about are deeply unconscious—without anything happening in the ancestral theater of experience that we call the primary-process BrainMind. We believe the evidence speaks otherwise.

Most neuroscientists are willing to agree that many physiological and emotional behavioral responses are initiated by subcortical structures located deep inside the brain, but they typically deny or ignore that these same structures can generate raw *affective feelings*. According to their view, if an animal is exposed to danger, deep brain structures generate automatic behaviors (like freezing or running away) as well as visceral responses (like increased heart rate and the secretion of cortisol, a universal stress hormone, into the bloodstream). They believe that the response is purely physiological—purely emotional *behavior* without any accompanying affect. Such scholars are all too ready to claim that anthropomorphism—the attribution of human-type psychological processes to other animals—is fundamentally incorrect (for a fine discussion of such issues, see Daston & Mitman, 2005). Many others choose to remain silent about such issues, preferring a more cautious agnostic stance. Our reading of the evidence for all mammals that have been studied in affective neuroscientific ways is that human and animal minds are grounded on genetically homologous—evolutionarily related—affective systems, providing many similar biological "value structures" for higher mental activities (see Figure 1.5 for the truth diagram that needs to guide everyone's thinking on the matter). Obviously, some systems will be very comparable, while others, especially the social emotions, will differ more because of selective pressures for evolutionary divergence.

Raw emotions are not everyday occurrences for mature humans, but most can remember clenching their fists and turning red in anger, being incredibly scared, and feeling both deep sadness and joy. Our task here will be to share evidence about such primary-process mechanisms of mental life, much of which comes from the study of animals. Such feelings create an energetic form of consciousness—one that is full of affective intensity—that we will call *affective consciousness*. Primal feelings are not intrinsically bright and intelligent, but they were built into our brains because they are remarkably useful for immediately dealing with the world and learning about its potential. Primal affects are ancestral memories that have helped us to survive. There are many ways these ancient brain networks can make us feel—experiences we sometimes call *core emotional affects* and *raw emotional feelings*. Regardless of which term we use, we are talking about the same thing.

Cognitive scientists who study humans are prone to claim that emotional feelings emerge from some of the highest regions of the human brain. Many scientists who are interested in human psychology, as much as we are, maintain that affects are created when a person or animal is able to make cognitive sense of the changing peripheral physiology of emotion. In other words, affects are defined by and derived from cognitive reflections upon the responses of the body, rather than being intrinsic to the brain itself. On this view, if a person has a churning stomach or clenched fists, the higher cognitive brain (neocortex) interprets these primitive physiological responses as they enter the brain via sensory nerves and label those feelings as emotions. And supposedly it is only then that the person has the subjective experience of feeling anxious or angry. This is the famous James-Lange theory of emotions that was proposed well over a century ago (see Chapter 2). Now we know that the brain itself typically instigates the bodily arousals that accompany emotions. But despite that, some colleagues go further and assert that affects only come into being when we can actually verbalize them—feelings emerge from our ability to conceptualize the unconscious forces of our minds. Since the neocortex, the outer rind of the brain, is the seat of cognition and language, these cognitive/linguistic theories maintain that affects are created when the neocortex "*reads out*" the physiological controls of emotion that are situated within the brain. For them, the deeper parts of the brain that we will focus on cannot generate any experiences. We believe that the evidence speaks otherwise.

Implicit in read-out theories is the equating of consciousness with cognitions—our self-conscious awareness of our feelings and accompanying thoughts. And if one believes that consciousness is always cognitive, then affects must somehow be cognitive too. According to read-out theories, affective consciousness cannot emerge from the deep brain functions that generate the physiological changes and instinctual behaviors of emotions, because these deep substrates are noncognitive and must therefore be deeply unconscious. Affects can only emerge from the conscious thinking that relies heavily on the very top of the brain, our neocortex, which is essential for all of our higher cognitive activities. However, a vast amount of animal research and many clinical observations oppose this equation of consciousness with cognition. If one accepts affective feelings as a fundamental form of consciousness, there are many ways to distinguish those states of mind from the kind of information processing that constitutes cognitive consciousness, the foundation of human rationality (Figure 1.3).

Here is one extreme example: Human babies who are born basically without cerebral hemispheres (they are anencephalic) and hence have essentially no neocortex will remain intellectually undeveloped, but they can grow up to be affectively vibrant children if they are raised in nurturing and socially engaging environments (Shewmon et al., 1999; for photos of such a child, see Figure 13.2). As we will see, many decortication experiments have been done on laboratory animals. To the untutored eye, these animals are indistinguishable from normal animals. In fact they are more emotional than normal. Since such children and animals have little neocortex, their affective capabilities must emerge from the other parts of the brain that lie below. This is as close to a proof as one can get in science, where conclusions are more typically constrained by multiple possible interpretations. Revolutionary neurologists and neuropsychologists are now pointing out that even our higher cognitive minds could not work without the low subcortical systems that permit them to do so (e.g., Damasio, 2010; Koziol & Budding, 2009). Our view is also that the ancient affective foundations of mind are essential for many higher mental activities. In short, to understand the whole mind, we must respect the ancestral forms of mind that first emerged in brain evolution.

Needless to say, aphasic stroke victims who have lost the ability to speak or even to think in words (usually due to left neocortical damage) will also

retain their affective capacity, which indicates that affective consciousness is independent of language. Thus clinical observation suggests that neither cognitive ability nor the ability to think in words is a necessary condition for affective consciousness. Felt experience can be *anoetic*—an unreflective, unthinking primary-process kind of consciousness that precedes our cognitive understanding of the world, or our so-called *noetic* (learning, knowledge-based) secondary-process consciousness. Continuing in the words of esteemed neuropsychologist, Endel Tulving (2002, 2005), this allows us *autonoetic* tertiary-process thoughtful consciousness—the ability to time travel and to be able to look forward and backward within our minds.

This perspective includes the radical assertion that primary-process core affects are *anoetic* (without external knowledge) but intensely conscious (experienced) in an affective form (which reflects intrinsic, unreflective brain "knowledge"). As we feel our affective states, we do not need *to know* what we are feeling. In other words, the primary-process emotional feelings are raw affects that automatically make important decisions for us, at times unwise decisions, at least based on the views of our upper cognitive minds. In civilized society, with rules of conduct, emotional acting-out is often unwelcome. Still, the capacity to generate such affective feelings was one critical event in brain evolution that allowed higher forms of consciousness to emerge. Full conscious *awareness* surely had to wait until we had enough cerebral cortex, especially in frontal regions, that allowed us to think, with *autonoetic*, executive, decision-making abilities. But all that fine mental machinery is still heavily influenced by our emotions. The intrinsic evaluations that affective feelings convey to the higher brain enable humans and animals to determine how well or badly they are doing with respect to survival. But at times, they simply get us in trouble. If that keeps happening, psychotherapy is commonly very useful.

Another helpful way to envision these evolutionary layers of mind is summarized in Figure 1.6. At the left, we envision the "magnitude" of these layers in early development—the infant is almost purely primary-process consciousness at first but as the infant matures and grows into an adult, those ancestral values "seem" to get smaller, as our higher brain becomes filled with knowledge and opinions (at the right side of the figure). Most psychologists try to deal with the upper levels of mind, and also the middle levels by studying basic learning and memory processes (Chapter 6).

Neuroscientists are the only tribe of scientists that will ever be able to clarify the mechanisms of mind—knowing how we come to experience ourselves and the world. Regrettably few so far have sought to illuminate the affective feeling side of consciousness, which may be especially important for understanding human emotional problems and psychiatric disorders.

Our main goal here is to deal with the nature of those primary emotional processes that are foundational pillars for the brain's mental apparatus. In early life, the primary processes guide what infants do and feel; in maturity, acquired higher brain functions seem to be in complete control—which, as every psychotherapist knows, is rarely the case. We will only tangentially touch on the higher emotional and cognitive processes, but it is clear that those higher brain functions would collapse without the solid affective/evolutionary foundation upon which they are built. This hierarchical scheme readily allows us to handle some traditional paradoxes in the field. For instance, it is often asked why humans like to go to frightening movies. The answer is simple: At the highest tertiary-process levels of mental activity—for instance, autonoetic consciousness—we can be superbly entertained by having our primary-process systems manipulated in situations where we are in fact safe. We can also enjoy a thunderstorm; however, most animals tremble. Without such higher reflective processes, we humans would be unlikely to "voluntarily" expose ourselves to perceptions that can trigger negative affects such as FEAR. We can also be confident that our thoughts often follow our feelings. One of the earliest demonstrations was simple enough: When people were coaxed to be happy or sad, their thoughts tended to follow their feelings (Teasdale et al., 1980). This is a universal observation. But this does not mean that the feelings that characterize happiness and sadness arise from our higher brain. There is no evidence such primitive feelings are "read out" by the neocortex. But such beliefs persist.

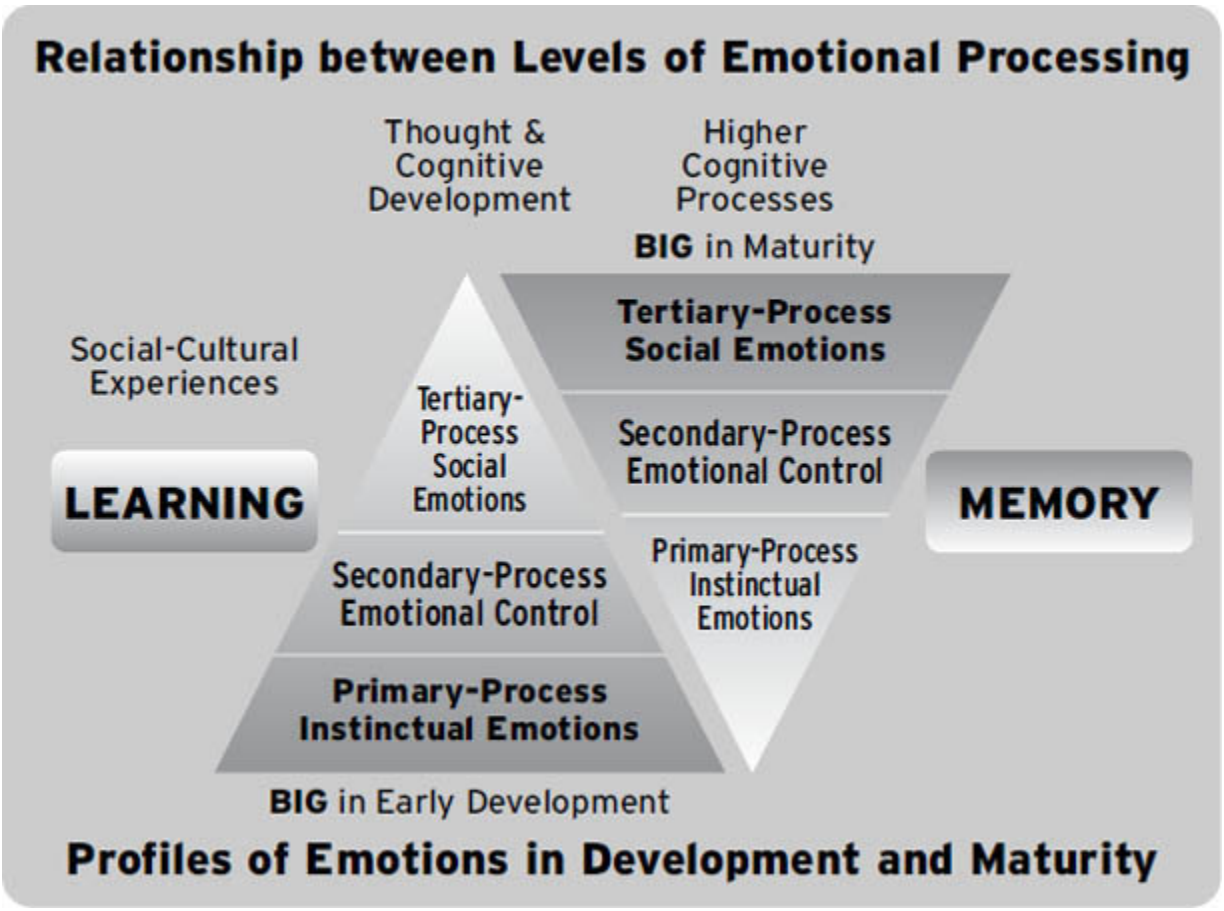

Figure 1.6. A diagram that summarizes the levels of control within an infant's BrainMind, where instinctual primary-process emotional responses are very prominent and higher mental processes are undeveloped. This can be contrasted with MindBrain organization in adults, where the higher mental processes (tertiary processes) are well developed, but primary processes are inhibited, which may indicate that primary processes have only a modest influence on mental life or that they are still quite influential, but, in well-bred individuals, are under higher mental regulations.

Read-out theories imply that affects can only occur either in animals that are intelligent enough to interpret emotional physiology or in animals that have language. This would mean that only human beings and perhaps some other primates are affective creatures. Presumably less intelligent mammals copulate without lust, attack without rage, cower without fear, and nurture without affection. They cannot feel the sting—the psychic pain—of social loss. This may be an extreme depiction of the prevailing view, but it is not

far off the mark among those who are actually doing animal brain research and hence (presumably) should be deeply concerned about such issues.

In spite of, or perhaps because of, recent changes in the zeitgeist—from animal rights movements to popular books about animal emotion—most neuroscientists remain steadfastly agnostic on the topic of affect in the animals they study. If you cannot measure affect directly, then, many say, you should not discuss it. But we *can* measure core affects. We simply need to take indirect approaches, such as determining whether artificially induced arousal of certain ancient brain systems, as can be done with localized brain stimulation, can serve as "rewards" or "punishments" in various learning tasks. In fact one of the general principles emphasized throughout this book, as the most compelling evidence for distinct emotional experiences in animals, is that whenever we arouse instinctual emotional behavior patterns with direct brain manipulations, animals treat those artificially evoked internal states as rewards and punishments that can lead to approach and escape learning. Such evidence provides rigorous support for affective mind-sets in other animals. It also tells us which brain regions we need to understand in greater detail before we understand how those feelings are constituted by neural networks.

Brain scientists have to learn how to use such evidence effectively as did those who have already studied the nature of the world in such great detail. Had physicists ignored such relatively hidden aspects of nature—taken a head-in-the-sand approach, so to speak—we might have been spared the quantum revolution that led to warheads of tragic proportions. While an understanding of the raw emotional feelings of animals may not be that explosive, it will change the way we scientists discuss human nature and its various psychiatric disorders. It may change the way we envision the evolution of mind as clearly a bottom-up process that eventually permits top-down control (Fig. 1.6). But a whole generation of behavioral neuroscientists has to learn how to speak explicitly about internal affective states in the animals they study. There are still major resistances to engaging in a full conversation on such topics that have traditionally been shunned.

AFFECTS ARE PRIMARY EXPERIENCES

In later chapters we will argue that it is now most credible to believe that the varieties of (i) raw emotional feelings, (ii) instinctual emotional behaviors, and (iii) accompanying visceral responses, are all orchestrated by at least seven "relatively" distinct subcortical systems—the systems for SEEKING, FEAR, RAGE, LUST, CARE, PANIC/GRIEF, and PLAY. We say "relatively" since many of these systems have overlapping controls: for instance, general purpose arousal/attention-promoting systems that are mediated by famous transmitters such as acetylcholine, norepinephrine, and serotonin—the cell bodies of which are heavily concentrated deep in the brain stem (see Figure 1.1, which provides approximate locations of a few key groups in the human brain).

We must also emphasize "relatively" since the biggest systems, such as SEEKING, are crucially important for the other emotional systems to operate. We seek many things and in many ways, as this system guides diverse kinds of anticipatory learning. To the best of our knowledge, the SEEKING system, and all the other emotional systems are remarkably similar in all mammals that have been studied. The feelings of other animals are surely not identical to those that people talk about when they use various vernacular terms (anger, anxiety, etc.), which typically are connected to specific life events, but they are bound to be quite similar because the feelings are generated by the same brain regions and involve the same neurotransmitters and other brain chemistries. Thus, the *core emotional affects* we will discuss as existing in other animals are bound to have strong correspondences to the emotional feelings that humans experience.

But there are yet other types of affect that we do not call "emotional" (including the brain representations of various bodily states such as raw HUNGER and THIRST, namely the *homeostatic affects*, which include, in the vernacular, urges to pee and poop). In addition, there are the pleasures and pains of externally provoked sensations (e.g., sweetness and bitterness, and other *sensory affects* such as DISGUST and many others, including distinct types of pain). We will not discuss these *homeostatic* and *sensory* affects here in any detail. The behavioral side of these topics has received substantial research attention by behavioral neuroscientists, albeit with hardly a mention that they may also be accompanied by affective states. Why are we then focusing on the emotional feelings, besides the simple fact that they are so interesting? This is because a study of those kinds of affects

is most important for understanding human psychiatric disorders, and it will also enable us to have effective animal ("preclinical") models of human emotional problems. We cannot make as much progress if we talk about only the behavioral changes of animals, without talking about their feelings and how they are controlled within the brain.

In sum, our claim is that we are prudent to accept that affects are integral parts of emotional expression in *all* mammals, rather than cognitive afterthoughts in just a few species. Do we mean to say that animals feel exactly the same as we do? Of course not! Diversity is the rule in evolution. Surely all the fine details of brain and bodily processes differ substantially in each species. Indeed, even identical twins are not identical in the fine structures of their nervous systems. When raw feelings mix with our higher mental abilities, many further variations and permutations are bound to arise—these will create complex social emotions like envy, guilt, jealousy, and shame, as well as awe, hope, humor, . . . even the capacity to experience reverence and the sublime (Hoffman, 2011). We may never scientifically know whether animals have such higher feelings, for that requires us to know their thoughts, which we cannot do yet, with as much confidence as we can read their emotional feelings. Surely some higher emotions are unique to different complex creatures, especially those, like ourselves, who have the brain power to think and speak deeply about their existence.

In the normal course of life, especially in childhood, affects become enmeshed with the *development* of higher cognitive abilities. This is due to interaction between the primal affective substrates, which we will focus on, and the maturing neocortex. The neocortex varies dramatically in size and complexity from one mammalian species to another, resulting in rather different levels and types of cognitive abilities and intelligences. As already noted, higher-order emotions are bound to diverge enormously among different mammalian species. Most of the complex emotions (the cognitively elaborated, socially constructed "mixed emotions" that are so common in humans—think of shame and scorn) have not yet been subjected to any detailed neuroscientific analysis. Realistic laboratory models do not exist for envy and guilt, albeit some progress is being made on feelings like jealousy (Panksepp, 2010c). Because of advances in technology, such as functional Magnetic Resonance Imaging (fMRI) brain scans, we can now image even such subtle higher mental processes within

the human MindBrain. And jealousy yields different pictures in male and female brains (Takahashi et al., 2006), with male jealousy arising more from lower emotional brain regions while female jealousy emerges from higher cortical regions. Perhaps this indicates female jealousy is more of a cognitive response, based on the evaluation of how much they have to lose economically. Males are more concerned about sexual matters. Remarkably, when a brain-imaging study of jealousy was done with "lower" primates (rhesus macaques) by having a dominant male view submissive animals having sex with his consorts, the brain arousals resembled those observed in the aforementioned human study (Rilling et al., 2004). It is quite easy to envision male jealousy to be a mixture of feelings of SEEKING, LUST, FEAR, and impending GRIEF (Panksepp, 1982, 2010c), but that is only a theoretical conjecture at the present time.

In our own intelligent species, complex ideas become intertwined with affects. Differing cognitive capabilities of other animals would undoubtedly create different higher mental landscapes. However, homologous affective substrates, lying deep in the subcortical brain, are anatomically and neurochemically distinguishable from the neocortex and are very similar in all mammals. These facts indicate the existence of systems that generate a variety of similar primary-process affective experiences across mammalian species. It is possible that most complex social emotions arise, through learning, from the more primitive affective dynamics combining with cognitive attitudes. Namely, primary-process affects surely control secondary-process learning mechanisms, and then these both combine with higher cognitions into a tertiary-process mental landscape that most psychologists focus on. There is much interest currently in the complex learning and even higher mental abilities of other animals, but little of that intriguing work has been connected to brain research.

Because of the intermingling of affects with complex ideas and personal experiences in our forward-looking and backward-reminiscing *autonoetic* consciousness, we humans often have difficulty imagining that affects can exist independently of the higher mental contexts in which they occur. We often find it hard to conceptualize feelings in their purest form. It is much easier to view them in the detailed cognitive contexts of our lives. We think that someone specific has made us feel angry or that a frightening experience causes us to experience fear. (In philosophical terms this means that affects are intentional—they are always "about" something. They are

"propositional attitudes" that arise from "emotional appraisals"—issues we will only consider in passing here.) Because of the way the brain is so highly interconnected, we experience ideas and affects as totally intermeshed experiences, and because we are highly cognitive creatures, we tend to see cognition as primary, assuming that affects are created by thoughts or perceptions. There are still some psychologists who assert that life experiences teach us to have affects, and that without these experiences we would not have affective capacities. They claim that people who have never encountered dangerous or painful situations before would not be capable of feeling afraid. For such theorists, emotions are largely learned responses.

But at the primary-process level, emotions are not a matter of individual learning. They were built into the brain by evolution: They are ancestral "memories." To the best of our knowledge, we are born with innate neural capacities for the full complement of seven basic emotions that are hardwired into the subcortical networks of all mammalian brains. We see this clearly in studies of animals that use techniques such as localized stimulation of specific brain regions. For example, if one provides artificial arousal in the form of electrical or chemical stimulation to the system that generates FEAR (a long pathway from amygdala to the center of the midbrain—the periaqueductal gray [PAG], described further in Chapter 5), even young, inexperienced animals will cower, and if the stimulation is sufficiently powerful, they will attempt to run away in terror. They will also rapidly learn to turn off such brain arousals and avoid places where they have had such experiences. Yet artificial stimulation does not provide any information about the environment. Thus the capacity to experience FEAR, as well as the other basic affects, is independent of any environmental experiences. In a sense, the ability to feel affects is largely "objectless"—initially only a few stimuli are able to turn on such Brain-Mind states, but this array of stimuli is rapidly expanded by learning (see Chapter 6).

FEAR is an inborn capacity of the mammalian brain. However, FEAR, just like all other basic emotions, rapidly gets enmeshed with world events as it comes to be regulated by learning and encoded in our conscious minds. Hence, at least in humans, our basic emotions become entwined with intentions and thoughts about the world (what philosophers, as we have noted, call "propositional attitudes") with the result that our appraisals of the world can then engender feelings.

Most basic emotions need not be expressed immediately after birth. Some, including CARE, LUST, and PLAY (more variable across species), come online long after others, such as SEEKING, RAGE, and FEAR. But all of these emotions have genetically hardwired neural substrates. In some mammals, the PANIC/GRIEF response becomes active early in life (as with herbivores that are born remarkably mature or *precocious*); in others, it becomes active later (as with most carnivores that are born very immature or *altricial*). In some others, such as laboratory rats that have been bred in laboratories for many hundreds of generations, certain emotional primes (indeed, perhaps only their behavioral expressions) have become vestigial because of a massive relaxation of natural (evolutionary) selection pressures. For instance, rats and mice do not have a robust separation call like most other mammals, perhaps because of the inadvertent selection of animals that could be housed individually without much distress. Their modest calls may simply be distress calls engendered by bodily stressors such as feeling cold. Because our genes control primary-process emotions, there can be great variability in the emotional temperaments of different species, as well as different laboratory strains bred for research, such as mice, of which there are thousands of variants, many with distinct personalities, some of them artificially created (Crawley, 2007).

Although the ability to experience affects is built into the brain, at birth humans and animals have unconditional or instinctive affective responses to only a few specific stimuli. Almost all animals are frightened by loud noises and by pain. Human babies cry if they are not held securely or are allowed to fall. And almost all young mammals cry quickly if they are left alone without their mothers, but this response takes some time to mature in many species, including dogs and humans. There are also some instinctual affective tendencies that are specific to particular species because of sensory specializations. For example, rats are inherently afraid of the smells of predators, such as cats or ferrets. Even if a rat has been raised in captivity and has never before been exposed to a predator, it will become wary and frightened if a bit of fur from a predator is placed in its cage. Smell is the specific instinctual trigger in this case, or in behavioral parlance, it is the *Unconditioned Stimulus* (or *Stimuli*, UCS) that evokes the *Unconditioned Response* (UCR) of fearfulness (which, if paired with any neutral cue, namely *Conditional Stimuli* [CS], can lead to *classical conditioning*—the generation of *Conditioned Responses* [CRs] as discovered by Ivan Pavlov,

who created the famous experiment where dogs salivated to the sound of a metronome that predicted food). While the behaviorists recognized that aversive UCS, such as predator odor or electric shock, can serve as "punishments" in many learning tasks, they could overlook as irrelevant the fact that UCRs, such as fearfulness, also have an internal feel to them. Other UCS could serve as "rewards" that would promote the learning of approach behaviors rather than avoidance behaviors. There has traditionally been little discussion, however, of any corresponding feelings underlying the logic of behavioral learning in animals. Of course, it is likely that rewards and punishments only work so well to control learning because they generate affective feelings in the brain. The spooky process of *reinforcement* may reflect the way feelings work in the brain.

The short list of conditionally arousing stimuli soon multiplies exponentially as people and animals undergo conditioning and other learning experiences in the ordinary course of life. Conditioning experiences, for example, allow animals to acquire an emotional response to a stimulus, which to them was previously neutral. For instance, if a cat wears a bell around its neck and a rat has a confrontation with that cat, the rat will soon learn to be afraid and run away when it hears the sound of a bell. More intelligent animals have a cognitive appreciation for cause and effect (often dramatically flawed, as we will see in Chapter 3) and for the passage of time. Humans can draw flexibly upon past learning in order to formulate behaviors that will enhance comfort and survival while decreasing chances of discomfort and death. When people go on a mountain hike, for instance, they frequently will have learned to take along a variety of safety devices—plenty of water, an extra jacket, sunscreen, waterproof matches, and so on—because they are intelligent enough to anticipate and appreciate the consequences of various possible changes in conditions that could become dangerous.

Affective responses, along with the explicit emotional behaviors we can see, are among the least well-studied aspects of the brain in all of neuroscience. Affects feel good or bad in a variety of specific ways. Sexual gratification, arising from our capacity for LUST, feels good in a rather different way from the joys of rough-and-tumble PLAY or the tender bliss of caressing, nurturing, and CAREing for one's infant. FEAR is an entirely different kind of emotional "pain" than frustrated RAGE; both differ from the PANICked misery of social isolation. And SEEKING things in the

world—whether safety, nuts, or knowledge—has a very special, energized, and, at times, euphoric feel to it but it can also create many negative events.

These diverse pleasant and unpleasant affects provide guidance for living due to the survival-enhancing advantages each of them has conferred over the course of evolution. Affects are ancestral memories of how effectively we play the game of survival and reproduction; these memories are passed down through the collected mindless "wisdom" of our genetic code. Interactions that evoke various pleasant affects—encounters with food, water, a mate, offspring, or playful friends—help animals to survive and reproduce. Life experiences that evoke painful affects—predators, rivals, chaotic weather, and so on—put life and reproductive capacity in jeopardy.

Thus raw affects provide the essential infrastructure for our most basic instinctual behavior patterns—approach and avoidance—without which we could not survive. Humans and other animals approach things that evoke pleasant affects, and they stay away from things that make them feel bad. Hence affective changes can *reinforce* new behavior patterns, although behaviorists never learned much about the brain process of *reinforcement* (a term that may mean, as just noted, little more than how "affects"—and not merely the *basic*, primary-process affects—work in the context of learning). Animals do not necessarily "know" or dwell on these feelings—the feelings may simply be raw *anoetic* experiences in most species. However, humans surely have many thoughts and ruminations about their personal experiences that can further elaborate affects, allowing *noetic* (factual knowing) and *autonoetic* (autobiographical time-travel) forms of emotional experiences (for a summary, see Vandekerckhove & Panksepp, 2009). The extent to which other mammals, even highly intelligent animals like the great apes and most carnivores, have such higher levels of cognitive (thoughtful, reflective) consciousness is surely a more difficult problem than the one we are addressing, which is the existence of raw affective-emotional experiences in all mammals.

THE TRIANGULATION OF STUDIES OF BRAIN, MIND, AND BEHAVIOR

Why are animal affects so important for understanding human well-being? Because understanding them provides us with knowledge of our own basic value systems—aspects of life that feel intrinsically good and bad. We

cannot study such processes at the fine neural level in the human brain. In order to understand affects across mammalian species, it is extremely helpful to use a triangulated method of research that focuses equally on our understanding of (i) the mammalian brain, (ii) the instinctual emotional behaviors of other animals, and (iii) the subjective states of the human mind. Such triangulations are the primary means by which we can investigate the neural underpinnings of affective life in our own species as well as in other animals (Panksepp, 1998a). This method can have great impact on the advancement of affective research-based understanding in general and on the practices of biological psychiatry and psychotherapy in particular. It also provides a way of understanding scientifically, for the first time, some of the experiences of other animals.

The first component of this triangulation method concerns brain systems and function. The physical brain must always be the primary component of rigorous neuroscientific research. Only when we know how the brain works can we achieve deep understanding of the behavioral and mental processes of animals and humans. In the general coverage of this book, however, we will not delve as deeply into the underlying neurological, neurochemical, and neurogenetic issues as we would in a professional scientific forum (for many of those details, see Panksepp, 1998a).

The second component is a careful study of animal behaviors, especially their natural (instinctual) behavioral tendencies—their unconditioned responses (UCRs). Abundant evidence now demonstrates that the brain networks that generate unconditioned emotional behaviors are, in fact, accompanied by affective experiences (conscious, unconditioned within-brain processes that can serve as “rewards” and “punishments” in learning tasks). Thus, we can further conclude that brain manipulations that arouse natural emotional behaviors in animals also induce the accompanying affective states. Of course, the brain could have been built in other ways. But the now well-established correspondence between raw emotional affects and instinctual behavioral expressions adequately demonstrates that affective experience is part and parcel of emotional arousal in all mammals, and probably most vertebrates.

The third component is psychological analysis, which preeminently includes human verbal self-reports about affective experiences. Human beings can talk about their feelings at great length. So if a given brain manipulation produces emotional behavior in animals, and if human beings

describe related affective experiences when they are stimulated in similar brain regions, then this complements the animal observations. Also, since there are abundant ways to determine whether animals are feeling something by their tendencies to avoid or pursue certain states of their nervous systems, we can at least be confident that they do actually have desirable and undesirable mental experiences. For instance, we can experimentally "ask" animals whether they will work for or avoid certain brain manipulations, such as applying electrical stimulations to specific brain regions, or whether they will return to or avoid places where they have had such brain manipulations. Their responses provide the answers we seek, especially when viewed alongside the verbal self-reports that humans in similar situations can provide.

In sum, at present the most compelling knowledge about how emotional feelings and other affects are organized in the mammalian brain comes from direct manipulations of specific brain systems. Although we cannot ask the experimental animals about the precise quality of their experiences, if their experimentally induced emotional behaviors are distinct, and humans report distinct emotional experiences when similarly aroused, we have *prima facie* evidence for a more resolved affective infrastructure in the brain than the simple global "positive" and "negative" affects espoused by many psychologists. We can also devise discrimination tests in animals to determine whether they distinguish certain feelings (e.g., Stutz et al., 1974) but that field of inquiry has barely begun.

The Critical Importance of Neurochemical Manipulations

In addition to localized electrical stimulation of the brain, specific chemicals can be applied to particular regions of the brain within animals to produce specific emotional behaviors. For instance, corticotropin-releasing factor (CRF)—the executive system for turning on the brain-body stress response—generates forms of FEAR (freezing and flight) and, we have good reason to believe, PANIC/GRIEF in mammals and birds, because CRF can dramatically elevate crying in response to social separation. If we are justified in concluding that changes in the animal's emotional behaviors indicate the animal's affective state, then we can assume that similar manipulation of the human brain would produce similar affective changes.

Although little work has been done with localized chemical stimulation of the human brain, the massive amount of such work on animals has abundant implications for how primary-process affects are generated in the human brain. Indeed, drugs that block the separation-distress system are at the forefront of new antidepressant development in biological psychiatry (for a complete overview, see Watt & Panksepp, 2009). There has been an enormous body of work with peripherally administered drugs that influence brain chemistries in specific ways. And the animal and human data line up remarkably well. For instance, all mammals typically get addicted to the same types of drugs. This knowledge is of great practical value because it allows direct neuro-pharmacological translations to be made between human and animal affective experiences.

We will not cover the diverse neuroanatomies and neurochemistries of the brain in any great depth here, but we will at least share a thumbnail sketch of current thinking. For example, in all mammalian brains, internal opiate-like transmitter chemicals that are called "opioids" (these are functionally similar to addictive drugs such as morphine or heroin) operate to transmit "information"—sometimes better envisioned as "states of being"—between nerve cells. For instance, beta-endorphin binds with what are called mu receptors (large "listening" molecules concentrated within the synaptic surfaces of nerve cells) in specific subcortical regions, to produce various desirable internal states—the pleasure of social companionship, or pleasing tastes and touches. Such internal opioid-sensing mu receptors can take away feelings of pain and send messages of pleasant satisfaction into the brain. As will be summarized in Chapters 8 and 9, the first subtle emotional satisfaction that was discovered to be controlled by opioids was the addictive feeling of love we experience when in the presence of those whom we care for and when we are emotionally secure and socially satisfied (Panksepp, 1981a). More recently, such chemistries have been found to mediate our addiction to sweets (Avena et al., 2008). There are many other affective examples we will highlight throughout this book. Indeed, many natural pleasures can counteract drug addictions. One of the most remarkable findings is that motherhood, which "lights up" many of the same subcortical brain regions as the effects of cocaine do, is as attractive as such drugs of abuse (Ferris et al., 2005).

The binding of transmitters to their specific receptors occurs in "key" and "keyhole" fashion, where relatively small transmitter molecules serve as the

keys and the much larger receptor molecules serve as the keyholes to "locks" that control neural firing. In the emotional regions of the brain, such molecules can unlock our feelings. In less poetic terms, specific key-like molecules bind with specific receptor molecules, which cross many synapses (the information transfer gaps between neurons) and can initiate complex chemical cascades that result in several distinct types of emotional arousal. It is important to note that many of these emotional chemistries act in global ways in the brain—they are released in many brain regions to bring various network functions under the orchestration of one emotional conductor. It currently appears that some of the larger transmitters, constructed from chains of many amino acids—the neuropeptides—provide considerable specificity to the distinct emotional tendencies and feelings we can experience.

Neuroscientists have not mapped out all of the neurological steps between neurochemical system activities and emotional expressions. That will take a long time. But it is now quite clear that certain brain chemicals, especially neuropeptides, can produce highly predictable emotional-feeling responses. For instance, see Figure 9.3 in Chapter 9 for the power of corticotropin-releasing factor (a transmitter molecule composed of 31 amino acids) in activating the type of crying that reflects separation distress within the brains of young birds.

As will be extensively discussed in Chapter 9, just the opposite feeling emerges when a small amount of an opiate binds with mu receptors. This starts a chemical cascade that produces emotionally contented responses. Animals appear happy and relaxed, and they seem quite self-satisfied. Even if placed in isolation they exhibit no motivation to cry and do not appear sleepy in the least; in fact, at the very low doses needed to quell their emotional distress, these animals are often more active. They play more. If the dose is larger, the animals do become sleepy. At high "pharmacological" as opposed to "physiological" doses, they exhibit a catatonic, almost comatose state. However, the tiny doses that simply reduce crying do not produce any such effects, except in certain neonatal "preemies," such as the fetus-like newborn rat, in whose underdeveloped nervous systems such small doses have much bigger brain effects. If we assume that the contented behaviors following tiny doses of opiates reflect contented feelings in animals, then given the similarity of subcortical neural networks and functions across mammalian species, we can assume that people will have

similar responses. And indeed they do. This is well known for all addictive opiates. When people are under the influence of opiates, they say that they feel soothed and comforted. This is because their PANIC/GRIEF system is less active, and it helps explain why lonely, disenfranchised people are more likely to get hooked on such drugs. Indeed, opiates would be almost perfect antidepressants if they were not so addictive. There are now much safer, much less addictive opiates (e.g., buprenorphine) that can be used to treat depressions that have resisted other therapies (Bodkin et al., 1995). Because of an abundance of animal research, we can now generate comparable ideas for an enormous number of neuropeptides and even smaller transmitter molecules that control a variety of emotional states.

One such molecule is dopamine, which is synthesized from a single amino acid, tyrosine. This little transmitter molecule prompts animals to engage in enthusiastic investigations of their environments (Chapter 3). Such affective and behavioral arousal can be achieved by the administration of drugs called "psychostimulants," which increase dopamine release in the brain. Dopamine then acts as the key that binds with dopamine receptor keyholes (there are five major varieties of dopamine receptors, each with slightly different functions). Many of the stimulant molecules that increase dopamine activity at synapses—for instance, amphetamines and cocaine—are also highly addictive in all mammals, although they evoke different feelings than opiates.

Whenever there is an increased release of dopamine in the brain, animals are more aroused in a distinct type of way. They become more eager and inquisitive. As detailed in Chapter 3, when this happens animals exhibit excited SEEKING behaviors that can anticipate *all* kinds of attractive events in the environment. The lateral hypothalamus (LH) is one brain structure that becomes aroused when animals are in this excited state. Others are the nucleus accumbens further up in the brain and also the medial frontal cortex, which is even further up. All these brain regions are connected by a remarkably large pathway that connects the lower and higher areas of the brain, known as the medial forebrain bundle (MFB), which contains many, many distinct neurochemical networks, some of which operate with dopamine. Direct electrical stimulation of each of these brain regions, all along the MFB, also produces such excited responses. Animals love to self-activate such electrode sites—and they readily begin to self-stimulate their own brains in compulsive, addictive ways.

It no longer comes as a surprise that brain dopamine systems are essential intermediates for practically all forms of drug addiction as well as all the natural appetites of mammals. When the MFB in people is stimulated, either by dopamine or by an electrical current, they report euphoric feelings of excitement, interest, and anticipation. They can become manic. Animals readily return to locations where they received such experiences. Human subjective reports allow us to surmise that animals experience similar affects. When activity in this brain system is dampened, animals accordingly appear depressed, and humans report feeling psychologically sluggish, with no enthusiasm for anything.

Exogenous chemical keys (those introduced from outside the body) that fit into receptor keyholes but do not initiate changes in the firing rates of receiving neurons, but in fact disable them for a while, are called receptor *blockers* or *antagonists*. For example, chemicals such as naloxone and naltrexone can block mu receptors. Naloxone and naltrexone also inhibit the effects of external opiates such as morphine and heroin as well as some of the *endogenous opioids*—opiate-like chemicals that are produced within the brain. When endogenous opioids are blocked from binding with mu receptors, animals appear more on edge, and they do not seem to like the psychological effects. Human beings report similar undesirable affects, but often the changes are subtle, requiring long-term administration of large doses. In the same way, key molecules that block the effects of dopamine can induce lethargy and depression in both people and animals. The large variety of synaptic receptor antagonists that have been developed have been especially useful in studying the psychological effects of various endogenous brain synaptic neurochemistries.

On the other hand, when an exogenous agent binds with a receptor and produces the same result as an endogenous brain chemical, the exogenous agent is called an *agonist*. Opiates found in certain poppy plants (*Papaver somniferum*) produce the similar affective feeling as endogenous opioids do. Both are emotionally comforting. Thus, opiates act as agonists for endogenous opioids. There also are a large number of other receptor agonist drugs that can enhance the effects of many of the specific endogenous chemicals of the brain. For example, both cocaine and methamphetamine facilitate dopamine activity by enhancing the availability of dopamine at synapses.

There are many other drugs that work in all mammals to modify how rapidly neurotransmitters are synthesized or degraded, giving neuroscientists an incredible set of tools for triangulating among neural, mental, and behavioral analyses of emotional states. All of these drugs can be used locally within the brain in animal studies. One can also directly measure the release of a large number of neurochemicals while animals are behaving emotionally. From such work we know that dopamine is released under practically any condition that makes the animal behaviorally excited. Other drugs produce distinctly different behavioral effects and feelings by acting on other neurochemical systems.

Before proceeding, let's deal with an issue most readers will wonder about. Do even "lower" animals, like invertebrates, have affective feelings? Will they also pursue drugs that are addictive for mammals? Many will. We now know that crayfish develop preferences for places where they have been given either psychostimulants or opiates (Panksepp & Huber, 2004; Nathaniel et al., 2009). This suggests that affective experiences go much deeper in BrainMind evolution than just at the mammalian level of development. But there can be other explanations, and the vastly different nervous systems of invertebrates do not allow us to readily triangulate between their behaviors, brain mechanisms, and mental feelings as we can with other mammals. Thus, we will not dwell on these interesting issues here, but we must always keep the door open to reasonable possibilities that few have experimentally considered.

Modern Brain Imaging of Higher and Lower Brain Functions

Although neuroanatomical and neurochemical analyses are essential for the cross-species triangulation method, the detailed study of animal behavior, especially the natural emotional behaviors that animals themselves spontaneously exhibit, is presently a crucial element in affective neuroscience. Perhaps in the future we will know enough about brain function to be able to routinely predict affective experience from the "pictures" that we see, using modern human brain-imaging devices (e.g., positron emission tomography [PET] and fMRI). But this is not yet possible in either humans or animals. However, some progress is being made. For instance, by contrasting brain regions such as the nucleus accumbens,

which receives abundant dopamine messages, with other regions such as the insula, which mediates feelings of disgust, investigators have shown that when shopping, people will decide to buy things that "light up" their nucleus accumbens but will have little desire to purchase something if it activates the insula (Knutson & Greer, 2008).

Unfortunately, some of the techniques such as fMRI require humans and animals to be completely still, which is behaviorally incompatible with strong levels of emotional arousal. PET can be used more readily; researchers can even inject positron emitting imaging molecules *before* putting animals into brain scanners. PET has been used to monitor brain changes during "jealousy" in monkeys (Rilling et al., 2004), but this technique is much too expensive for routine animal research. While fMRI is being used effectively in increasing numbers of animal studies, again the animals have to be completely immobilized to obtain any useful images.

It must be recognized that most human neuroimaging studies provide a better view of the higher, neocortical parts of the brain, mainly because those regions of the brain are much bigger than the ancient subcortical structures and also because they are metabolically more active. It is often hard to visualize subcortical regions where cells fire less rapidly or simply change their patterns of firings (e.g., the dopamine neurons discussed in Chapter 3). Also, many nearby systems that can produce conflicting messages overlap more extensively. Furthermore, even when visualization of subcortical regions is possible it does not always render a clear picture of the neural details of what is going on, because neuroimaging techniques monitor overall regional brain activity (for example, blood flow or sugar consumption).

The underlying assumption is that brain function requires energy in the form of oxygen-mediated (aerobic) metabolism; therefore, local blood flow, or oxygenation, or glucose levels change as a reflection of regional brain activity. However, the energy expenditure and blood flow can be a reflection of neuronal *inhibitory* signals as well as of excitation—the generation of neuronal firings that produces inhibition at downstream synapses also requires the expenditure of energy. Therefore it is not even possible to know, for sure, if the many "lights" that seem to turn on in the human brain reflect brain excitation (increased firing) or inhibition (reduced firing downstream). In addition there are a host of statistical pitfalls, too complex to consider here, that can result in a false impression of the

strength of the effects that are seen (e.g., Vul et al., 2009). The worst of it for the uninitiated is that incredibly small but consistent brain signal changes are converted into arbitrarily intense colors on monitors, which easily fool the unwary into believing that the brain changes are larger than they really are. From the perspective of affective neuroscientists, perhaps the most troublesome aspect is that these techniques are not well designed to envision the most ancient regions of the brain, where the power of neurochemistries is often more influential than the absolute changes in neural firings. Still, the data being obtained with human brain imaging *are* quite spectacular.

So while the observation of animal behavior may seem simplistic in comparison to state-of-the-art neuroimaging techniques, animal behavior provides remarkably good and useful scientific data because an animal's primary-process (instinctual) emotional behaviors are probably accurate reflections of its primary-process affective experiences. Human brain imaging is rather poor in illuminating the primary-process emotions of humans. Human beings are able to think about their affects and to inhibit their emotional behaviors precisely because they are so intelligent. In general, deeper emotional parts of the brain arouse the surface cortical regions that control our cognitions, while the higher cortical layers often inhibit and regulate the affective arousals that emerge from below. Human beings, who have prodigiously large neocortices, are often able to inhibit the behaviors that typically attend emotional arousals. For example, frightened people can often feign calm. Indeed much of human social life involves some degree of affective inhibition and obfuscation. We do not grab for things that we want, we tend to diminish feelings of triumph and defeat, and we try to appear friendly even when we are irritated. Animals usually do not have this self-generated ability to inhibit and disguise their emotional responses. When a rat or a monkey experiences an affect, its behavior usually reflects the way it feels. Thus no modern brain imaging will ever replace the careful study of animal behavior in our quest to understand how emotional behaviors and affective feelings are created in brains.

AFFECTS DO NOT FEEL LIKE ANYTHING ELSE

If affects are not cognitive read-outs of the changing physiology of the body, and if they emanate from deep noncognitive parts of the brain, then what do affects feel like? We maintain that affects do not feel like anything else. They are primary phenomenal experiences that cannot be adequately explained just in terms of accompanying changes in the body, even though there are bound to be many distinct bodily feelings during emotional arousal. Much of the intermingling of emotional feelings and physiological arousal could be because the primary-process emotional systems are situated in the same brain regions that regulate the activities of our viscera, our hormonal secretions, and our capacities for attention and action.

To be sure, bodily responses can also influence emotional arousal. For example, anger is invariably attended by heightened blood pressure. Blood pressure also exerts influence on affect, as any chemical agent that raises blood pressure will make an angered person or animal feel more enraged. This is because pressure receptors in arteries can directly facilitate RAGE circuits in ancient visceral brain regions (i.e., the parts of the brain that represent our internal bodily organs). However, the artificial elevation of blood pressure does not produce anger in a person or animal who is not already irritated. Thus it does not appear that affects simply reflect peripheral emotional physiology. Affects are, as we have already stated, ancient brain processes for encoding value—heuristics of the brain for making snap judgments as to what will enhance or detract from survival.

Those who maintain that language is the hallmark of affect are even further off the mark. Words are best suited for explaining the workings of the world around us. Words can explain that the George Washington Bridge connects New York and New Jersey. Words can tell you how to bake a cake. But words cannot explain primary experiences. Words cannot even explain the primary perceptual experience of seeing the color red. Words like “scarlet,” “crimson,” or “ruby” do not describe anything. They are mere labels or symbols for the common experience of seeing variations of redness, which is strictly a subjective brain function. One could use any symbol, including a nonverbal one, as a label for the experience of seeing red. “Red” has no intrinsic meaning, but the experience of redness does—it signifies some of the most exciting things about life, from the ripeness of fruit to the passion of sex and of spilled blood. Words cannot describe the experience of seeing the color red to someone who is blind.

Words do not describe affects either. One cannot explain what it feels like to be angry, frightened, lustful, tender, lonely, playful, or excited, except indirectly in metaphors. Words are only labels for affective experiences that we have all had—primary affective experiences that we universally recognize. But because they are hidden in our minds, arising from ancient prelinguistic capacities of our brains, we have found no way to talk about them coherently.

The science of how these systems connect up to the higher conscious abilities of humans is still largely a task for the future. However, because of the importance of these systems for clinical psychiatric phenomena, we will briefly address these higher cognitive aspects in each of the chapters devoted to the "big seven" emotional affects.

AFFECTIVE TAXONOMY: THE SEVEN BASIC AFFECTIVE SYSTEMS

So far, the triangulation method has revealed the existence of seven basic systems, which are homologous throughout all mammalian species. We do not know when animals first began to have affective experience, but current research indicates that some affects exist already in nonmammalian vertebrates. For example, isolated young birds experience separation distress in much the same way as isolated young mammals (Chapter 9). Also, as briefly noted in the earlier crayfish example, there is suggestive evidence that some invertebrates have affective experiences.

It is reasonable to believe that the full complement of seven basic emotional systems, in rudimentary form, had already evolved with the advent of mammalian life. This is because of the clear and distinct emotional nature of birds. In all mammalian and avian species, similar chemicals arouse and inhibit these systems; to the best of our current knowledge, each system generates a distinct affective experience. But there are many overlapping aspects among these systems; for instance, the SEEKING system participates in most of the other systems. And all of the systems are regulated by general-purpose brain arousal regulators, such as serotonin, norepinephrine, and acetylcholine. These confront us with complexities that cannot be avoided, just as our words will create complex and overlapping meanings.

Language, especially arcane technical language, cannot adequately describe affects. So we will use common vernacular terms—simple words—as labels for the seven emotional systems. To avoid confusion, however, we will (as already noted) use all capital letters in order to emphasize that we are speaking about distinct brain systems from which the particular affects and emotions emanate; we are not *simply* speaking of the common feelings ordinarily denoted by those words.

It is also important to be clear that the knowledge we have gained through the triangulation of affective neuroscience does not explain the complexity of *whole* emotional experiences as they occur in real life. While part and whole confusions are rampant in much of cognitive neuroscience (Bennett & Hacker, 2003), we hope to avoid them here. We are speaking about specific neural systems that are important, integral parts of the psychological wholes of our lives. We do not claim to be speaking about emotion as a total entity. Science is limited to studying parts of phenomena.

Only theoretical narratives manage to unite the parts into an understandable whole. For example, Darwin collected fossils and observed life on different islands. The diverse items of data he observed were the scientific parts that enabled him eventually to devise his holistic theory of evolution (survival of the fittest). We do not yet have a totally unified theory of affect—in which the integration of higher and lower brain functions can be understood in neural and psychological detail. To make such advances possible, far more data remain to be collected. But what kinds of data will be most informative? Perhaps the most important are the neural mechanisms of distinct emotional behaviors, and their rewarding and punishing effects. Meanwhile, the great success of modern neuroscience has generated more and more fancy analytical tools, looking at ever finer aspects of neural activities, often ones that are impossible to apply, with insightful clarity, to global psychological questions. Thus we have an abundance of knowledge about neural mechanisms that are looking for functions; this is a rather peculiar, but intellectually stimulating, state of affairs. For instance, what are the functions of "silent synapses"? To stand at the ready, waiting for the right neural conditions for learning (see Chapter 6)? This embarrassment of technological riches also has its downside. It promotes a "ruthless reductionism" where a study of neural mechanisms counts but experiences they generate do not. We do not support such neglect of the mind here.

In this book, we will focus on the substantial empirical and theoretical advances that have been made possible through the identification of the seven emotional brain substrates that reliably evoke distinct emotional behaviors and produce affective experience in all mammals that have been studied. We do not claim that these seven constitute an exhaustive list. More may be discovered. Furthermore there is much to be learned about the different chemicals that regulate these systems or parts of the systems. We also do not yet understand precisely how affects and other mental processes actually arise from the fine intricacies of the brain. Our approach does, however, encourage new ways of considering such difficult neuroscientific and phenomenological issues. This can be pursued because we now do know much about the essential brain regions and processes, especially some of the key neurochemistries.

Toward the end of this book we will present a novel, perhaps revolutionary, hypothesis for the generation of affect—one that relies on our capacity to envision a "core-SELF," encouraging us to contemplate the ancient neurobiology of "the soul" (Chapter 11)—that provides a center of gravity, laid out in emotional-movement/action networks, for the primal emotional feelings to emerge from brain activities. Here, theory (supported by some provocative data) is grasping for relatively intangible aspects of mind that remain to be adequately explored with neuroscientific tools. However, at present, we can be confident that arousal of one or more of the seven emotional systems is a necessary condition for the generation of affect in mammals. Future investigators will have to work out many, many additional details of how affect actually arises within the brain, and how, in order to work properly, such brain functions synergize with the rest of the body.

Although words cannot describe these seven basic affects fully, we will do our best, sometimes resorting to physiological correlates in order to literally flesh out their meaning. Here we provide a synopsis of the "big seven". For a fun depiction of these primal emotional systems, as well as some higher emotional complexities, see Figure 1.7.

1. The SEEKING, or expectancy, system (discussed in Chapter 3) is characterized by a persistent exploratory inquisitiveness. This system engenders energetic forward locomotion—approach and engagement with the world—as an animal probes into the nooks and crannies of interesting places, objects, and events in ways that are characteristic of its species. This system holds a special place among emotional systems, because to some extent it plays a dynamic supporting role for all of the other emotions. When in the service of positive emotions, the SEEKING

system engenders a sense of purpose, accompanied by feelings of interest ranging to euphoria. For example, when a mother feels the urge to nurture her offspring, the SEEKING system will motivate her to find food and shelter in order to provide this care. The SEEKING system also plays a role in negative emotions, for example, providing part of the impetus that prompts a frightened animal to find safety. It is not clear yet whether this system is merely involved in helping generate some of the behaviors of negative emotions, or whether it also contributes to negative feelings. For the time being, we assume it is largely the former, but that the positive psychological energy it engenders also tends to counteract negative feelings, such as those that occur during FEARful flight and the initial agitation of PANIC/GRIEF. For this reason, animals may actually find fleeing to be in part a positive activity, since it is on the most direct, albeit limited, path to survival.

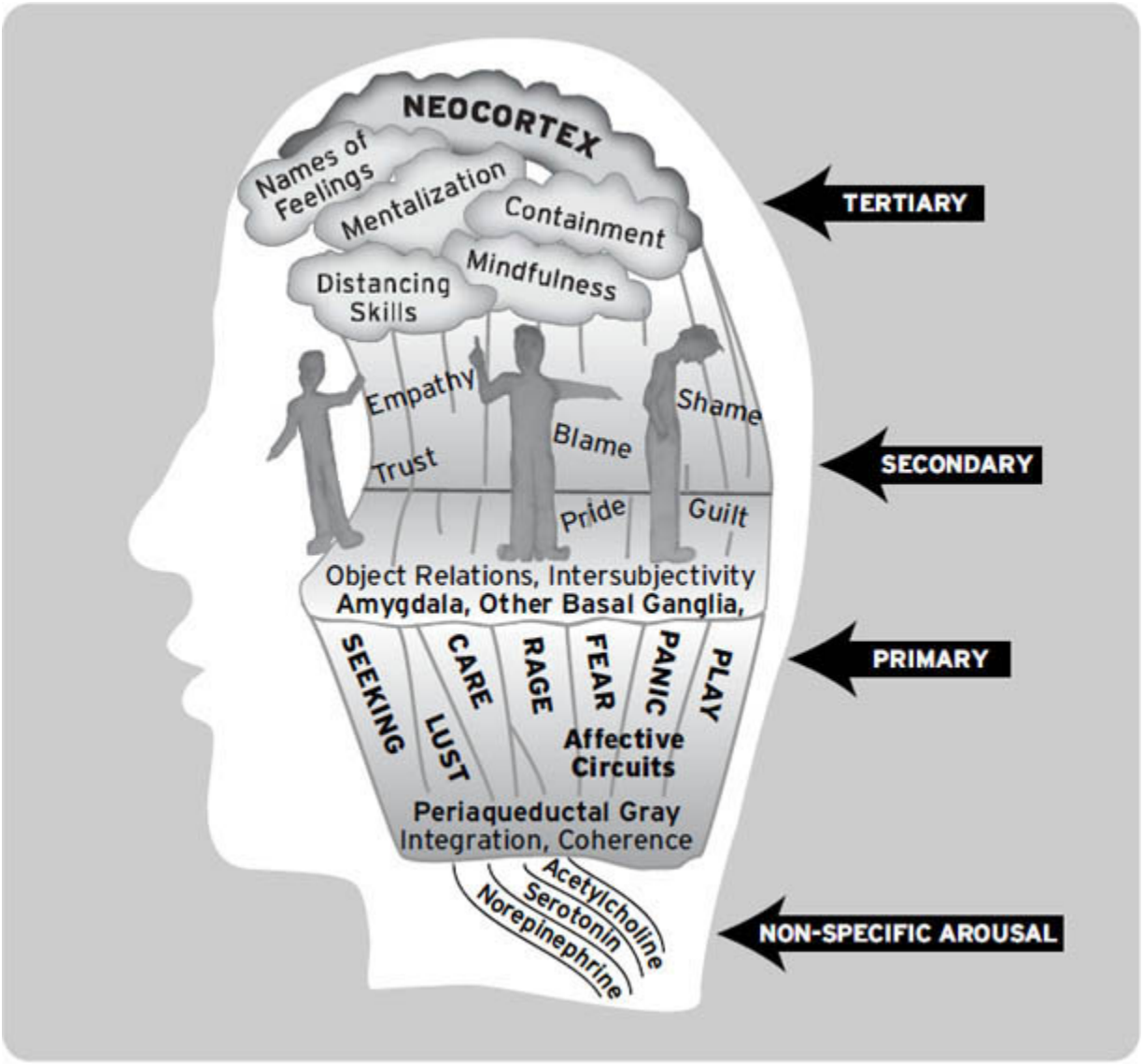

Figure 1.7. A cartoon of the primary-process emotional systems and their various secondary- and tertiary-process consequences. This figure was

adapted from a piece of art kindly drawn for this book by Sandra Paulsen and is used with her permission.

2. The RAGE system (see Chapter 4), working in contrast to the SEEKING system, causes animals to propel their bodies toward offending objects, and they bite, scratch, and pound with their extremities. Rage is fundamentally a negative affect, but it can become a positive affect when it interacts with cognitive patterns, such as the experience of victory over one's opponents or the imposition of one's own will on others who one is able to control or subjugate. Pure RAGE itself does not entail such cognitive components, but in the mature multi-layered mammalian brain (Fig 1.4), it surely does.

3. The FEAR system (see Chapter 5) generates a negative affective state from which all people and animals wish to escape. It engenders tension in the body and a shivery immobility at milder levels of arousal, which can intensify and burst forth into a dynamic flight pattern with chaotic projectile movement to get out of harm's way. If, as we surmised above, the flight is triggered when the FEAR system arouses the SEEKING system, then the aversive qualities of primary-process FEAR may be best studied through immobility "freezing" responses and other forms of behavioral inhibition, and reduced positive-affect, rather than flight.

4. When animals are in the throes of the LUST system (see Chapter 7), they exhibit abundant "courting" activities and eventually move toward an urgent joining of their bodies with a receptive mate (Figure 7.1), typically culminating in orgasmic delight—one of the most dramatic and positive affective experiences that life has to offer. In the absence of a mate, organisms in sexual arousal experience a craving tension that can become positive (perhaps because of the concurrent arousal of the SEEKING system) when satisfaction is in the offing. The tension of this craving may serve as an affectively negative stressor when satisfaction is elusive. LUST is one of the sources of love.

5. When people and animals are aroused by the CARE system (see Chapter 8), they have the impulse to envelop loved ones with gentle caresses and tender ministrations. Without this system, taking care of the young would be a burden. Instead, nurturing can be a profound reward—a positive, relaxed affective state that is treasured. CARE is another source of love.

6. When overwhelmed by the PANIC/GRIEF (also often termed "separation distress") system (see Chapter 9), one experiences a deep psychic wound—an internal psychological experience of pain that has no obvious physical cause. Behaviorally, this system, especially in young mammals, is characterized by insistent crying and urgent attempts to reunite with caretakers, usually mothers. If reunion is not achieved, the baby or young child gradually begins to display sorrowful and despairing bodily postures that reflect the brain cascade from panic into a persistent depression. The PANIC/GRIEF system helps to facilitate positive social bonding (a secondary manifestation of this system), because social bonds alleviate this psychic pain and replace it with a sense of comfort and belonging (CARE-filled feelings). For this reason, children value and love the adults who look after them. When people and animals enjoy secure affectionate bonds, they display a relaxed sense of contentment. Fluctuations in these feelings are yet another source of love.

7. The PLAY system (see Chapter 10) is expressed in bouncy and bounding lightness of movement, where participants often poke—or rib—each other in rapidly alternating patterns.

At times, PLAY resembles aggression, especially when PLAY takes the form of wrestling. But closer inspection of the behavior reveals that the movements of rough-and-tumble PLAY are different than any form of adult aggression. Furthermore, participants enjoy the activity. When children or animals play, they usually take turns at assuming dominant and submissive roles. In controlled experiments, we found that one animal gradually begins to win over the other (becoming the top dog, so to speak), but the play continues as long as the loser still has a chance to end up on top a certain percentage of the time. When both the top dog and the underdog accept this kind of handicapping, the participants continue to have fun and enjoy this social activity. If the top dog wants to win all the time, the behavior approaches bullying. As we will see in Chapter 10, even rats clearly indicate where they stand in playful activity with their emotional vocalizations: When they are denied the chance to win, their happy laughter-type sounds cease and emotional complaints begin. The PLAY system is one of the main sources of friendship.

To reiterate, these seven systems are considered emotional systems because the arousal of each produces robust visceral, behavioral, and affective responses. For example, the hormone oxytocin, along with some other chemicals, plays a crucial role in generating maternal behaviors within the CARE system, while also reducing separation distress from the PANIC/GRIEF system. Under normal conditions, a substantial oxytocin cocktail is generated endogenously at the end of pregnancy. It induces uterine contractions during labor and encourages milk letdown following the birth. Both of these responses are visceral components that occur when the CARE system is aroused. There is a psychological bonus in the brain, however. Animals become both less aggressive and more confident and nurturant when their brains are awash in oxytocin.

If a virgin rat is injected with oxytocin, and several other physiological changes transpire, she will exhibit arousal of CARE behaviors and feelings. She will look for pups to nurture; she will start to build nests for them; she will hover over them to provide warmth; and she will gather them up when they stray. These are all typical CARE behaviors one sees in postpartum mother rats. We know from verbal reports that postpartum human mothers, whose brains secrete a similar oxytocin cocktail, feel tenderness and strong protective impulses toward their babies. These are the affective responses that occur when the CARE system is aroused. But is oxytocin, a hormone that is released when babies nurse but which can also be elevated by various stressors, the main cause? Human research can resolve this question, but only at the tertiary-process level of mind. Might animal research on primary processes help provide critical clarity about the primal affective principles? Let us consider this possibility in some detail.

OXYTOCIN AND SOCIAL EMOTIONS—LOVE OR CONFIDENCE?

Work with direct brain injections of oxytocin in animals has been proceeding for three decades, ranging from better maternal care and mothers bonding to infants (Kendrick, 2000) to infants bonding to mothers (Nelson & Panksepp, 1996). And such lines of research have led directly to abundant work with humans.

Currently, fascinating findings about intranasal oxytocin effects in humans continue to emerge at an ever-increasing pace, and our text, finished in August of 2010 will not reflect all the very recent activity. Because of all this interest, in the popular imagination, oxytocin has become almost equivalent to "the love molecule": When we Googled "oxytocin love" on the web there were 205,000 hits, most of them lightweight hype or marketing, even though the scientific research that has supported such conjectures has been growing. But to this day there is practically no compelling evidence that oxytocin *robustly* elevates positive moods, the way many, many addictive molecules can do. Shouldn't it, if it was the mediator of love? There is no solid evidence that it is dramatically rewarding to animals. Indeed, if it were found to consistently promote positive moods under certain conditions, then one could even surmise that the effect may have been due to oxytocin-facilitating opioid activity in the brain (Kovács et al.,, 1998), which would be in line with a better supported theory of social attachments, and by extension companionate love, being a brain opioid-mediated process (Panksepp, 1981a, 1998a).

Still, in many experiments oxytocin does promote various pro-social behaviors and attitudes in animals and humans. Among humans, it increases the willingness to trust others in economic exchanges (see Meyer-Lindenberg, 2008). When couples are discussing topics and there are differences of opinion, the ratio of positive interactions (eye contact, interest, emotional self-disclosure, validation, caring, nonverbal positive behavior) compared to negative ones (criticism, contempt, defensiveness, domineering behavior, belligerence, stonewalling, nonverbal negative behavior, interruption) went up significantly (Ditzen et al., 2009), and so forth (Heinrichs & Domes, 2008). In other words, under the right conditions (with someone you already love) oxytocin makes us more pro-social—more tolerant and friendlier. However, we should recall that oxytocin systems are

all deeply subcortical, quite low and ancient in the brain, so clearly its primal function is not to control such higher cognitive activities like romantic love and calculations of others' trustworthiness. Thus, the effects must first of all be explained by changes in some type of primary-process brain mechanism. So one key question is, do pro-social friendly feelings go up following oxytocin administration, or was there only a reduction of stressful anxiety-like feelings that sometimes emerge during interpersonal encounters? In our hands, one of the strongest and most replicable preclinical effects ever seen is a reduction of separation distress (Panksepp, 1992). See Figure 1.8 for a sampler (this constitutes about 5% of the data we have collected on this measure). From this vantage, we might anticipate that without normal oxytocin secretions, mothers are susceptible to post-partum depression, and as we copy-edit this book, a recent paper suggested just such a relationship (Skrundz, et al., 2011).

Recently, a series of studies have appeared that question even the more level-headed "pro-social" conclusion. For instance, in economic games where one can win or lose to imaginary (computer-based) opponents, if a competitor happens to lose, oxytocin will increase gloating. If the virtual opponent receives more points than you, it increases envy (Shamay-Tsoory et al., 2009). Now this is not very pro-social. Thus, this maternal-behavior facilitating peptide has a prickly side. And when others have tested folks in settings where altruism could be exhibited, the feelings are mixed. It does tend to promote cooperative fellow feelings toward your in-group—your friends—but it does the reverse for out-group strangers, where it increases defensive aggression (De Dreu et al., 2010). These are not the kinds of effects one would expect from a pro-social love molecule.

So where is it the catch? What is the actual affective change in the brain that can lead to such diverse effects? The transmitter pathways for oxytocin are pretty limited, with no indication of how it could produce such changes directly at the tertiary-process level. Might there still be a single type of primary-process affective shift that could explain these, and other, perplexing human results? Perhaps, but no one has come up with a compelling proposal. Take another paradox: Oxytocin, when given to those who have borderline personality disorders (BPD), will decrease trust and the likelihood of cooperative responses (Bartz et al., 2010). We would like to suggest a solution that may bring these divergent results together, based on oxytocin effects we have observed in birds.

In the quail species, strange males are especially intolerant of each other. They peck each other's heads until one gives up and simply submits to the other's pecking. In this way, the quails forever know where they stand in the pecking order, as long as the dominant animal still thrives. So, we wondered, what happens if very young birds who are strictly operating with primary processes, receive oxytocin directly into their brains (into their cerebrospinal fluid, as is commonly done in animal research). We tested infant domestic chicks, and when they were separated from the security of their flock, the obvious effect was that they hardly cry (Figure 1.8). Furthermore they also exhibit more yawning. They shake their heads more and exhibit more wing flapping (Panksepp, 1992). If tested in groups, the animals with injected oxytocin show much more wing flapping than when tested alone, which seems to indicate that they are "feeling their oats"—they are generally more confident (the yawning and head shaking were not socially facilitated).

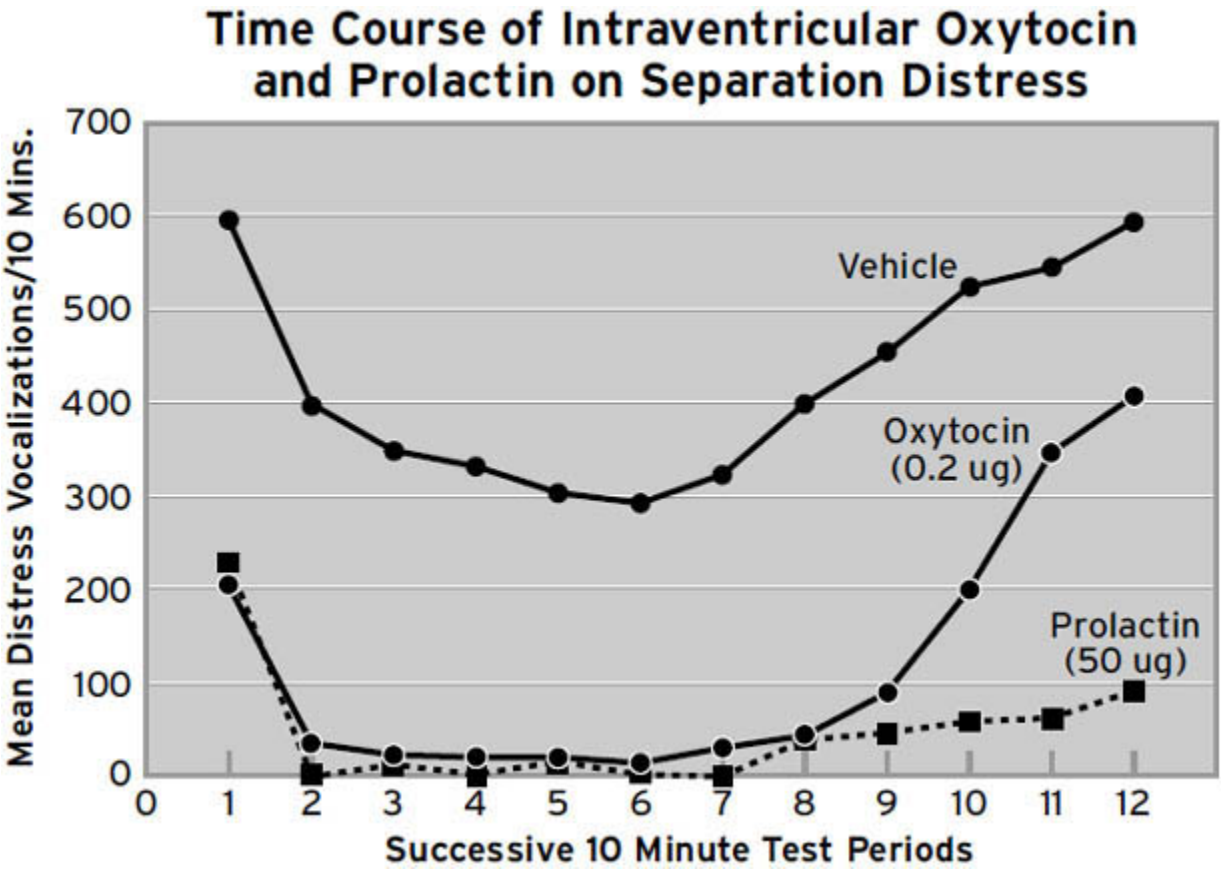

Figure 1.8. The effects of intraventricular oxytocin and prolactin on the separation distress calls of 5- to 6-day-old chicks that were socially isolated from their flock for a 2-hour period. These dramatic effects on crying were produced without any apparent sedation, just as with low doses of opioids that stimulate mu receptors.

So we wondered what would happen to social dominance in quail when one animal got an oxytocin-like boost. Well, to our surprise, the quails with this boost really got their heads pecked by the other birds. Perhaps they were more submissive but alternatively, perhaps they simply became more tolerant of the other birds' "bad behavior." If this amount of head pecking happens to a normal quail, it is subsequently super-submissive. But when we tested identical pairs again the next day, the quail that had seemed so submissive now came back like gangbusters and became the winner (Riters and Panksepp, 1997). This kind of "turning of the tables" practically never happens in normal quail. Once you have become the loser, you keep that position. So why did an oxytocin-like boost on the initial day, when one was losing, allow the "submissive" bird to become the winner the next day? Had the bird just forgotten? If that were the case it should have been an even match at best. In fact, the quail that had lost came back stronger, suggesting that it had simply been peaceful the previous day but was still feeling pretty strong. Can we say "confident"? That certainly seemed to be a reasonable hypothesis.

So how does one test for such a subtle affective construct in animals? How about putting a group of young birds (domestic chicks in the unpublished study we did) under a bucket in a large room, in order to see how far from each other they move to explore the new room. Indeed, normal young chicks tend to hang together and will move as a tight-knit group. But when we put oxytocin into their brains, they spread out more loosely, as if they were less pro-social or, alternatively, they were more "confident" with diminished anxiety. We had already known from the mid-1980s that this molecule is super strong in reducing separation anxiety in birds (Figure 1.8).

Perhaps oxytocin can increase confidence in animals. Would this explain the human studies? It seems reasonable that when you are confident, you would be more secure in economic transactions. Wouldn't you be likely to

be more friendly and tolerant—less defensive—with your spouse when discussing conflicting ideas? If a stranger won less money than you in a wager, would you not be more willing to gloat if you felt more confident? If you won less, would you not be more willing to admit that you are a bit envious in the higher reaches of your mind, and be more willing to express it? If you had the chronic insecurity of BPD, might it not be reasonable that a confidence-boosting dose of oxytocin would shift you toward a sense of independence, and you might be more willing to assert your views, as opposed to remaining in the grip of your chronic dependency needs? Thus, what we need is a good psychological test of confidence. Meanwhile, there are already some modest data that show that oxytocin can decrease social anxiety disorders (Guastella et al., 2009).

Thus, with a small shift in affective focus, to a very fundamental aspect of social living, all of a sudden, the perplexing diversity of findings in human studies begins to make sense. Surely confidence is a very important trait for competent motherhood—a can-do attitude serves moms well when personal responsibilities have increased dramatically. If such an interpretation is likely, here is a prediction, based on the well-known fact that lots of people are scared of public speaking. If oxytocin increases social confidence, then performance anxiety should decrease. With the study of primary-process systems in animal brains, and oxytocin circuits, we can make some remarkable predictions to psychological changes we might expect in human beings. For instance, oxytocin should increase our tendency to explore the eyes of another person, to try to read their mind, because you are feeling more secure. And in fact, it does that, especially if you are depressed, even though a single intranasal dose of oxytocin does not significantly improve depressive symptoms (Pincus et al., 2010). Oxytocin has even alleviated some of the symptoms of schizophrenia (Rubin et al., 2010). And the evolutionary span of such molecules is vast. Even fish show various social effects, including faciliation of monogamous mating behaviors, when they have these kinds of molecules infused into their brains (Oldfield & Hofmann, 2011).

So is oxytocin a love molecule or one that reduces anxiety and promotes confidence? The smart money should be on the latter. Perhaps you will have more sex and more children if you are confident in love.

AFFECTS AND EVOLUTION

When we think about evolution, we usually refer to the ways that animal species have physically changed and developed over the ages. When we speak about affects, however, we usually refer to the *mind*, which is commonly thought of as a nonphysical entity, which is nonsense. Mind simply implies that there is a subjective feeling to certain brain states, and this serves some kind of adaptive function, such as providing an "intentions-in-action" foundation for higher volitional behaviors, namely "intentions-to-act" (Fig. 1.4). Thus, if we understand that affects are functions of the physical brain then it makes sense to speak about primary-process affects as evolutionary phenomena. Their similarities across so many species indicate that affective capacities are ancient functions of the brain. Like many adaptive evolutionary developments, the brain systems that support biologically successful affective capacities have been retained as animals evolved. Other such evolutionary developments that have been retained include DNA replication, metabolic functions such as digestion and respiration, and the cellular production of energy. If you understand how the Krebs cycle works in one animal, you have a good understanding of how it works in all animals.

The logic of evolution suggests that affective capacities were retained as various species emerged through natural selection because these brain functions provide efficient ways to live and reproduce. These brain functions provide selective advantages in that they effectively anticipate universal, future survival needs. Animals that had these capacities survived and bred with greater success. Affects, from this perspective, are inbuilt anticipatory neuropsychological mechanisms of the brain. Just imagine how useful pain is for your survival.

Affects provide a flexible guide for living. Prior to the evolution of emotion, animals must have behaved in more stereotypical ways. For example, primitive sea creatures had no choice but to undulate with uniform motions as they made their way through the sea. Relatively inflexible behavior can also be quite complicated—honeybees perform a multiplicity of instinctive functions, some of which we think probably have affective dimensions. For instance, honeybees do show a frustration-like response when experimenters shift access from a high, very-sweet concentration of sugar to one that is much less concentrated, and presumably less desirable (Wiegmann et al., 2003). The full-blown affective capacities of mammals,

however, allow animals to respond to the here-and-now challenges of life in highly flexible ways.

For instance, if a rat is accustomed to feeding in a particular corner of a field, and if a ferret takes up residence nearby, the rat will smell the ferret even when the ferret is not present. The smell of this predator unconditionally arouses the FEAR system of the rat. This arousal triggers fearful affects, which feel bad. The rat avoids the smell of the ferret in order to avoid feeling frightened. In order to avoid feeling frightened, the rat will find another feeding ground. In this way, affects allow animals to anticipate events. But please note that this anticipation is not a *cognitive* function. It is a spontaneous affective response, leading to unconscious learning mechanisms to be engaged that allows an animal to avoid the fearful feeling.

Although the rat's behavior could suggest to us that the rat is somehow aware of where the ferret might be found, this is not necessarily the case. FEAR alone is a reliable way of anticipating future events, even if the rat's modest cognitive capacities are unable to conceptualize ideas about the future (clearly a tertiary aspect of the BrainMind). Defensive affects such as those produced by the FEAR system protect the survival of the individual, while the nurturant affect of CARE protects the survival of others (particularly others who carry part of the CAREing individual's genes). LUST likewise protects the survival of the species. The point is that innate affective capacities guide animal behavior in ways that enhance survival in the here and now, and across generations.

While affective systems lie deep in the subcortical brain, cognition, on the other hand, emerges from the neocortex, which is the brain's outermost layer and the part that is evolutionarily newest. This indicates that the capacity for affective experience evolved long before the complex cognitive abilities that allow animals to navigate complex environmental situations. It is also noteworthy that the deeper evolutionary location of the affective systems within the brain renders them less vulnerable to injury, which may also highlight the fact that they are more ancient survival functions than are the cognitive systems.

We have said that affects are primary-process experiences because they are unalloyed mental elements, unlike anything else. But we may also be justified in considering affects as the original forms of consciousness—affects may have been the first sources of felt experiences that ever evolved

within the brain. But they come in several varieties—emotional, homeostatic, and sensory (Figure 1.4). Raw affects may be the primordial source of anoetic consciousness—primary-process experience without understanding.

To summarize, the kinds of layering we envision in BrainMind evolution coaxes one to first focus on the most ancient levels and to use that knowledge to clarify secondary processes, where primal emotional functions are integrated with perceptions, allowing conditioned learning. For example, a rat that begins to fear the sound of a cat's bell is using a secondary emotional process, as are the rudimentary cognitive strategies, such as a rat learning to run to its sequestered home when it hears the cat's bell. This provides animals with factual knowledge of the world—a primitive *noetic* or knowing form of consciousness. But do rats also think about this consciousness? Are they "aware" that they are experiencing something. We simply do not know. And no one has suggested a way to solve that dilemma.

We refer to a tertiary level of processing for higher emotional functions when the first two levels of mind begin to generate more complex cognitive abilities, like the planning that goes into preparation for a weekend hike or planning one's future professional goals. Tertiary processing allows for intelligent reflection about the world and about oneself, considering both past and future frameworks—within *autonoetic* consciousness. That level of mental activity is remarkably hard to study in animals. The tertiary level is strongly linked to functions of the frontal cortex and the parietal cortex—the most recently evolved regions of the neocortex that exist in superabundance in humans and a few other well-cerebrated creatures.

SUMMARY

All of us would like to understand what is happening inside our minds and in the minds of those we know, including the minds of wild creatures and the minds of our various tame domestic and companion animals that bring such richness to our lives. Affective neuroscience provides a new and unique evidence-based perspective on the nature of emotional Mind-Brain functioning, opening a window on the ancestral sources of our deepest affective values.

In the next chapter, we will examine some of the scientific and historical reasons why affect has been marginalized as a topic for neuroscientific study. We will also give a brief synopsis of the research that supports the existence of affects in other animals. We will examine the same research more fully when we discuss the SEEKING system, which provides decisive evidence about this issue. Separate chapters will be devoted to each of the seven primary-process emotional systems. Because so much research in the area of learning has focused on the FEAR system, we will pause after that chapter to summarize some of the learning (secondary-process) mechanisms of the brain. In particular, we will show that conditioning, which some people regard as a cognitive function, is nothing of the kind. It is an automatic brain response that does not require any neocortical participation in order to succeed. And unlike the primary-processes of the mind, that level of BrainMind integration seems to be deeply unconscious, but provides us with a foundation for noetic consciousness. We also will highlight ways in which the emotional instincts—the unconditioned affective networks—may be critical in "opening the doorways" to learning (a topic largely ignored by those who work on the brain mechanisms of learning in animals, especially fear conditioning).

All along we will return to human clinical issues that focus on complex tertiary processes and emotionally tinged thoughts, as well as emotional regulation and dysregulation. It is in this area that human research is essential, with many directions for study and development currently being advanced by various modern psychotherapeutic schools of thought that are increasingly emphasizing emotional issues (see Chapter 12, in which Panksepp elaborates on some of his views about the future of psychotherapy from the perspective of affective neuroscience). Along the way, we will also reflect on the nature of the "self" and on the possibility of a new reverence for life that these brain systems encourage us to consider.

Overall, our perspective is that an understanding of affect is of critical importance for an understanding of human nature. Not only are our personality structures rooted in affect (Davis et al., 2003; Davis & Panksepp, 2011), but a remarkable number of societally important human issues need to be approached from affective as well as from cognitive perspectives. Insightful modern psychotherapists have known for a long time that the goal of psychotherapy is affect regulation. Even though psychotherapy may appear to focus on thoughts, insofar as patients largely

communicate in words, the aim of treatment is to positively change the patient's affective experience. This inevitably entails changes in the way that he or she thinks, but the aim of psychotherapy is not simply to alter cognitive style or content. In contrast, many psychiatric medications modify affects directly, without cognitive interventions, but often with robust cognitive changes following in the footsteps of better regulated affects. Indeed, it is increasingly evident that environmental, interpersonal and medicinal approaches to the treatment of mental problems work better together than any of these approaches by themselves. Toward the end of this book Panksepp will discuss some possible directions alternative therapies might take in addressing affects more directly.

Ultimately, affects are the very base of our psychological being. When the affects are satisfying, life is a joy. When they are disturbed, life can be hell. As noted by John Sterling (1806–1844), a poet who lived on the Scottish Isle of Bute, "Emotion turning back on itself, and not leading on to thought or action, is the element of madness." In Chapter 11, we will make the case for the conclusion that raw affective feelings lie at the primordial foundation of the mental apparatus—that they are the primal biological substrates of a core-SELF—perhaps the neural foundation for the concept of "the soul."

There is now inferential evidence that a universal core-SELF type structure, essential for organismic coherence, exists deep in ancient regions of the brain where primary-process emotional systems are found. The diverse, evolutionarily "given" emotional tools of our brains may all rely on this extensive substrate for primal body representations for the generation of the many types of raw emotional feelings that all mammals experience, with many nuanced evolutionary differences that we currently know little about.

In contrast, our many higher emotional viewpoints—from blame to shame, and feelings of jealousy to empathy and kindness—are intimately enmeshed with our cognitive apparatus. Our higher cognitive apparatus allows us an enormous number of emotional options, including concurrently distancing ourselves from ruling passions and immersing ourselves in acceptance or "mindfulness."

Cognitive science, still relying almost exclusively on a computational theory of mind, may be turned on its head once academicians realize how profoundly human thoughts are influenced by affective feelings (Davies,

2011). The final picture of how emotions govern our learned viewpoints and the reprocessing of our experiences may turn out to be very different than the provisional visions we currently have (see Chapter 6). With a better understanding of affects, it is conceivable that the therapeutic enterprise will move toward a more refined, neuroscience-based perspective on how one human being can help another move toward emotional balance, with the synergistic use of psychotherapies and mind-medicines.

An understanding of the primal passions may make it easier for people to aspire toward Aristotelian *phronesis* (see the epigraph for Chapter 4)—namely, knowing how to work cognitively with one's own emotions, with wisdom, as opposed to being a hapless victim, living in perpetual conflict, in the unyielding grasp of the ancestral powers of our minds. And it should be recognized that these powers are the same ones that guide the lives of many other animals. The way we will eventually understand our deeper mental nature is by understanding the deeper neural nature of animals. What are we waiting for? Let the conversation begin.

CHAPTER 2

The Evolution of Affective Consciousness

Studying Emotional Feelings in Other Animals

> *We cannot be absolutely certain that other humans have experiences, let alone that nonhuman animals have experiences (the problem of 'other minds'). But on the basis of evolutionary theory, it seems reasonable to assume that forms of consciousness evolve along with the biological forms that embody them. But what is it that the bee sees?. . . . And what do the moth or dolphin hear?*
>
> —Max Velmans (2009, p. 192)

MAX VELMANS'S REMARKS HIGHLIGHT OUR dilemma. How does raw experience—*phenomenal consciousness* as philosophers put it—emerge from brain activities? This is not just the "hard problem" of consciousness studies, but of neuroscience in general. Indeed, perhaps it will be much harder to decode how the brains of other animals experience sensory inputs than the affective qualities of basic emotional feelings. Why? (i) Because we can evoke distinct emotional action patterns by stimulating specific regions of animal brains, and (ii) because each of the primary-process emotions so evoked is accompanied by negative or positive affective states, which can be objectively monitored through various learning tasks, with no need for linguistic self-reports. Thus, we can determine how neural circuits generate emotional "rewards" and "punishments" within the brain more

easily than perceptions. What we can be sure of is that animals are not neutral about any of the various forms of artificially induced emotional arousal. By the various learning and preference measures available to us, we know that all mammals that have been studied dislike some of these kinds of brain arousal (RAGE, FEAR, and PANIC/GRIEF) while they like others (SEEKING, LUST, CARE, and PLAY).

However, it must be emphasized that each of these positive emotions shares the SEEKING urge to some degree (arousal of the negative emotions may share it as well, as in the seeking of safety in FEAR and maternal CARE during GRIEF). These affective-evaluative abilities are shared with all other mammals that have been studied—this much we know with scientific confidence. Many such brain circuits are present in other vertebrates. And the relevant brain chemistries may even mediate affect in some invertebrates: Some species (e.g., crayfish) exhibit marked preferences for addictive drugs that captivate humans, such as morphine and amphetamines (Huber et al., 2011).

Can we conclude anything more about the experienced qualities of the various primary-process positive and negative emotions of other mammals? Perhaps. The internal dynamics of each of these various feeling states may bear more than a passing resemblance to the corresponding instinctual outward display of emotion. Each of the felt emotions is behaviorally expressed in visible signs that are particularly unambiguous in "lower" animals—displays ranging from SEEKING to GRIEF. Human adults can readily inhibit their emotional displays, allowing their feelings to go "underground," so to speak (indeed, the neocortex functions best when such primitive emotions are regulated—kept under control). In our children, however, such bodily dynamics still convey the overall qualities of our most intense forms of emotional arousal. Just consider the pounding insistence of RAGE, the trembling of FEAR, the light rambunctiousness of PLAY, the gentle caress of loving CARE, and as we will focus on more than any other, the eager searching and poking around of SEEKING. These are the kinds of behaviors that can also be evoked by stimulating specific regions of the brain. These natural emotional expressions probably have more than a passing resemblance to the emotional feelings themselves. And this is a key point: Emotional feelings and their spontaneous behavioral expressions arise from the same ancient neural systems. As a result, we now know

where to look for the constitution, the neural mechanisms, of emotional feelings.

But how can we know that the various negative and positive feelings are actually distinct, as opposed to modest variants of one type of primordial good and one type of bad feeling? Among the positive affects, one could determine whether animals discriminate the different emotional states evoked by various neurochemicals (e.g., neuropeptides and psychopharmaceuticals) or among the various rewarding and punishing forms of direct brain stimulation. In fact, we do know that animals distinguish the positive feelings of certain distinct "reward" sites of the brain (Stutz et al., 1974), as well as the internal states engendered by addictive opioids such as morphine and psychostimulants such as cocaine (Overton, 1991), all of which are highly rewarding to all mammals (Tzschentke, 2007). But much more research along these lines needs to be done before we know the actual number of distinct primal affects, and the brain mechanisms, by which diverse emotional feelings are created.

Animals in basic emotional states also make characteristic sounds that are often not that different from the emotional sounds we make. Just consider the squeal of pain, the growl of anger, the repetitive chirpy sounds of laughter. These sounds arise from distinct brain networks in primates (Jürgens, 2002). And at the same time, each type of sound arises from essentially the same brain regions across all species of mammals that have been studied (for summaries, see Brudzynski, 2007; Brudzynski et al., 2010; Newman, 1988). Thus, the subcortical brain systems from which emotional affects emerge are remarkably similar throughout the mammalian kingdom. There is also abundant evidence that basic emotional feelings in humans arise from these same lower brain systems rather than from the higher regions of the neocortex (Damasio et al., 2000; Northoff et al., 2009; Vytal & Hamann, 2010).

The likelihood that primary-process emotional feelings in animals resemble our own is thus not only based on abundant data but also on the substantial cross-species evolutionary continuity in our primary-process emotional nature (Darwin, 1872/1998; Panksepp, 1998a). Similarities are also dramatically demonstrated in the basic emotional learning mechanisms of the brain (LeDoux, 1996). We cannot as easily generalize such concepts to the tertiary-process level of mental complexity. It seems unlikely that other animals experience reverence or feelings sublime, and lack of credible

evidence will prevent us from even considering such possibilities. Although chimpanzees certainly show reconciliation behaviors following squabbles (de Waal, 2009), perhaps they do not experience the grace of forgiveness the way we do. Higher order feelings are simply impossible to study with current procedures. Thus, there is no experimental evidence that other animals dwell on the meaning of happiness or have enough self-reflection to feel the sting of embarrassment, guilt and shame. Perhaps they harbor resentments when poorly treated by someone (think of stories of elephants rampaging when repeatedly treated poorly by human beings). But we cannot peer into their thoughts as effectively as we can into their emotions. Questions about subtle tertiary-process emotions of considerable importance for human affairs—from avarice to sympathy—may never be addressed in neuroscientific detail in other animals. Even though some possibilities may be inferred from careful behavioral observations (Bekoff, 2007; Grandin & Johnson, 2009), there are no scientifically sound models for studying such complex, tertiary-process emotions in other animals. However, the primal emotional feelings can finally be experimentally studied, and that knowledge may have profound implications for understanding our own deeper nature and our kinship to other animals.

Thus, in contrast to the primary-process emotions, which have dedicated (evolved) neural controls in the brain, the behavioral indicators of most higher-order emotions in animals (empathy, humor, jealousy, shame, and so on) are bound to remain vague and controversial, even though human opinions can be systematically collected (Morris et al., 2008). Their existence, at a scientific level, for now, is based on anecdotal evidence. Of course, the plural of anecdote may be data, at least according to those who are open to the likelihood that many other animals do have higher emotions (Bekoff, 2007). And there is abundant behavioral evidence indicating that many higher primates exhibit complex social emotions (de Waal, 2009). Even mice show behavioral and autonomic changes (e.g., fearful freezing and heart rate changes) that may be indicative of empathy (Chen, et al., 2009).

These subtle, higher-order emotional processes can of course be addressed by human brain imaging (Decety & Ickes, 2009; Iacoboni, 2009a, 2009b). When such tools of research become sufficiently refined to routinely visualize the changes transpiring in the subneocortical emotional networks[1] that we discuss in this book (e.g., perhaps through use of more

powerful magnetic fields, and more highly sophisticated statistical techniques), we may find that all of the affective powers of higher human emotions—marvelously wonderful and subtle feelings—remain grounded in the ancient neural terrain from which mammalian primary-process affects arise. The primary processes may remain the solid evolutionary platform for such emergent diversity. Indeed, there is growing evidence for that. Affective change in brain scanners is correlated positively much more with subcortical arousals than with neocortical ones; cortical arousal tends to reflect a decreased intensity of feelings. Thus cortical arousal is commonly at a low point when our minds are full of emotional feelings, and it is high when feeling intensity is low. This suggests that higher brain activity tends to inhibit the feelings arising from lower brain regions (Northoff et al., 2009), as the hyper-emotionality of decorticate animals has long indicated. However, it is also known that when humans ruminate on their emotions within brain scanners, their self-involved dwelling typically arouses medial frontal regions of the brain (Northoff et al., 2011). Meditative maneuvers such as "mindfulness"—learning how to be at peace within present moments—may often be more effective in reducing such ruminative emotional arousals than more traditional psychotherapeutic approaches (Siegel, 2007).

It remains possible that only humans and related primates (in addition to perhaps elephants, whales, and dolphins), through their rich and complex family lives and extended early cognitive development, can experience more complex social emotions than most other animals do. But these remain unstudied issues, perhaps out of reach of current scientific scrutiny. In humans, the confluence of basic emotions and complex cognitions is bound to have profound effects on one's emotional life, sometimes for the better, but rather too often for the worse. We certainly seem to be more susceptible to emotional disorders than other animals because of our ability to keep emotions percolating through the power of higher cognitive processes. When people dwell and ruminate on their troubles, this can sustain and stir up unique emotional upheavals. However, we will not say much about such higher human emotions here. Our task is to develop robust arguments for the inclusion of other animals in the circle of those who experience primary-process affects, and we will endeavor to make this case objectively and neuroscientifically.

We have said that a comprehension of primary-process affects, in both humans and other animals, is crucial for understanding how the Mind-Brain operates. We believe this is one of the key areas of inquiry if we are to crack the neural codes of consciousness and to bring new and better treatments for psychiatrically significant problems of living. Scientific triangulations among *neural*, *behavioral*, and *mental* analyses that cross species scientific studies now permit, finally are providing a more sophisticated understanding of shared animal and human emotions than ever before. But to do this well, we will also have to examine some historical reasons why psychological science and neuroscience have tended to marginalize the study of the mental life of animals and of the affective life of animals in particular, at least until quite recently (Panksepp, 1998a). We will then summarize neuroscientific evidence that demonstrates how raw affects, the ancestral feelings of our minds, emerge from the subneocortical systems we share with so many of the other creatures of this world. Before we proceed to discuss each of the primary-process emotional systems in subsequent chapters, we share here a history of emotion studies, especially the study of emotional feelings, to put various cross currents that still influence the field into perspective. For those who do not wish to reflect on these historical forces, please feel free to move to the next chapter that discusses the SEEKING system.

THE MARRIAGE OF THE BRAIN AND THE MENTAL APPARATUS: A HISTORY

Until quite recently, many philosophers and even some scientists tended to see mental life as immaterial and epiphenomenal—as a topic that the hard biological sciences could never address. Neuroscience, like the other hard sciences, must rely on objective observations of physiological and behavioral facts, and many colleagues still argue that animal experiences (primal consciousness) cannot be measured. It cannot be weighed. It has no length or breadth; it is made of only murky neurodynamic depths that cannot be rigorously monitored in any way, even in humans, where linguistic feedback can be idiosyncratic and deceptive. Just consider the confabulations of people with strokes that affect the right hemisphere, which leave their speaking hemispheres without deep affective guidance. Such people often deny their blatantly obvious left-sided paralysis with

fanciful stories generated by their self-serving and linguistically capable left hemispheres—confabulations that sometimes disappear in the midst of psychoanalytic sessions (Kaplan-Solms & Solms, 2000). For instance, such people may speak at length as if they have no impairments, only to suddenly acknowledge their infirmities and fears when they drop their social façade and speak freely about the meaning of such disabilities for their "ruined" lives.

Many neurobiologically oriented scientists maintain that we cannot say anything deeply substantive about mental life, certainly not in other animals, and that we cannot even assert that consciousness is real—that it is anything more than a figment of our imaginations. In 1992 an eminent evolutionary biologist, George Christopher Williams (1992, p. 4), wrote, "I am inclined merely to delete it [the mental realm] from biological explanation, because it is an entirely private phenomenon, and biology must deal with the publicly demonstrable." Many colleagues concur. We do not. If we do not deal with the real feelings of people in distress and try to scientifically understand their deep, often negativistic, feelings, we will never really understand what emotionally ails them. A large part of this understanding will have to come from the study of our fellow animals. We can envision a day when mental ailments like depression are treated by using our knowledge of positive affects to rebalance minds that have been overwhelmed by negative affects. Of course, this will also need synergistic human interactions, especially as we develop new and more effective psychotherapeutic practices (e.g., see final chapters of this book). In sum, our claim is that a biological understanding of the affects cannot be obtained without a proper, theoretically guided study of animal brains and minds. This may surprise many. But that must surely be the case if one thinks through all of the relevant scientific and ethical issues.

In any event, when we study the BrainMind, we are confronted not merely with brain circuits and molecules, but with how the complex textures of feeling, arising from these neurophysical substrates, help create mental lives. To make sense of the diverse psychiatric disorders, we must scientifically confront the nature of affective experience. We cannot go from the diagnostic label of "depression" to a thoroughly brain-based understanding of this neuro-mental phenomenon, unless we ask, "Why does depression hurt?" (Solms & Panksepp, 2010; Panksepp & Watt, 2011) and more precisely, "What kind of hurt is it?" (Watt & Panksepp, 2009).

There is a long history of the tendency to "delete the mind from the brain"—and it has two major strands. One strand is *dualism*, a belief in the existence of two ontological realms: the immaterial alongside the material. Dualism was integral in the thinking of the ancient Greeks. And during the past four centuries its most famous proponent was the philosopher Rene Descartes (1596–1650), who had many followers, until recently. The second relevant strand of history stems from a scientific movement that arose among a revolutionary group of German physicians committed to modernizing the medical curriculum in the latter part of the nineteenth century, long before scientists knew much about the nervous system. Let us examine the arguments one at a time.

How Did the Other Animals Lose Their Emotional Feelings?

Dualism had been accepted by many scholars for centuries before Descartes' writings. It was an integral part of thinking among the ancient Greeks, who typically saw immaterial reality as more important than the material world. Plato (424–348 B.C.) believed that "forms"—nonphysical conceptual realities—captured the true essence of material reality. For example, one can see beauty in individual objects, but to understand the essence of beauty, one must understand beauty as a "form," as a concept that exists above and beyond all the individual instances of beauty. Thus, for Plato, physical reality was merely a reflection of the ultimate nonphysical reality: the reality of the ideal forms (Copleston, 1962a; Plato, 1941).

Aristotle (384–322 B.C.), the great biologist of ancient times, proposed that all living creatures are imbued with a *soul*, which he viewed not as a personal soul but rather as an immaterial force of nature that accounted for changes in the physical world. For example, the soul of a seedling would account for its potential to grow into a tree (McKeon, 1941). Now we recognize that such causes arise from genetic inheritance. Saint Augustine (354–430), one of Christianity's most influential early thinkers, accordingly described the soul as a special substance, endowed with reason, that helped to rule the body. Descartes gave a particularly religious gloss to the Aristotelian ideas propagated by Augustine, probably at least in part for political reasons (he had no wish to be censored by the church, as Galileo

had been, made deeply meaningful by the threat of torture). He thought about immaterial forces in terms of personal consciousness, which he described as an expression of God's spirit in the mind of man. In this way, God's immaterial spirit determined man's behavior (Copleston, 1962b).

Descartes saw animals in a different light. He did not see them as conscious creatures because he believed that God would not manifest his divine spirit in such lowly life forms. He viewed animals as nothing more than living machines, creatures without the divine spark. This view led to inhumane experimentation on animals (e.g., live dissections with no anesthesia); their noises of protest and efforts to escape were seen as nothing more than reflexive reactions devoid of any conscious experience. Only man was a conscious being and man's consciousness was a part of God's divine realm. As such, man's consciousness determined his actions. To make this far-fetched idea work, the Aristotelian soul and the Cartesian divine mind of man had to be seen as being controlled by immaterial forces that also determined the behavior of the physical world. Aristotle's theory accounted for changes in all living things and Descartes' theory accounted for human behavior in particular.

The notion of an immaterial existential realm was also found in ancient Hippocratic medicine, which espoused the notion of *vitalism*. Vitalism followed the Aristotelian belief in an immaterial force that caused changes in the material world. According to Hippocrates, vital forces created sickness and health (Smith, 1979). Hippocrates (ca. 460 B.C.—ca. 370 B.C.) was known as the father of medicine because he ascribed illness to states of the body, rather than attributing them to mystical forces. However, although he rejected a wholly mystical basis for medicine, he was still a dualist; he firmly believed in the existence of immaterial vitalistic forces. He maintained that four basic bodily *humors*, or fluids (yellow bile, black bile, phlegm, and blood), were the physical expressions of the vital forces that determined excellent or wretched health. In his view, a stable balance of these humors resulted in good health while all ill health resulted from states of imbalance. In the Middle Ages this way of thinking was extended to emotional temperaments, with the concept of choleric (angry), melancholic (sad), phlegmatic (cold and fearful), and sanguine (happy) personalities.

Medical interventions during the premodern European era were largely designed to rebalance the humors, which in turn meant that the immaterial

vital forces that governed the body and mind were brought into balance (Smith, 1979). For example, wine was believed to counteract an excess of yellow bile by promoting levels of blood and sanguinity. Citrus fruit was thought to reduce phlegm and so on. At its worst, Hippocratic principles induced doctors to bleed patients or to administer poisons like hellebore, prompting vomiting and diarrhea. Except for possible beneficial placebo effects, it is likely that such interventions often harmed patients or did nothing at all.

From Nineteenth-Century Medical Science to Behaviorism

Medical science was a blunt instrument in the days of Hippocrates, especially because autopsies were prohibited by the state. Hippocrates knew little about the internal workings of the body. Nevertheless Hippocratic principles dominated medicine for more than two millennia. After the Renaissance, however, scientific advances began to undermine confidence in the Hippocratic theory. Modern inventions like the microscope allowed scientists to learn that some diseases were caused by microorganisms rather than by imbalances of fluids. But change is always slow and unwelcome. It was only in the middle of the nineteenth century that a group of Continental physicians devoted to empirically based medicine (led by such luminaries as Carl Ludwig [1816–1895], Emil du Bois-Reymond [1818–1896], Hermann von Helmholtz [1821–1894], and Ernst von Brücke [1819–1892] —all interested in the brain to some extent) formed a group of like-minded physician-scientists, later called the Berlin Biophysics Club (Greenspan & Baars, 2005). They rejected Hippocratic ideas about the four humors that did not tally with modern physical discoveries about illness.

The Berlin Biophysics Club also rejected vitalism in general. They rejected the existence of all the spooky forces that had been postulated to govern the functioning of bodies. These eminent scientists maintained that nonphysical forces cannot be subjected to scientific scrutiny, so one cannot know if claims about them are true or even whether they really exist. For these reasons, members of the Berlin Biophysics Club decisively abandoned dualism in science. For them, science had to be rooted in a study of the physical world alone.

These revolutionary physicians were content to carry out experiments on the physical body and to construct mechanistic theories based on their observations. But they did not see their theories as an overriding immaterial truth. Scientific theory was simply seen as the best explanation of the available evidence. Facts were more important than theories. In principle, theories could always be overturned in the face of contradictory evidence.

This revolutionary movement rapidly succeeded in establishing a new, rigorous medical curriculum on a solid scientific base. The club's victory led to an evidence-based approach to medicine that remains the foundation of medical education to this day. In psychology, however, antivitalism took a special form. How does one study the mind from a biophysical perspective? The stop-gap preneuroscientific solution proposed by the powerful behaviorist movement that dominated academia until the last quarter of the twentieth century—and it is not yet dead in neuroscience, particularly in behavioral neuroscience—was that consciousness didn't matter. Behaviorists chose to study only the externally observable dimensions of brain functions (that is, behaviors, and the incoming "stimuli" to which the behaviors were outgoing "responses"). Behaviorists' most important tools were entities they called unconditioned stimuli (UCS) and unconditioned responses (UCRs)—things like electric shocks and the resulting freezing behaviors (see Chapters 5 and 6)—which coaxed animals to rapidly exhibit learned coping strategies. In this way the behaviorists were able to bypass the "black box" (Skinner, 1938) of the brain, and thereby the mind. They speciously equated the making of inferences about *mental* forces of any kind (from observable behaviors and other scientific data) with the discredited notion of *vitalistic* forces. Accordingly, they saw no way to study the actual nature of the mind itself in any scientific way. And the mind ceased to exist, at least as far as most of the researchers within twentieth-century scientific psychology were concerned, most especially when it was discussed in the context of the study of animals.

The positivistic movement in philosophy saw strict definitions of all concepts (positivism), as the only way to build a solid science. Ludwig Wittgenstein (1922/1981), the great philosopher of language, in his *Tractatus*, provided the "definitive" statement of support for ruthlessly materialistic challenges to the study of the mind, in his famous assertion that "When the answer cannot be put into words, neither can the question" (Proposition 6.5) and since mental qualities are impossible to put into clear,

operationalized scientific language, one is left with the following dilemma: "Even when all possible scientific questions have been answered, the problems of life remain completely untouched. Of course there are then no questions left, and this itself is the answer" (Wittgenstein, 1981, Proposition 6.52).

For more of Wittgenstein's skeptical guidance, see the end of Chapter 13. It is poignant that, soon after the human tragedy of the Second World War, Wittgenstein, an emotionally tortured person for most of his life, proposed a more forgiving vision for the study of mental life in his *Philosophical Investigations*—one where our relativistic word-games prevailed, leading to a powerful social-constructivist movement in psychology that thrives to this day.

Of course, vitalism and mentality are crucially different. Vitalism proposes the existence of a fundamental nonphysical reality. Vitalistic forces were not envisioned as having any biological antecedents or physical basis. On the contrary, they were believed to be the unseen forces that determined the health of the physical body. Mentality, on the other hand, has clear biological antecedents and is unequivocally a property of the physical brain. It is not a disembodied force of nature. It is a brain function and can therefore be studied in normal scientific ways, just like any other biological fact. All we need to do is get on with the difficult job, which is what researchers within affective neuroscience (Panksepp, 1998a) seek to do. Because of advances in neuroscience, this is finally a doable project.

Unfortunately, these distinctions were lost on some peripheral members of the Berlin Biophysics Club. The physiologist Jacques Loeb (1859–1924) worked in the United States, first at Bryn Mawr College, then the University of Chicago and eventually at Rockefeller University (at that time "Institute"). While at the University of Chicago he influenced John B. Watson (1878–1958), the eventual "father of behaviorism." At Harvard, B. F. Skinner (1904–1990) was also persuaded by Loeb's ideas. Together, Watson and Skinner, inspired by Loeb, laid out a new, methodologically rigorous—and eventually doctrinaire—radical behaviorism.

They were heroes to many psychologists, even though they discarded "mind" from the curriculum. To some extent they succeeded because of the Cartesian foundations of modern science, which is deep skepticism. Let us recall that Descartes started his philosophy by doubting everything. He readily imagined that the world around him was little more than a dream or

hallucination. He saw no problem in doubting the reality of logic and mathematics, for he believed that evil demons could be controlling his reasoning. The only thing he could not doubt was that he doubted, leading to his salvation from infinite doubt: the one piece of incontrovertible evidence—*cogito ergo sum*. And so skepticism became the coin of the scientific realm. "Prove it to me" became the slogan, even as it became clear in twentieth-century science that there were no scientific proofs, only mathematical and logical ones. Science, because of its nature, had to be based on the *weight of evidence*. And from that perspective, the major claims of this book should come as welcome news for those who abide by scientific rules: Abundant facts indicate that other mammals do have emotional experiences, and we all share very similar neural foundations for our own primary-process emotionality. But on this one momentous item, for many neuroscientists, their love affair with skepticism still outweighs the reasoned weight of evidence . . . to the point where there is hardly any discussion of this topic, at least among behavioral neuroscientists, who have the best tools to take such questions farthest toward empirical solutions.

And thus, the fathers of behaviorism, those extreme skeptics about the need for *any* mental construct in psychological science, brought a new level of sophistication to the analysis of behavior, which provided a rigor that had been missing in the field of psychology. They gave us the first promising way to analyze the causes of acquired behavioral change—namely learning. They offered scientists tools that could reliably produce behavioral changes in the laboratory. But to achieve that, they felt they had to reject all references to internal emotional and motivational processes. Watson (1929) was initially interested in emotions but thought that intellectual capacities, independently of any temperamental issues, were learned without much influence from inborn functions. His famous claim was "Give me a dozen healthy infants, well-formed, and my own specified world to bring them up in and I'll guarantee to take any one at random and train him to become any type of specialist I might select—doctor, lawyer, artist, merchant-chief and, yes, even beggar-man and thief, regardless of his talents, penchants, tendencies, abilities, vocations, and race of his ancestors." Skinner went even further. He disdained emotional concepts in the new science of behavior from the outset and famously claimed: "The 'emotions' are excellent examples of the fictional causes to which we commonly attribute behavior" (Skinner, 1953). Curiously, neither of these scientists thought it

was essential for psychology to engage in the study of the brain in order to be a complete science, but that was long before neuroscience matured as the most important scientific discipline for understanding what organisms do.

Thus, conscious experience—affective experience, in particular—had no meaning for these radical behaviorists. They ignored Darwin's suggestion that animal behaviors provided an indication of their affective states and also William James's belief that emotional feelings are not aroused prior to emotional actions, but they follow (or are identical) with the expressions. In a sense that is the message of this book, but it simply recognizes that it is the emotional-action systems of the brain that carry the affective message, not the emotional actions of the peripheral body. This is not a small distinction, for even Damasio (1994) was enticed by a similar cortical vision of emotional feelings.

All of this kind of thinking, was for behaviorists, "just talk". The behaviorists also ignored the wording of the original, celebrated "Law of Effect" put forward by Edward Thorndike (1874–1949), one of the first psychologists to study animal learning systematically. Thorndike's original version maintained that animals experience feelings of "satisfaction" and "discomfort," which not only impel them to display preferences and aversions, but which also guide their learning. The original "Law of Effect" was really a "law of affect." The behaviorists rejected that aspect. Here is exactly what Thorndike put forth:

> Of several responses made to the same situation, those which are accompanied or closely followed by *satisfaction* [emphasis added] to the animal will, other things being equal, be more firmly connected to the situation, so that, when it recurs, they will be more likely to recur; those which are accompanied or closely followed by *discomfort* [emphasis added] to the animal will, other things being equal, have their connections to that situation weakened, so that, when it recurs, they will be less likely to occur. The greater the *satisfaction* or *discomfort* [emphasis added], the greater the strengthening or weakening of this bond. (Thorndike, 1911, p. 244)

Rather than using subjective words like *satisfaction* and *discomfort*—words that suggested a motivated mental state accompanied by a feeling tone—the behaviorists substituted more objective terms, referring to externally observable events: *rewards* and *punishments* (or *reinforcements* when used in the context of learning). They thought that all behavior was learned on the basis of psychologically undefinable aspects of rewards and punishments. They explicitly chose to ignore the likelihood that affective changes in the brain gave rewarding and punishing events the power to

control behavior. Rather than leaving open the possibility that rewards and punishments worked by generating experiences within the brain, "reinforcements" were defined in purely operational terms—in terms of the ability of objects in the world to "reinforce" behavioral changes in one direction or another. To this day, we do not know whether "reinforcement" is a specific kind of non-affective brain function, or simply a word used to describe how we train animals by systematically manipulating brain systems that control their feelings.

One thing is certain, animals do reliably work to obtain rewards and avoid punishments. Humans do the same. That humans and animals alike do these things for affective "reasons" is what the behaviorists could not accept as being scientifically workable, and hence credible, and their bias has been passed down to behavioral scientists to this day. Few have chosen to question those suppositions. Since references to affective and motivational states (such as hunger and thirst) were not accepted, and hence not allowed, such concepts disappeared from the lexicon of most psychological discourse. Third-person objective language was the coin of the new behaviorist realm; first-person subjective language was literally banned from scientific discourse. This was the case for discussions of both animals and humans. But now, thankfully, in our enlightened age, the ban has been lifted. Or has it? In fact, after the cognitive revolution of the early 1970s, the behaviorist bias has largely been retained but more implicitly by most, and it is still the prevailing view among many who study *animal* behavior. It seems the educated public is not aware of that fact. We hope the present book will change that and expose this residue of behaviorist fundamentalism for what it is: an anachronism that only makes sense to people who have been schooled within a particular tradition, not something that makes any intrinsic sense in itself! It is currently still blocking a rich discourse concerning the psychological, especially the affective, functions of animal brains and human minds.

Interestingly, there is no indication that the members of the Berlin Biophysics Club would have objected to the study of feelings or consciousness simply because they were not easily studied bodily processes. If a patient complained of pain, modern doctors in the nineteenth century surely took their claims seriously and tried to discover the physiological causes of the pain. Yet the experience of pain is not just an unconscious physical entity. It is a physical mental state, a phenomenal

experience. It is subjective but it is real—a physiological process of the brain. Pain has causes and it has effects. It helps us survive. Therefore, even though it is subjective, it is nevertheless worthy of scientific consideration in diagnosing physical injuries and illness in both humans and animals. And perhaps most important is the fact that pain is not only caused by bodily dysfunctions; it is also caused (actually generated or constituted) by neural activities in the brain. Even though the pain is localized to a specific body part, the experience is not contained where it is initiated and psychologically seems to exist, despite the fact that some philosophers think otherwise. In fact, to the best of our knowledge, the brain projects the feeling of pain onto the neural space where the body is represented. Sometimes the pain (for instance, neuropathic pain) is largely due to internal irritability of nervous tissue. In any event, pain is a property of the brain and it is not something experienced in the body outside the brain.

The ancients were not sure whether the brain was the substrate of mental events. Plato and Hippocrates thought it was, but Aristotle believed emotions emanated from the heart. However, long before the Berlin Biophysics club rejuvenated medical science, some researchers had already embraced the study of the physical brain as a means to better understand the functioning of the mind. Among the great historical pioneers with modern views there was Thomas Willis (1621–1675), an English physician who dissected the brain in elaborate detail (as described in his *Cerebri anatomi* of 1664), followed by a treatise on the pathology of the brain, and another on medical psychology: *Two Discourses Concerning the Soul of Brutes* (1672). Willis sought to describe how mental changes were related to brain functions, while not abandoning the idea that the classic humors of the body controlled emotional temperament. By the turn of the nineteenth century, the even finer brain dissections of the phrenologists Franz Joseph Gall (1758–1828) and his protégé, Johann Gaspar Spurzheim (1776–1832), led to general acceptance of the idea that the mind emerged from brain activities—even though Gall's and Spurzheim's practical method of linking personality to the formations of ("bumps" on) the skull was a failure. Cranial shape was erroneously thought to reflect accurately the size of the underlying brain regions, or "mental organs," but it took some time for that conjecture to be recognized as a scandalous oversimplification.[2] In any event, by the middle of the nineteenth century, many scholars of the nervous system were ready to dispense with dualism and envision the brain

as the organ of mind, just as many physicians were ready to discard medical superstitions and to modernize medical science.

Although most members of the Berlin-centered empirical medicine coterie were not concerned with emotional matters, it is noteworthy that Ivan Pavlov (of Russia, who developed a systematic way to condition reflexes) studied under Carl Ludwig, while Sigmund Freud (of Vienna, the father of psychotherapy) studied under Ernst von Brücke. Pavlov never marginalized affect in his studies of autonomic reflexes in dogs. He recognized the power of emotions, especially after his laboratory was flooded by the Neva River, almost drowning his dogs. Many of his pups subsequently exhibited what we would now call Post-Traumatic Stress Disorder (PTSD). Freud, of course, made affect a centerpiece of his premature aspirations (brain science was not sufficiently ripe) to create a scientific depth psychology called psychoanalysis. Freud eventually abandoned brain science and developed an emotion-based psychoanalytic *metapsychology*, but he conceded that it lacked the "hard stamp of science" (Freud, 1895/1968).

Members of the Berlin Biophysics Club probably would have accepted a theory of the emotional mind that was rooted in brain science. Indeed there were scholars during the nineteenth century, such as Charles Darwin and William James (1842–1910), who had quite modern views about emotions and consciousness (Darwin, 1872/1998; James, 1892). Neither of these great thinkers had the benefit of modern brain science. Indeed, most psychological research on emotions to this day seems little concerned with the underlying primary-process neural details, and the tertiary-process details are currently next to impossible to obtain, although we can estimate regions of interest, and their interactions, with modern brain imaging. In contrast to followers of classic "psychology-only" theories such as psychoanalysis, there are currently several new movements including *neuropsychoanalysis* (see www.npsa.org), which offer a judicious blend of mental and neural analyses. However, few have followed in the footsteps of pioneers like Walter Hess (1881–1973), a 1949 Nobel laureate. Hess was the first to demonstrate that one can provoke full-blown primary-process rage behaviors in cats, along with the appropriate autonomic responses, by electrically stimulating specific regions of the hypothalamus (for a full summary, see Hess, 1957).

Perhaps Hess had few followers in psychology because he avoided talking about the emotional feelings of the animals he provoked. Like others in his time, he chose to call such electrically induced displays of anger *sham rage*. In his retirement he admitted regrets about having been too timid, not true to his convictions, to claim that his animals had indeed felt real anger. He confessed that he did this because he feared that such talk would lead to attacks by the powerful American behaviorists, who might thereby also marginalize his more concrete scientific discoveries. To a modest extent, he tried to rectify his "mistake" in his last book, *The Biology of Mind* (1964), but this work had little influence. Nonetheless, he at least provided data that could have provided a neurophysiological basis for psychology, something that both William James and Charles Darwin would have greatly admired.

Behaviorism dominated academic psychology for some 50 years and only gradually began to lose influence in the last third of the twentieth century when the *cognitive revolution* resurrected the scientific legitimacy of the mind. Cognitive scientists, inspired by the development of computers, maintained that the mind was like a living computer that allowed people and animals to calculate contingencies and make decisions that guided behavior. The Computational Theory of Mind was born, which again could be understood, presumably, without brain research. Of particular interest was the notion of unconscious or inborn cognitive capacities, such as Noam Chomsky's hypothesis (1968) that human children have an innate knowledge of the basic grammatical structure common to all languages. For the most part, however, cognitive science was concerned with the mechanics of information processing—perception and learning—and not with the endogenous and generative properties of the living mind. The cognitive revolution focused mainly on those aspects of mental activity that most closely resembled computer software—the "information processing" parts of the mind—and therefore did not address questions of affect or motivation and emotion until quite recently (Gardner, 1985; Panksepp, 1988). Also, as already noted, the cognitive revolution was largely concerned with cognition in human beings, so in the field of animal research, behaviorism still held sway. Only a few scientists, such as Donald Griffin (2001) of Harvard, pushed the animal behavior field to become more liberal in its thinking, but he focused largely on the cognitive realm, which neuroscientifically is a more difficult problem than emotions.

To be fair to the behaviorists, their goal was to create a highly replicable science whereby investigators could specify the variables for "behavioral control" (the buzzword for specifying the precise environmental conditions necessary to channel learned behaviors in predictable directions). Most of them never really claimed that they were seeking to *understand* the fundamental mechanisms that control animal behavior. In their limited domain, they simply wanted to specify and predict how animals would behave in well-controlled environments rather than in the real world where they find themselves in nature (that was the province of *ethologists*). Hence they built artificial compartments (Skinner boxes) where every aspect of the animal's external environment could be controlled and systematically manipulated. The behaviorists simply were not interested in the unobserved events that went on inside these organisms, and they did not believe they could ever contribute to a scientific understanding of behavior. The tragedy, however, is that once neuroscience matured, many of those events, even affective ones, *could* be scientifically studied. But behavioral neuroscientists remained largely uninterested in, indeed resistant to, studying them. Psychologically profound aspects of brain function such as the primary-process nature of emotions, which by this time had become solvable scientific problems, were neglected and purposefully ignored. Thus, the failure of neuroscientists to tackle the topic of emotional feelings was directly due to the chilling effect of behaviorism. That remains substantially unchanged to this day in animal research.

The Modern Neuroscience of Emotions

The modern neuroscientific revolution began some 40-odd years ago with the development of fantastic new procedures for studying the workings of the brain, culminating in the neuroimaging devices of today that allow researchers to observe *in vivo* (in the living organism) what happens inside the human brain while someone is performing various activities. Many who are enthralled by this marvelous new technology have been educated in *behavioral* or *cognitive* traditions. The former don't accept emotional feelings as part of their program of research. The latter are prone to see affective feelings as just a subset of cognitive processes, which is a large mistake, at least at the primary-process level of brain organization, which is our main concern here. Cognitions are created by perceptions, learning, and

higher brain functions. Primal affects are ancestral tools for living that have dedicated circuits for various "lower" brain functions. Although cognitive mind functions in human beings are now commonly accepted as matter of fact, most researchers engaged in animal research still cling to behavioral doctrines and will ignore, deny, or remain agnostic about the existence of any affective life in animals.

As noted, certain animal behaviorists, under the banner of cognitive ethology, did begin to ponder the potential mental capacities of animals (Griffin, 2001). But generally most shunned discussion of emotional issues, and few pursued affective brain research. This then was the strand of thinking that led to the tendency within modern neuroscience to reject the existence of, and hence the systematic scientific study of, affects in other animals. This first strand of thinking is anchored in the erroneous ancient belief that mentality is vitalistic—that it is an independent, immaterial force that cannot be scientifically scrutinized. As already noted, this equation of consciousness with vitalism is incorrect. Primary-process mentality—the experience of intrinsic evolutionary values—is a function of the brain and can be scientifically analyzed in the same way as any other biological function (indeed, in the same way as any other inferred function or process in nature such as gravity or the activity of quantum particles in physics).

Another strand of thinking, which persuaded neuroscientists either to reject or ignore the question of affect in other animals, has its roots in the latter part of the nineteenth century, when William James and Carl Lange (1834–1900) independently and almost simultaneously developed a peripheral *feedback* theory of affect. They saw emotional behavior (like fleeing a scary situation) as an automatic, reflexive bodily response that is in itself devoid of affect. They proposed that information about these bodily responses is subsequently *fed back* to the thinking and observing part of the brain, namely the neocortex, which cognitively experiences the emotion. Thus, a higher brain function was thought to generate the affective experience (Damasio, 1994; James, 1884/1968; Lange, 1885; LeDoux, 1996). So you would not run away from a knife-wielding thief because you were afraid; rather, you became afraid because you were running away, which created all kinds of changes in how your body felt, as "read out" by higher brain functions. In fairness, we will point out that William James, the great defender of mind in psychological science, also noted that all instincts

have a feeling to them and that the feeling and the emotional response occur simultaneously (the position we defend here).

Although there is now scientific evidence showing that the enactment of emotional behaviors can generate weak shifts in affective feelings (Clynes, 1977; Schnall & Laird, 2003; Stepper & Strack, 1993) and that such effects can be obtained also by emotional action imagery within the human mind (Panksepp & Gordon, 2003), there is little or no evidence to suggest that intense affective feelings during emotional actions requires feedback to the brain from the peripheral body. Most of the evidence suggests, to the contrary, that raw emotional feelings are generated directly by brain tissues, indeed by those circuits that generate instinctual emotional actions. This does not mean that inputs from the body have no effects. They can certainly intensify or weaken feelings engendered within the brain. But they are not decisive in generating the specific way we feel emotionally. In any event, the classic interpretation of the James-Lange theory, proposed 120 years ago, is still the favored view of how emotions are created by those who know little about subcortical regions of the brain.

To this day there is no solid line of experimental evidence that supports the traditional version of the the James-Lange theory. However, the data support William James's alternative conjecture for primary-process emotions—that instinctual actions have feeling components—while his traditional cortical read-out theory can help us understand how the brain understands its emotions. Thus, to the best of our current knowledge, the brain generates affects in two ways: The lower parts of the brain can generate specific affective feelings that accurately signal both what the body needs (homeostatic and sensory affects) and what the brain needs (emotional affects). Then our higher brains deal with these powers of the mind in a large variety of idiosyncratic cognitive ways, which often contributes spice to the "human comedy." In addition all feelings have an arousal-intensity dimension which is often shared by many different feelings.

However, it should also be recognized that the brain and body have many arousal systems, including a major stress axis (the pituitary-adrenal system) and if one activates those without any true emotion being aroused, then people will tend to interpret the arousal in terms of the emotional scenario that the environment has promoted (Schacter & Singer, 1962). General arousal by itself does not an emotion make. A person also has to feel good

and bad in a variety of ways that correspond to various instinctual acting-out urges. When someone is angry, he may want to strike someone. The urge to strike someone, at the subcortical primary-process level, is concurrently accompanied by an enraged emotional feeling. That is what the data indicate, so far. But we also need to point out that every scientific fact *always* has multiple interpretations. The aim of science is to sift among these interpretations. That is why decortication experiments, which indicate that emotional feelings survive massive damage to upper (neocortical) brain regions, are so important.

If you are satisfied with the above synopsis of our views on the James-Lange "bodily feedback" account of emotion, feel free to skip to the next section devoted to the influential views of Antonio Damasio. But if a more detailed discussion would be of interest, please read on. . . .

Although we do not ascribe to the James-Lange feedback theory (or to its modern "read-out" progeny) we are admirers of James. As already noted, the concept of a peripheral "read out" of bodily commotion to higher brain regions was not his only theoretical observation concerning emotion. He also suggested, more correctly in our estimation, that every instinctual emotional response is accompanied by characteristic feelings. Had he only known that such instinctual responses were generated by distinct brain circuits, he might have surmised that there was no need to posit a cognitive "read out" to have emotional feelings, although the tendency to dwell on our feelings, even modify them through our capacity for conscious awareness, is certainly part of our higher cognitive apparatus. That is why emotional regulation is such a favored topic in psychology these days (Gross, 2009) and is also of great importance for psychotherapy. In any event, as we will argue throughout this book, raw emotional feelings are part of the subneocortical circuitry that also generates emotional action readiness. Because of the heavy weight of intellectual history (consider the case of radical behaviorism), James's alternative approach to understanding emotional feelings was not fully developed until recently (Panksepp, 1982, 1998a, 2005a).

We now know that feedback from the body in general cannot be the main source of the generation of feelings. Quadriplegics with no somatic sensory input from below the level of their high spinal damage have essentially normal emotional feelings (Borod, 2000). Of course, their spinal damage spares functioning autonomic nerves such as the vagus, as well as

circulating endocrine factors in the blood that can influence various brain regions. Thus it is especially important to note that even individuals with high spinal cord transections or brain-stem damage of the type that produces the "locked-in" syndrome—people who can only move (and hence communicate with) their eyes or their brain waves—still have emotional feelings (Bauby, 1997; Birbaumer, 2006; Laureys et al., 2005) even though bodily sensory input is quite dramatically reduced.

Walter Cannon (1871–1945), a Harvard physiologist who studied the peripheral autonomic nervous system, provided many other cogent arguments against a James-Lange view of emotions, and he advocated that emotionality was an intrinsic function of the brain. Cannon noted that many autonomic responses take time to develop and cannot be fed back to the brain quickly enough to generate an instantaneous affective response (Cannon, 1927). He concluded that affects are not a matter of feedback but that they emerge from the brain itself. It was Paul MacLean (1913–2007), a physician, who first developed this idea in greater evolutionary detail by generating the concept of an old mammalian layer in the human brain—the "limbic system," which was responsible for primary social emotions. MacLean initiated intensive brain analysis of emotional changes in epileptic patients in the 1950s and 1960s, and he subsequently developed animal models for sexual behaviors and various other social displays (1970s and 1980s). With considerable imagination, MacLean (1990) envisioned how emotionality, including affective experience, was linked to various primitive structures in the limbic system. As it turns out, MacLean did not have all the details correct (who does?), and for that he was unjustly chastised by various "young Turks" (for a rebuttal, see Panksepp, 2002). For example, MacLean thought that the hippocampus was among the most important emotional brain structures, but it is not. As we shall see in Chapter 6, the hippocampus is very crucial for memory formation: the encoding of autobiographical memories and the mapping out of our spatial environments. Still, it also facilitates learning about places where fearful events have occurred, and the ventral part of the hippocampus is quite important in emotional learning, especially issues related to space, as in place conditioning. However, one can also evoke certain strong emotions, for instance, one can readily cause rats to have erections by local infusions of oxytocin to the hippocampus (Melis et al., 1986).

Lack of evidence, however, was not the main reason that some investigators rejected the idea that the subcortical limbic brain generates raw affective experience. Some researchers profoundly disliked the anatomical imprecision of the mammalian "emotional brain" concept (i.e., the limbic system), and some also rejected the idea that emotional experiences can emerge directly from activities of subcortical systems. Indeed, as noted, the majority of emotion scholars still prefer the James-Lange idea that affects emerge from higher cortical brain regions, in which emotional behavior is interpreted (read out) by the neocortex.

At the same time that modern "read-out" theories were being developed, the senior author of this book, was developing the evolutionarily based concept of cross-species "affective neuroscience," detailed in an earlier book (Panksepp, 1998a). The approaches of MacLean and Panksepp converged substantially, although Panksepp began developing affective neuroscience at the beginning of his career, while MacLean was moving more and more in the direction of animal neuroscience models toward the end of his. Concurrently and independently, both became interested in understanding the social-emotional networks of the brain—especially of separation distress, social bonding, and playfulness. Both were followers of Cannon and Darwin, because they recognized that emotional feelings were direct reflections of specifiable activities in distinct brain networks, rather than peripheral feedback or higher brain readouts. According to this alternative view, which has gradually become the minority position, the ancient affective brain is designed to intrinsically anticipate life-challenging events with affective-instinctual unconditioned responses, which help guide learned behaviors and thinking accordingly.

Although modern read-out theories differ from the James-Lange model in many details, the principle remains the same: The emotional states of the brain are higher brain responses to or reflections of lower brain or bodily processes. It was strongly argued, by eminent neuroscientists, that the ancient subcortical brain regions that we share homologously with other mammals do not possess intrinsic affective properties (Damasio, 1999; LeDoux, 1996; Rolls, 2005). Parenthetically, as this book was ready to go to press, Damasio (2010) made a 180-degree turn and explicitly recognized the importance of subcortical functions in the construction of minds, although he still envisioned emotional feelings in high cortical regions. To the extent that modern neuroscientifically oriented read-out theorists

express any interest in affect (the feeling dimension of emotions), which is rare, they tend to conclude that affective experiences emerge only when unconscious emotional information is read out by the cognitive-thinking parts of the brain (especially by the neocortex). This has led to the most popular current view of emotional feelings and all other forms of phenomenal consciousness, namely that they are simply a variant of higher cognitive processes. In our terminology, the prevailing view among cognitive scientists became that emotional feelings are a tertiary process of the brain. Some still go so far as to suggest that there are no basic emotions —that all emotions ultimately reflect higher conceptual acts (Barrett, 2006). Although this may be true for tertiary-process emotions, such views neglect a great quantity of the available behavioral evidence from humans (Izard, 2007) and the cross-species neural evidence for primary emotions in all mammals (Panksepp, 2007d, 2008a). (A full issue of the new journal *Emotion Reviews*, as well as a recent monograph [Zachar & Ellis, 2012], are devoted to full discussions of this topic.)

We will pass over much of the theorizing about emotions that transpired in psychology during these last several decades, because little of it has been based on understanding the brain. It is noteworthy however, that Darwin's seminal work on the bodily expressions of emotion, *The Expression of the Emotions in Man and Animals*, was finally reintroduced to modern science in the 1970s and 1980s by the investigators Paul Ekman and Cal Izard. They worked in the tradition of basic emotion theory, pioneered by their mentor, the clinical psychologist Silvan Tomkins, who coaxed them to study intrinsic human emotional behavior patterns, replicable across development and across cultures, especially as expressed in the face. Others such as Ross Buck and Robert Plutchik cultivated basic emotion theory in different directions, especially the formulation of new introspective and clinical measures. It is true to say that only a few psychologists during this period were willing to discuss the nature of basic emotional feelings. Included prominently among the 'rebels' was the aforementioned Silvan Tomkins (1962, 1963), and more recently the social psychologist Ross Buck (1999). And even though psychotherapists have long recognized the importance of emotional feelings, currently an increasing number of practicing clinicians are focusing on emotions in new ways in order to help establish affective well-being (e.g., see Fosha et al., 2009a; Greenberg, 2002). We will not cover the ideas of these influential psychotherapists in

any detail, since their work has not focused on an understanding of the underlying brain mechanisms, but their impact on the evolution of new emotion-dynamic therapies will be contextualized by Panksepp in the twelfth chapter (i.e., the junior author did not wish to be affiliated with those views).

We now briefly describe three modern read-out theories, proposed by prominent neuroscientists: Antonio Damasio (1994, 1999), Joseph LeDoux (1996), and Edmund Rolls (1999, 2005). Although we disagree with their ideas about the foundations of affect, we admire their impressive experimental contributions. Obviously, in the following sketches we cannot do justice to the details of their wonderful empirical work—but each has written extensively about those achievements in the above cited book-length monographs. We also wish to emphasize that what we wrote about Damasio's views below, became somewhat dated during the writing of this book, because of his acceptance of robust subcortical contributions to emotional feelings and consciousness, quite resonant with the perspective advocated by Panksepp for three decades. However, a close reading indicates that Damasio still envisions emotional feelings to be largely constructed by higher sensory processes. Thus, we leave our discourse unmodified in light of this timely development (Damasio, 2010), especially since our aim here is simply to convey the prevailing historical perspective which Damasio was among the most influential in reinforcing.

We think that these scientists' ideas about *primary-process* emotional feelings have not been well developed. Indeed, few have emphasized the evolutionary layering of both the brain and mind. And hence, to us, their claims about affective experiences have often seemed far off the mark, especially when it comes to other animals. But we would not wish to talk past each other either. We suspect that these esteemed colleagues may have been referencing secondary-process emotions based on learning (LeDoux and Rolls) and tertiary feelings that arise when cognitions and basic emotions are combined into complex amalgams (Damasio). These researchers have largely disregarded the possibility of evolved primary-process affects. Our main concern throughout this book is the nature of those ancient feelings that form the foundations of human emotionality. To provide a sketch of the current state of the field, we now briefly summarize the "classic" approaches of these prominent contemporary investigators of emotionality.

THE NEUROPSYCHOLOGICAL VIEWS OF ANTONIO DAMASIO

Damasio, who has done some spectacular human brain imaging of affective processes (Damasio et al., 2000), proposes a James-Lange type of sequence of events that precedes the emergence of affects. He proposes the existence of two primary maps, one of which (the *protoself*) stores information about the state of the body. The other primary map stores sensory information about the environment. A third mapping process (*core consciousness*) plays the role of linking information from both primary maps and ascertaining that some state of the environment has coincided with some change in the state of the body. This generates a *feeling of knowing* the environmental object. This feeling of knowing is a conscious experience, an "inner sense"; it is the "feeling of what happens" but it is not an affect. Damasio refers to this feeling of knowing as a *somatic marker* because the bodily responses of the protoself *mark* (evaluate) sensory stimuli in the environment. Core consciousness combines these stimuli and responses and generates the nonaffective feeling of knowing the object.

Damasio maintains that core consciousness is a fleeting phenomenon that is expressed in continuous unconnected pulses. When one adds the neocortical capacities of memory and sophisticated cognition to the mix, then the pulses of core consciousness can be remembered and one can make sense of them. Then consciousness becomes extended in time and it becomes autobiographical because an individual can remember events in his or her life. This allows for the ability to reflect intelligently on feelings about objects, a process that generates affects. Thus the personally meaningful generation of affect is a neocortical achievement.

Damasio believes that only a few primates are capable of generating such extended and autobiographical consciousness. Therefore, humans and a few of our close mammalian cousins are the only animals that are capable of fully experiencing affects. In his next to last book, *Looking for Spinoza*, Damasio (2003) went further and maintained, a few too many times, that "animals have emotional behaviors, while we humans have emotional feelings." Damasio's classic theory has fundamentally been a variant on the "read-out" or "feedback" theories of James and Lange, but it develops those theories in productive directions. Insofar as he speaks of the protoself maps that *store* information about the state of the body, Damasio at least

recognizes that the brain itself is capable of generating affects (even if he calls them "as if" affects, and situated all emotional feelings quite high in the brain). However, as noted, in his most recent writings, Damasio (2010) has explicitly accepted that animals do have emotional feelings, and that subcortical regions of the brain have the right stuff to contribute much to experienced feelings and hence consciousness. This has been Panksepp's position for four decades.

THE COGNITIVE NEUROSCIENCE VIEW OF JOSEPH LEDOUX

LeDoux, who has done some of the finest work on the brain mechanisms of fear conditioning in rats, also makes a distinction between emotion and affect, maintaining that emotion is a purely physiological response that is devoid of affect. Affect is something of an emotional afterthought that emerges when emotional physiology is read out by the parts of the prefrontal cortex that support *working memory*. The substrates of working memory are found in the dorsolateral parts of the prefrontal cortex, the most intelligent, or at least thoughtful, parts of the brain. Working memory can be seen as a mental workspace for *thinking about* current information (as detailed in Chapter 6). For example, as you read this paragraph, you keep some of the salient ideas in mind while you perhaps remember a relevant article that you read last week. All these ideas are items in your working memory. Working memory is therefore a highly intelligent function of the brain that can make sense of incoming information. When one makes sense of things, one consolidates many pieces of information into a coherent concept. LeDoux states that working memory performs a multiplicity of cognitive tasks, one of which is the creation of affects. According to LeDoux, the physiology of emotion (the behavioral, visceral, and low-level unconscious brain responses) is transformed into an affective feeling state in these cognitive regions of the brain.

It is important to note that LeDoux's research, which has focused almost exclusively on FEAR, also points to ancient subneocortical regions as an emotional-behavioral and autonomic (but not affective) substrate of fear. His research has revealed how the amygdala, a subneocortical structure long implicated in fearfulness, plays a central role in the generation of fear-conditioning but not feelings. The amygdala consists of more than a dozen

specialized cell groups, or nuclei, each of which performs a somewhat different function. The central nucleus of the amygdala plays a primary role in the downstream generation of unfeeling FEAR responses although, from the perspective of affective neuroscientists, it, along with other deeper structures (especially the periaqueductal gray), forms a part of the FEAR system. A few other lateral nuclei in the amygdala play their parts in conditioned learning but not in the generation of FEAR itself (for more details about the FEAR system, see Panksepp, 1991, and Chapters 5 and 6 herein).

Somehow, after LeDoux's 1996 book, it has become popular folklore to see the amygdala as the wellspring of all fear, indeed of all emotion—which is a sadly uninformed view. Individuals with totally damaged amygdalae (i.e., people with the congenital Urbach-Wiethe disease, leading to gradual calcification and destruction of the amygdala) can still experience worries, fears, and plenty of other emotions. Also, PLAY, GRIEF, CARE, and SEEKING arousals do not prominently involve the amygdala. Indeed, only one of the subnuclei of the amygdala, the central nucleus, is part of the primary-process emotional system that helps integrate the evolutionarily provided FEAR state with higher-order learning processes (yielding secondary emotions). In contrast, LeDoux, and other fear-conditioning theorists, consider the central nucleus of the amygdala simply to be the "output system" for a variety of fear responses (e.g., freezing, heart acceleration, increased blood pressure, fear-induced defecation and urination, and a host of other stress responses). LeDoux and other fear-conditioners have not yet explicitly considered that an integrative FEAR system, with its many descending and ascending components interconnecting the amygdala with many other brain regions, suffices to generate the *raw feelings* of fearfulness. They prefer to assume that emotional feelings emerge from higher regions of the neocortex (and LeDoux has claimed that he is interested in human emotional feelings as opposed to affective processes of animals). We disagree, because we do not believe that one can understand human emotional feelings without understanding those of our fellow animals.

THE BEHAVIORAL NEUROSCIENCE VIEW OF EDMUND ROLLS

We understand Rolls to maintain that, in animals, emotion is a nonaffective evaluation of various stimuli and that feelings only emerge when various bodily sensations are reinterpreted by tertiary-order brain processes (i.e., the neocortex) that elaborate symbolic functions such as language. His superlative research has focused on sensory processing, particularly the faculty of taste. He maintains that nonaffective emotional reactions occur in subcortical structures, including, in early formulations, some older cortical regions of the brain that evolved just before the neocortex. Overall, the assumption that *emotional* feelings are generated within higher cortical regions in the brain is at variance with the evidence showing that emotional systems that can elaborate rewards and punishments are located in much deeper brain regions. We think it is more likely that deeper structures program (or teach) the old cortical structures how to generate evaluations. For instance, in fear-conditioning, it is the arousal of the FEAR system (the so-called UCR) that permits conditioning to occur in the amygdala. In other words, the mere fact that newer cortical structures can generate evaluations does not eliminate the possibility of fundamental participation by deeper regions of the brain in generating the primary, raw feelings upon which secondary evaluations are based.

For the moment though we will stay with Rolls's formulation of how nonaffective evaluations of environmental stimuli, as generated by lower brain regions, can be transformed into phenomenal experiences. This supposedly nonaffective information, organized by the higher brain stem (the thalamus and hypothalamus), can be sent in two directions. The information sent in one direction will arrive at the basal ganglia—deep forebrain structures that control unfeeling instinctual behaviors such as those involved in eating and adopting a particular posture during elimination, sexual and aggressive stances, and so on. So, for example (according to Rolls), if a rat happens upon a piece of cheese, the rat's older brain structures would evaluate aspects of the taste and texture of the food. This evaluation would be nonaffective and the information it generates would be sent to the rat's basal ganglia, which would instruct the rat to continue eating the cheese. The nonexperienced information generated by older brain regions can also be sent in another direction, up to the neocortex (actually in this case, an older cortical region called the orbitofrontal cortex, right above the eye sockets). However, in his general formulation of emotional feelings, a large and complex cortex, such as that possessed by most

humans, is needed to construct a symbolic interpretation for the nonaffective lower-brain evaluations. This symbolic interpretation can be rendered in words. And these symbolic and linguistic transformations create the affective experience, which Rolls (following the lead of many philosophers) calls "qualia." In animals with humble neocortical endowments, such as rats, however, no affects supposedly accompany emotional behaviors. This is because such animals have rather little of the right kind of upper brain to generate symbolic concepts of emotional evaluations—which are presumably necessary to generate affects. For this reason, Rolls concludes that 'unintelligent' species have no emotional experiences—hence the animals we routinely study in the laboratory, certainly rats and mice, are not affective creatures.

To summarize, according to Rolls's general construct of consciousness, if you were to taste a spoonful of cheesecake made by a gourmet chef, various structures in the older brain regions (including the orbitofrontal cortex) would evaluate nonaffective information about the taste and texture of the cake. This information would be sent to your basal ganglia, which would instruct you to eat more cake. In addition, your old cortex would send the information to your neocortex, which would be able to symbolize and therefore speak about the delightful affective experience of eating this elegant confection. Thus, for Rolls, the ability to verbalize or at least conceptualize evaluations is a necessary condition for the affective experience. In his view, only human beings, along with a small number of other intelligent species, have affective experiences.

Perhaps the biggest problem with Rolls's formulation is that he uses sensory affects to discuss emotional affects, which in our estimation is a category error. At the same time, since writing his first book on emotions, he has provided a great abundance of human brain-imaging data that show how the orbitomedial frontal cortex (an old cortical region) participates in the generation of hedonic value as a response to food taste and texture variables, and also pleasant touch (Rolls, 2005). In short, his work applies more to the affects arising from sensory experiences than to the types of emotional circuits we discuss here.

CLASSIC AFFECTIVE NEUROSCIENCE VIEWS

Very briefly, since this view is summarized throughout the book, the classic affective neuroscience perspective envisions that ancient emotional circuits are concentrated in primitive regions of the brain, but with abundant linkages to higher brain regions. Emotional systems are *defined* in terms of the properties of these circuits, which have at least seven characteristics as summarized in Figure 2.1, including (i) a few unconditioned stimuli that can initially activate emotions, (ii) distinct unconditioned behavioral responses along with the triggering of diverse autonomic bodily changes to support these actions, (iii) the ability to gate and valuate concurrent incoming stimuli, partly by basic learning mechanisms (i.e., controlling incentive salience), (iv) positive feedback that outlasts the presence of the unconditioned stimuli, (v) regulation by higher tertiary-process cognitive functions and (vi) the emotional systems strongly influence higher mental processes, and (vii) this whole system generates distinct affective feelings, with the most important generators of the feelings being within the subcortical command circuits (as depicted in Figure 2.2). We would emphasize that one can *never* have a scientifically adequate verbal definition of primary-process emotions; such definitions must be based on neural circuit criteria that are successively refined as more and more replicable evidence is accumulated.

Of course, each primary-process emotional system (SEEKING, RAGE, FEAR, LUST, CARE, PANIC/GRIEF, and PLAY) has its own specific infrastructure that interacts with both inhibitory and synergistic relations with the other emotional systems, as well as a host of general arousal functions, as controlled, for instance, by vastly distributed acetylcholine, norepinephrine, dopamine, and serotonin systems, where the neurons are localized in the same ancient brain-stem regions in all vertebrates (see Figure 1.1 for general approximations). Each system is longitudinally organized, extending from lower midbrain regions to higher medial frontal cortical regions of the brain. All emotional systems tend to be situated near the midline, which highlights their very ancient status in brain evolution. Figure 2.2 provides a cartoon summary of the SEEKING system and its various functional connections (for anatomical connections, see Figure 3.1). The next chapter offers an in-depth discussion of this profoundly important emotional system. Dopamine lies at the heart of this vast emotional system, controlling practically everything that organisms do. Its interactions with

other brain regions are so extensive that it helps to facilitate most other emotional urges.

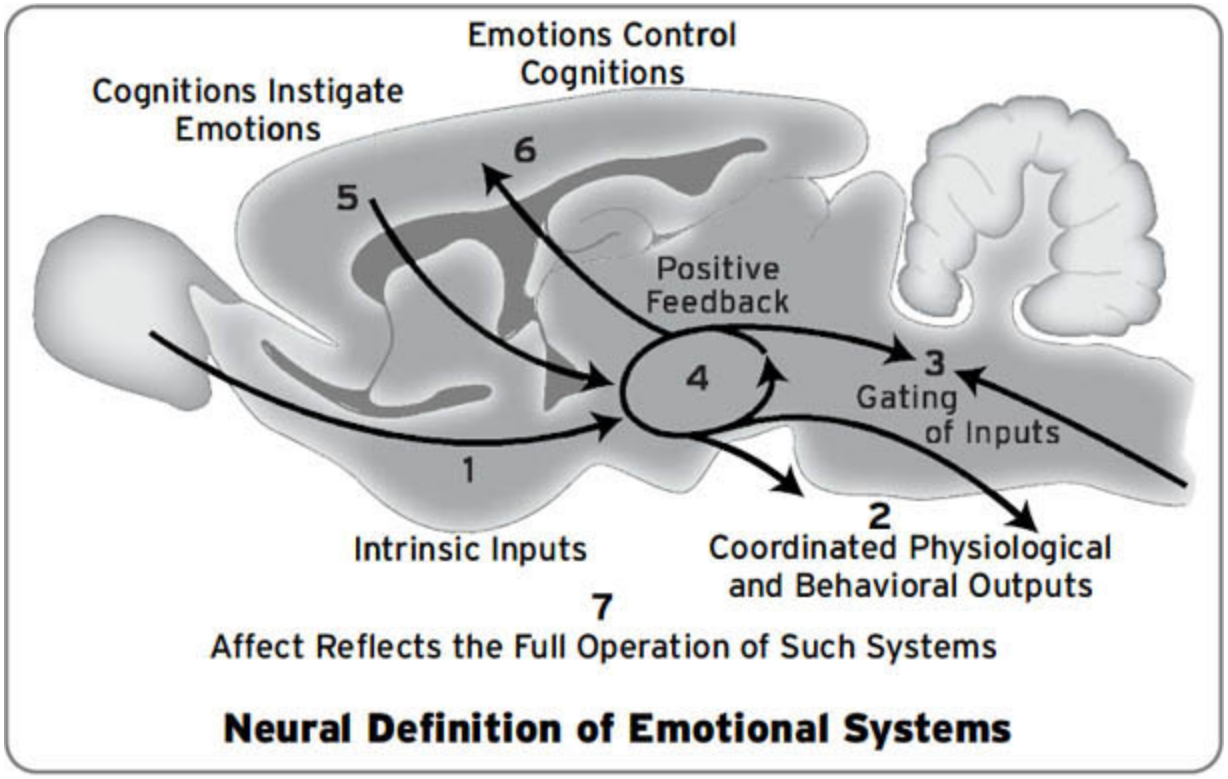

Figure 2.1. A schematic summary of the defining characteristics of basic instinctual emotional systems. They all have a few (1) intrinsic inputs, which behaviorists called Unconditional Stimuli (UCS); (2) various instinctual behavioral and bodily, especially autonomic-visceral, outputs, which behaviorists called Unconditional Responses (UCRs); (3) the input of various other stimuli into higher brain regions—potential conditional stimuli (CS)—if they predict rewards and punishments are controlled by emotional systems (yielding what some people call "incentive salience"); (4) emotions outlast the stimuli that activated the systems, whether external (UCS) or internal ruminations, such as those that arise from (5) higher cortical areas, especially in the frontal cortex activating or inhibiting emotions, and (6) emotional systems clearly have the power to control and modify higher brain functions. The affective feeling of an emotion is largely produced by an internal brain process summarized by attribute 4. Still, as highlighted by attribute 7, all of the other aspects of the system can modify and regulate the intensity, duration, and patterning of emotional responses.

Thus, the final affect is a consequence of the interactions of all of the BrainMind attributes that define each primal emotional network.

Likewise, norepinephrine, an even older system (since the cells are further down in the brain) facilitates attention during every kind of emotional arousal but more heavily so for euphoric feelings. Acetylcholine does the same but often for more negative emotions. Such general-purpose complexities need to be kept in mind for all of the primary-process "emotional-command" systems we will discuss in successive chapters. Much of the specificity of emotional responses are promoted by specific types of glutamateric (excitatory amino acid) influences in specific brain circuits, with a host of neuropeptides (chains of amino acids, see Figure 13.1) that promote specificity for many emotions.

The affective neuroscience approach does not envision emotional feelings being "read out" by higher cognitive brain functions, although there are pervasive interactions with those regions of the BrainMind. Affective states are part and parcel of each emotional operating system. However, this does not mean that higher cognitive mechanisms do not interact with or reflect on these ancient powers. Not only do the primal emotional systems regulate and motivate higher cognitive activities, but they are also surely states of great interest to the higher mental apparatus, which, depending on how children were reared, can often seem very perplexing. For instance, people diagnosed with borderline personality disorders (BPD), an adult developmental emotional problem, often have stormy social relationships, because of emotional insecurities, such as unregulated feelings of the PANIC/GRIEF system. These feelings can lead to "desperate attempts to avoid abandonment" that are paradoxically often "accompanied by efforts to downplay the importance of closeness and/or aggressive acts aimed at punishing significant others . . . leading to relationships marked by frequent arguments, repeated breakups and overall emotional volatility" with "difficulty sustaining cooperation" with others (see Bartz et al., 2010, p. 556).

Clearly, the higher brain can "fight" with the lower brain. In the above case, an overactive PANIC/GRIEF system may lead people to try to sustain self-esteem in self-defeating ways. One would think that oxytocin would mellow out such people, increasing their feelings of trust, but as the

aforementioned paper by Bartz et al. found, it actually reduced their feelings of trust and cooperation. Paradoxical findings like these are not uncommon when the higher, more rational, brain tries to cope with the changing affective terrain of the lower brain, and no one yet knows how to make sense of such unexpected results. Perhaps it reflects that many of us are a bit embarrassed by the intensity of our real feelings, so we cover them up, at times repress them to the extent that they are not even felt (a condition that may contribute to alexythmia). One would expect that with expert psychotherapeutic help, such individuals would be able to bring forth the more pro-social feelings of oxytocin (see Chapters 7 to 9) to help synergize the affective mind with cognitive perspectives that can have a mind of their own and that can often override the affective mind. Thus, it seems that the higher cognitive mind often does not wish to acknowledge, nor accurately read out, what is happening in the lower affective mind.

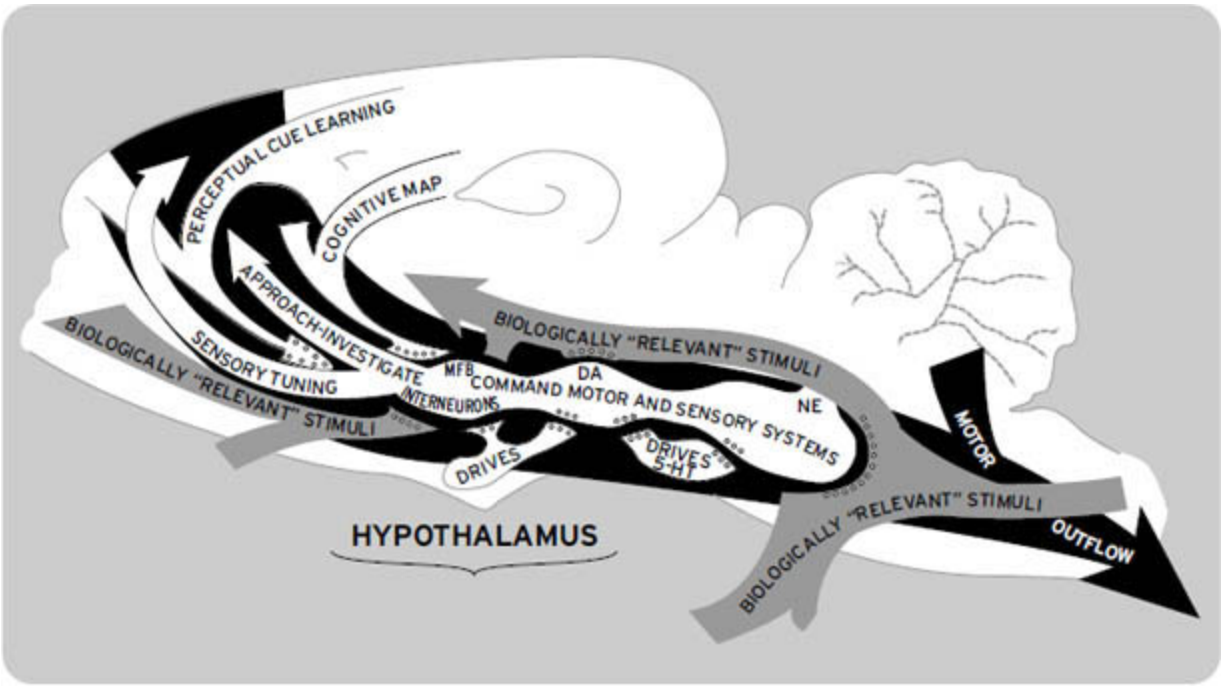

Figure 2.2. A semirealistic schematic conceptual description of what a full SEEKING system may look like in the brain, using an anatomical approximation of major interacting functions (adapted from Panksepp, 1981, with author's permission).

Problems With "Read-Out" Theories of Emotions

Professor Edmund Rolls, and many, many researchers working in non-biological fields of endeavor, maintain that we use words to generate concepts, which results in the semantic and conceptual construction of affects (e.g., Barrett, 2006). Who would deny that the higher mind can dramatically influence the lower affective landscape? However, much of the problem here, which can lead to bitter disputes, may simply reflect the fact that different theorists are discussing different levels of analysis in an ultra-complex, hierarchically organized set of MindBrain systems. It seems undeniable that all mammals share certain basic, primary-process emotional systems. To the best of our knowledge, the secondary-process learning mechanisms (e.g., classical and operant conditioning) are also remarkably similar across all mammals. However, as higher cortical cognitive regions evolved and diversified across species, the gateway to massive emotion-cognition interactions emerged. This gateway may be vastly different among different creatures. It is in this last realm of mind development where the largest scientific dilemmas arise. There has been a temptation among many theorists (who spend much of their own mental lives in the higher conceptual reaches of BrainMind processing) to put all psychological experiences within those highest realms of mind. This leads to the unjustified assumption that the lower brain functions are strictly unconscious. But that conclusion is simply not justified by the evidence (Merker, 2007; Panksepp, 1998a; Shewmon et al., 1999).

Clearly, scientists need to consider all the levels of emotion processing before concluding where the affective networks are located, which are complex enough to sustain experience. We feel it is better to envision how various levels of brain organization contribute to the complete emotional experience in terms of nested hierarchies (Figure 2.3). In this view, the lower BrainMind functions are embedded and re-represented in higher brain functions, which yield not only traditional bottom-up controls but also top-down regulations of emotionality. This provides two-way avenues of control that can be seen to be forms of "circular causality" that respect the brain as a fully integrated organ that can have dramatic intra-psychic conflicts. If, at times, it seems that we are not respecting this vision ourselves, it is simply because science is the intellectual discipline that aims

to pull things apart, so as to understand the details of complex mechanisms and processes. It is an epistemology that cannot yield detailed understanding without breaking the whole into parts, albeit without typically having the wherewithal to reconstruct the whole from the parts. Everyone who has ever stripped down an internal combustion engine to see how it works, knows that putting all those pieces, littering the driveway, back together into a working machine is a more daunting task. The social constructivists typically do not have the opportunity to study the brain in any detail, but they seem to believe that their descriptions of emotional conceptual "wholes" are dealing with the same issues as those who seek to understand how the brain actually works.

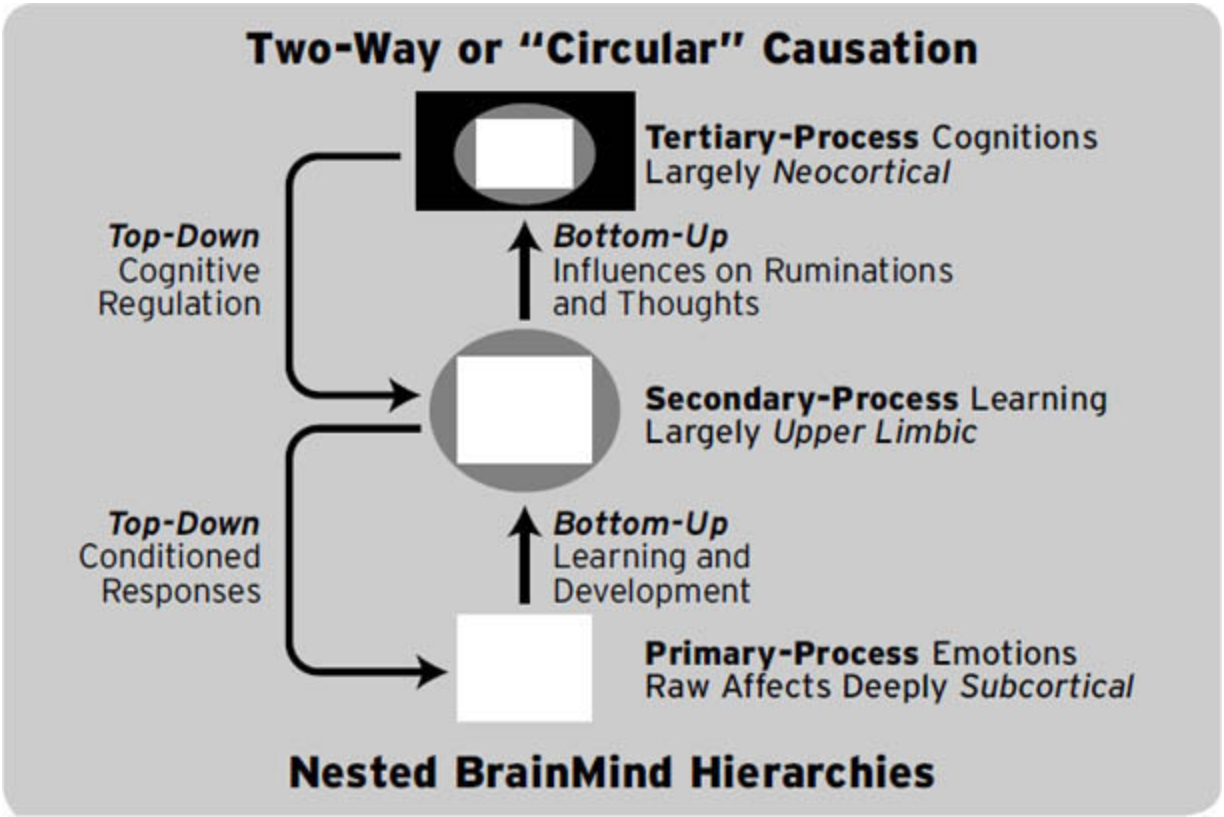

Figure 2.3. A summary of the hierarchical bottom-up and top-down (circular) causation that operates in every primal emotional system of the brain. The schematic summarizes the hypothesis that in order for higher MindBrain functions to operate, they have to be integrated with the lower BrainMind functions, with primary processes being depicted as squares, secondary learning processes as circles, and tertiary processes, at the top, as

rectangles. Please imagine each symbol being color-coded, to better envision the nested hierarchies that integrate the various levels of the BrainMind (adapted from Northoff et al., 2011).

In fairness, the social and personality psychologists, who have traditionally sided with *social constructivist* visions of mental life, have recently started to postulate preconceptual foundations for affects. Some who have limited their vision to dimensional views of emotions have suggested that some kind of primordial "Core Affect" which ranges from negative to positive (valence) is the fundamental process from which all other emotional feelings are constructed (Russell, 2003). This aspect of their views is provocative and to be welcomed, notwithstanding the fact that they often do not adequately consider the available evidence from cross-species affective neuroscience research (for a relevant published debate with commentaries, see Zachar & Ellis, 2012).

Social constructivists have traditionally maintained that concepts and language are the hallmarks of the affects, and many still do. If an animal cannot conceptualize, it cannot experience affects. A concept is an abstraction, usually gleaned from a multiplicity of experiences. For example, the concept of a chair is drawn from seeing many different kinds of chairs, and the word *chair* represents the overall category. The first time that you ever saw a chair, you might not have known what it was, because you certainly did not yet have a concept that it was a good place to rest. You had to learn that every individual chair is a constituent member of the broader group—leading you to conceptualize what a chair is.

Rolls has also suggested that nonaffective evaluations somehow become concepts too and that affects are created when you put these concepts into words. Only intelligent animals can do this, which is why he believes that only they can experience affects. We suspect this may not make sense evolutionarily, for we know that people experience pain before having the concept of pain. And so forth for all the primary-process emotions that we will discuss here.

However, some words represent concepts and others do not. As we suggested in the previous chapter, when you first saw the color red, you rapidly came to know all that you would ever know *directly* about this color. Your visual experience was not abstracted from other experiences,

except to the extent that your visual system is constructed progressively during development. Seeing red (or yellow or brown) is not a concept. Because you are intelligent enough to manipulate symbols in the form of language, you can use words like *red, scarlet, crimson*, and *ruby* to differentiate and label nuanced differences in your experiences. But the raw phenomenal experience of seeing red does not require intelligence. So words like *chair* represent intelligent concepts, while other words like *red* represent primary experiences that require no intelligence except, of course, if you wished to label the experience.

We maintain that basic affects are in a category of primary experiences, like seeing a color, and that language merely labels and represents such experiences. But affective experience itself, like seeing the color red, does not require any conceptual intelligence. Humans can use words to label their affects, but they do not need words to experience them. Thus, our use of words does not necessarily mean that other animals need to be competent with verbal concepts in order to experience affects. Primal affects are surely *prelinguistic* experiences—experiences common to all mammals and perhaps to other animals as well (Huber et al., 2011).

Damasio's (1999) sophisticated view of affective consciousness, in which he draws a line between lower unconscious processes and higher conscious processes that are fairly high in the brain, remains to be corroborated by empirical evidence; he has never been clear on what the critical tests of his theory would look like. Although his somatic marker hypothesis—the fact that information from the body is transformed into feelings that guide actions—has garnered much experimental attention (with a mixed track record so far), few of these experiments have actually monitored the time courses of affective change in the human subjects being studied. Also, neuroscientists do not yet have clear ideas about the details of the two primary maps Damasio postulates: one for changes in the body and the other for the external world. Nor do they yet know whether these maps are synthesized by the higher-order mapping that he calls *core consciousness*. Further research will be needed to test the idea that core consciousness generates the inner emotional feeling of what is happening by synthesizing information from maps about the body and about the environment. We tend to disagree with his 1999 view and not only because it was not spelled out in sufficient detail. Again, however, we were pleased to see that by the time this book went to press, Damasio (2010) had made a radical shift in his

views: He now accepts that subcortical structures do contribute to affective experiences of various kinds, a view that has had solid empirical support for almost half a century.

LeDoux claims that affects emanate from the parts of the neocortex that support working memory: the dorsolateral frontal regions. Yet there is now abundant evidence that during strong emotional states, the human brain exhibits *reduced* arousal of dorsolateral frontal regions, the regions that LeDoux and others have identified as substrates of working memory (Goel & Dolan, 2003; Liotti & Panksepp, 2004b; Northoff et al., 2009). Conversely, these dorsolateral frontal areas are most aroused when people are involved in cognitive, *nonemotional* pursuits. How can the dorsolateral frontal cortex be the font of affective experience if this area is so relatively quiet during emotional episodes? We would agree that this is the main area of the brain where we humans think about our emotional experiences in a cognitively reflective way, but it is likely that the more ancient medial frontal regions are the brain regions where we ruminate, and dwell, on our emotional troubles and other feelings. This medial part of the brain is commonly overactive in depressed people (Northoff et al., 2011).

Those who subscribe to read-out theories generally maintain that affects are cognitive constructs. Yet, to the best of our knowledge, the neocortex (the premiere cognitive structure of the brain) cannot generate affects when it acts alone. All three of the researchers discussed above seem to agree that information about perceiving a stimulus and the body's responses are nonaffective. But how can nonaffective information, interpreted by neocortical systems that cannot generate affects on their own, create a conscious affective experience? Read-out theories are riddled with problems and contradictions. And each of these contemporary theories, in their classic forms, chose to leave the other animals outside the charmed "circle of affect"—the capacity to experience and respond to events with feelings like eager anticipation, anger, anxiety, sexual feelings, maternal warmth, the psychic pain of separation, or playful social joy. We will show that an abundance of existing evidence argues otherwise. Indeed, if one reads Rene Descartes' *Passions of the Soul* carefully, it is clear that even the father of dualism probably accepted that other animals do have some coarse feelings; they simply do not have enough "res extensa" (higher mental abilities) to reflect thoughtfully *about* their primary-process mental conditions. If only that had been noticed and emphasized by many other

opinion leaders, perhaps research on the affective aspects of animal minds would have flourished. If only the James-Lange theory had not been so attractively counterintuitive, wonderfully stimulating to creative minds but with no robust (causal) scientific support to this day, would not the other animals have been bequeathed their emotional feelings by now (hopefully by behavioral neuroscientists most of all, since the general public is often appalled and at times chuckles when scholars can't handle such "no brainers")? If behaviorism had not been so arrogant about the denial of emotions, we would probably now have a rich understanding of human emotions, as opposed to the lingering false belief that affects are just a variety of higher mental abilities. Our higher (neocortical) mental functions can create art and madness out of our emotions, but they cannot generate feelings *on their own*. That is what the data has strongly indicated for a long time.

HARD EVIDENCE FOR THE EXISTENCE OF EMOTIONAL AFFECTS IN OTHER ANIMALS

It is all well and good to address weaknesses in the positions of other theorists. We can explain the various ills of behaviorism and the failings of the read-out theories. But none of these critiques entitles us to say that affects are primary, noncognitive, prelinguistic experiences. Science is not rhetoric. Only brain research, along with careful psychological experiments, can allow us to make this assertion. The following is a thumbnail sketch of some of the hard evidence derived in this way, on which we will elaborate more fully in the subsequent chapters, where we first discuss the SEEKING system, and then successively RAGE, FEAR, LUST, CARE, PANIC/GRIEF, and PLAY. This hard evidence allows us to conclude that other animals are indeed affective creatures and to advocate the minority view that if we understand their emotional feelings, we will have a solid science of the ancestral sources of these BrainMind powers in our own lives.

If this argument is valid, then behavioral science researchers made a big mistake in discarding emotional feelings from the study of organisms they wished to understand. In fact, their main concepts for training animals—rewards and punishments that "reinforce" learned behavioral change—may have operated successfully largely because of the unacknowledged affective

principles within animal brains. As soon as we recognize this as a high-probability neuroscientific fact, as opposed to just a supposition, we can have a revolutionary transformation in the way we use the knowledge that preclinical animal models can provide to understand human emotions and their many disorders. And none of that requires throwing away any of the superb knowledge that has been obtained about the behavioral, neurophysiological and neurochemical mechanisms of the brain harvested by many behavioral neuroscientists who will not tolerate talk about animal feelings.

So how did physiological psychologists "stumble" upon the facts that allowed us to conclude that animals do have emotional feelings? In the middle of the last century, James Olds and his colleague Peter Milner made the remarkable discovery that all animals, at least all animals that they tested (and all that have been tested since that time), would work intensely, to the point of exhaustion, in order to obtain electrical stimulation in the medial forebrain bundle-lateral hypothalamic area (MFB-LH), which is a remarkably extensive system, as succinctly described by Jim Olds (1977, published posthumously). Figure 2.4 is one of the earliest realistic depictions of this system in the rat brain. This system connects lower, middle, and upper brain regions. It is one of the most important brain systems for behavioral as well as psychological coherence.

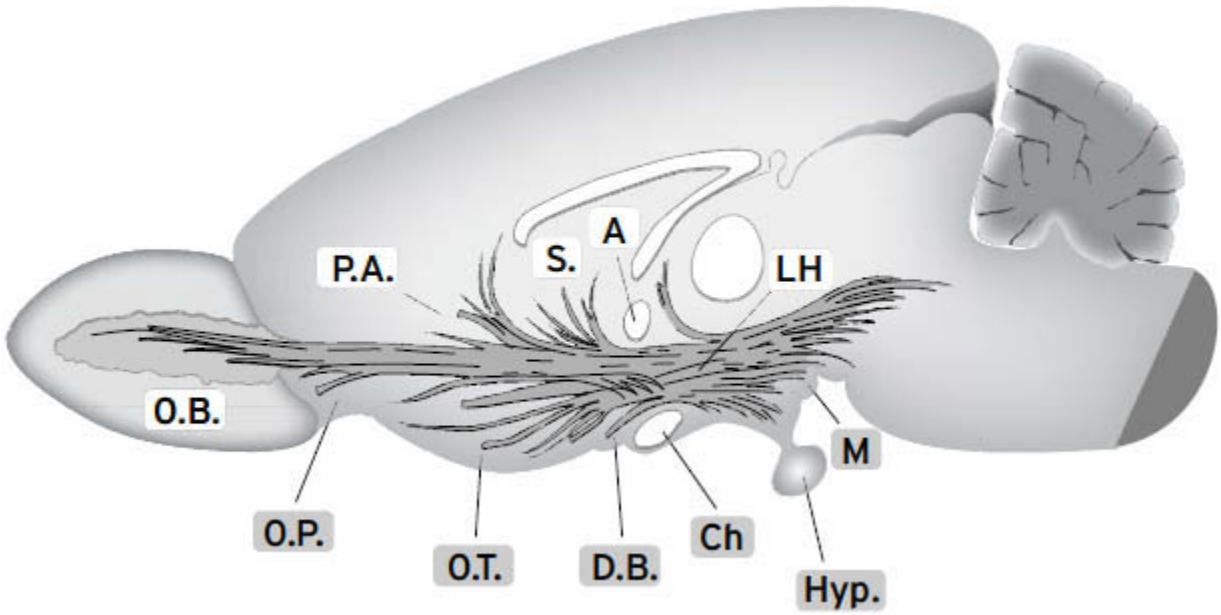

Figure 2.4. A schematic summary of the medial forebrain bundle (MFB), connecting central regions of the midbrain with higher brain regions. The MFB runs through the lateral hypothalamus (LH) situated just above and to the right and left of the optic chiasma (Ch), with the remaining anatomical nomenclature highlighting olfactory bulbs (O.B.), olfactory peduncle (O.P.), paraolfactory area (P.A.), olfactory tract (O.T.), diagonal band of Broca (D.B.), anterior commissure (A), the pituitary gland, or, as it used to be called, the hypophysis (Hyp.), and mamillary bodies (M). In the midbrain, parts of the descending branches of the MFB project to medial regions such as the periaqueductal gray. This classic figure is adapted from Le Gros Clark et al. (1938).

And, in retrospect, it is not surprising that stimulation of this complex network would be rewarding. But in 1954, it was a spectacular discovery that swept like wildfire through the field of psychology. The inclination of animals to work persistently for arousal of this system, usually by performing a task like pushing a lever, was called *self-stimulation*. This discovery was all serendipity; Olds and Milner were looking for ways to enhance learning with brain stimulation. But they were wise enough to shift their focus and to intensely investigate what this new phenomenon was all about (no doubt using their own SEEKING systems). Clearly there was something highly rewarding about this kind of stimulation. Why else would animals work so hard? It seemed reasonable to suppose that they had found "the reward system" of the brain, and that exorbitant idea survives, as the "definitive" concept, to this day. Even though there are many reward systems in the brain, there is only one that drives the animal energetically to seek all the other kinds of rewards, mainly sensory and homeostatic rewards, that it must discover in the world in order to survive. That is why we have long advocated giving it a more appropriate, albeit unusual, emotional name (Panksepp, 1981b, 1982)—first the EXPECTANCY and now the SEEKING system.

When we examine the SEEKING system in some detail in the next chapter, we will see that its hub is located in neural networks arising in low regions of the brain, including the ventral tegmental area (VTA) and the lateral hypothalamus (LH). In that chapter we will explain that the SEEKING system generates energetic exploration and foraging, along with

affects that can be better described as euphoric excitement rather than reward or pleasure—the feeling is one of anticipatory-expectant eagerness and, at a more cognitive level, the engendering of discrete expectancies. It is these highly energized, euphoric-foraging engagements with the world that animals find so rewarding. These are feelings that lie at the very heart of what some might call joyous aliveness.

In the middle of the last century, however, the SEEKING system was unknown; the only kinds of rewards that scientists thought about were those associated with the restoration of homeostasis. So food, water, warmth, sexual consummation, and so on were seen as rewarding experiences because they restored homeostasis in the body (a key idea of drive-reduction theorists). Even behaviorists as radical as Skinner saw homeostasis (drive reduction) as rewarding. The influences of homeostatic imbalances engendering hunger and thirst can be scientifically measured physiologically, for example, in terms of low blood sugar or low blood volume, and behaviorally in terms of increased food and water intake. This can all be done without ever needing to refer to affective or motivational states like hunger or thirst (Skinner, 1953).

Behaviorists observed that homeostatic imbalances, such as low blood sugar, would render an animal more inclined to work in exchange for food. However, most behavioral investigators eventually found that the sensory properties of rewards—*incentive properties* such as the quality, quantity, and delay of rewards—were much more important in controlling learning than changes in the homeostatic states of the body. In other words, the better the sensory rewards, the more rapidly would animals learn. Drive reduction alone is not as effective. For instance, although hungry animals will readily learn to work for tasty meals, it takes them a long time to learn to self-inject food directly into their stomachs, even though most will eventually learn even that with prolonged training (Mook, 1989).

A SHORT HISTORY OF THE SEEKING SYSTEM

Since the SEEKING system is the most thoroughly studied emotional system, albeit under the rubric of "the brain reward system" let's briefly discuss its characteristics, without any scientific references, which are easily found in great detail in many sources (including Panksepp, 1981b,

1998a), as well as the next chapter. We introduce this system here since this may be the most difficult one for both scientists and interested readers to understand, and because it is so important for all of the other emotional systems to function properly. Thus, this short sketch is a foreshadowing of much that is to follow in the rest of this book.

Unlike behaviorists, who thought about rewards only in terms of behavior, neuroscientists were interested in brain function. So when Olds and Milner discovered that animals would work especially hard to obtain MFB-LH stimulation, the news swept through psychology. Many physiological psychologists (as they were called in those days) started to assume that the MFB-LH was the common substrate for all manner of homeostatic and sensory rewards. With this thinking in mind, scientists who started studying the phenomenon, like Panksepp, originally assumed that electrical or pharmacological stimulation of the MFB-LH was rewarding because it corresponded to all manner of consummatory rewards. In other words, when one part of the MFB-LH was stimulated electrically, an animal's brain would respond in the same way as it did when the animal had a good meal. Another part of the MFB-LH would respond as it did when the animal quenched its thirst. Yet another part of the MFB-LH would respond in the way it did when the animal engaged in rewarding sexual activities.

However, the experimental evidence did not follow the expected pattern. When an animal finds resources that it needs and starts to engage in consummatory activities such as eating, drinking, or sexual activity, neuronal firings along the MFB-LH temporarily but dramatically slow down (Hamburg, 1971). This suggests that the reward afforded by MFB-LH stimulation is active *before* homeostasis starts to be restored. Indeed MFB-LH is most active when people and animals are in a state of homeostatic *need* and there are opportunities for finding good feelings in the environment.

So what kind of reward might actually be afforded by MFB-LH stimulation? It is certainly not *just* a homeostatic or sensory reward, although the system does respond to those events. A clue can be gleaned directly from the unconditioned behaviors that animals exhibit when they receive such brain stimulation. Rats get super excited when they are self-stimulating. And if one simply gives "free" electrical jolts to the MFB-LH without the rats having to work for them, the animals move about, eagerly

investigating their environments, even monotonous ones, such as an empty box. They explore all environments as if they are looking for something. Also, it has always been a puzzle why animals press levers during self-stimulation of the MFB-LH much more than they need to in order to get all the "rewards." They appear to do so because they are simply so overexcited, which is not the same as a state of pleasure arising from consuming rewards.

The MFB-LH is not the only part of the brain that will cause animals to self-stimulate. For example, animals will press levers to self-stimulate the septum. But they do so in a more methodical way, usually pressing the lever once for each electrical jolt, rather than pressing the lever many more times than necessary. In other words, something about MFB-LH stimulation causes a state of excitement. In fact, animals can tell the difference—they can discriminate—between septal and MFB-LH stimulation (Stutz et al., 1974). Clearly, the two sites of stimulation are generating distinct experiences. When the *human* brain is stimulated in the septum, people often report sexual feelings. When they are stimulated in the MFB-LH, they report more general feelings of excitement and anticipation—feelings that are hard to put into words. While the septal stimulation does participate in the consummatory-orgasmic reward of sexual activity, the SEEKING system of the MFB-LH elaborates the appetitive eagerness phase of sexuality as well as the anticipation of all other rewards.

The conclusion is inescapable. At a cognitive level, the MFB-LH provides an affective reward in the form of a euphoric general state of *expectation*, initially with no explicit goal in mind. Stimulation of the MFB-LH certainly does not produce brain states that correspond to those we feel when our bodily imbalances are restored toward homeostasis (i.e., feelings of *satisfaction*). When animals are satisfied, they tend to fall asleep. MFB-LH stimulation keeps animals awake. With MFB-LH stimulation, animals appear enthused and are keen to explore their environments. And people accordingly feel more interested in the world and make future plans—clearly a state of high-hearted expectation. No one reports a distinct feeling of experiencing a sensory pleasure, such as a wonderful taste. MFB-LH arousal generates a reward that is closer to euphoria than to any sensory-bodily pleasure.

Furthermore even if an animal has been *decorticated*—surgically deprived of its neocortex—it will still work to the point of exhaustion in

order to receive MFB-LH stimulation. Therefore the rewarding affect cannot emanate from the neocortex, because these animals do not have any neocortex. One is obliged to conclude that subneocortical structures generate these affective rewards, in the form of euphoric affective consciousness—a subjective feeling state that people and animals desire so much that they will work to the point of exhaustion in order to achieve it.

In everyday life, the MFB-LH, along with the rest of the SEEKING system, is typically more aroused when animals are in a state of homeostatic imbalance, but it is the ready availability of goodies in the world ("incentive stimuli," as scientists put it) that really turns the system on. Everyone knows that all the major homeostatic imbalances of the body feel unpleasant. Conversely, interacting with incentive stimuli, which evoke the delightful feelings of ingesting rewards, not only predicts restoration of homeostasis, but also provokes experiences of pleasure (Cabanac, 1992). But "the reward system" is not doing that for us. It is doing something equally important—it is allowing us to pursue rewards with gusto. SEEKING, a much better name for this system, generates the overriding sense of expectant euphoria that prompts people and animals to search for the resources that they need. This system not only helps animals satisfy bodily needs but also, as we now know, many other higher-order emotional needs, ranging from a desire for money and information to music and other aesthetic experiences.

The other six emotional systems do not lend themselves to this kind of homeostatic explanation, since they are not as intimately linked to satisfying bodily needs. The other emotions are related more strictly to intrinsic aspects of the BrainMind, but all of them require one to seek environmental resources. Thus, to some extent, all the other emotions also rely upon the psychobehavioral push of the SEEKING urge. In a sense, SEEKING is the "granddaddy" of all the emotional systems. To satisfy LUST, one must seek relationships. To feel tender loving CARE, one must seek to help those who need help, especially babies. To feel full RAGE, one must seek to harm those who would take resources away from you. To respond well to FEAR, one must seek safety. To make your PANIC/GRIEF work for you, you must seek out those who would support your needs. To PLAY with great joy, you must find friends.

Clearly the affect that accompanies artificial arousal of the SEEKING system emerges from subneocortical regions, as highlighted by the survival

of self-stimulation after massive forebrain damage (Huston & Borbély, 1973, 1974; Valenstein, 1966). This has long called into question all read-out theories, with their claims that affective experience is a neocortical achievement. It is not. Of course, the neocortex may help construct complex emotions (tertiary-process emotions) from the more primitive affective phenomena, a very interesting neuroscientific topic in itself, but currently we know little about how that really occurs. The above analysis also should have put an end to a long-standing behaviorist bias in animal research: that other animals are not affective creatures.

Although there is more relevant data available for the SEEKING system than any other, a study of each of the other primary-process emotional systems supports the same overall conclusion—raw emotional feelings arise from subneocortical networks of the brain that generate instinctual emotional action. And all other mammals are affectively alive, just as we are. But we should not claim their feelings are identical to ours—evolution always engenders variability in details—but we all do have primal feelings that are in the same general categories. In some species, some feelings are stronger or weaker than others, but they are all there to some extent. They lie at the foundations of our mind. If so, we can understand the general principles and sources of our own emotionality, if we study these systems, in great detail, in our fellow animals. Rats and mice will do just fine for much of this research. And the work can be done well, with very little stress to animals. Many of these instinctual emotional systems can be studied in anesthetized animals (Panksepp, Sacks, et al., 1991; Rossi & Panksepp, 1992). Indeed, decorticated animals exhibit all seven of the primary-process emotional behaviors (Kolb & Tees, 1990; Panksepp et al.,, 1994).

FURTHER SUPPORT FOR EMOTIONAL AFFECTS IN ANIMALS

All animals that have been studied demonstrably like or dislike the affective feelings generated by artificial activation of the emotional systems discussed in this book. This helps us understand why various UCSs and the provoked UCRs are so important for learning. Just as animals gravitate to places (exhibit *conditioned place preference*) where they have previously had positive incentive experiences, such as eating, drinking, or engaging in sex, they show similar preferences for environments in which they received

artificial activation of the circuits that promote those behaviors. Conversely, they avoid places (they exhibit *conditioned place aversion*) where they have had unpleasant affective experiences. They stay away from places where they have been frightened or hurt; it does not matter whether those emotions are produced by environmental events or by artificial activation of the brain systems that generate those types of affective behaviors.

Other related experiments indicate that affects emanate from subneocortical regions of the mammalian brain. For example, animals exhibit preferences for places where they have taken drugs of abuse—drugs that induce pleasurable or desirable affective states in humans. The critical networks for these effects are subcortically situated. It is only because they influence brain affective systems that addictive drugs can be used in animal research to understand the brain mechanisms of human addictions. The implicit assumption of most researchers is that animals seek these drugs for similar *affective* reasons, rather than just learning about "rewards," but this is rarely acknowledged (for exceptions, see Kassell, 2010). We now know that such drugs achieve their effects in humans by mimicking neurochemicals that generate specific types of feelings in our brains. It is unlikely to be any different in other animals. However, addiction has an additional property—an opponent process, namely a dark affective hole is left behind when drugs wash out of the system. And that horrible aftereffect grows larger the more one consumes certain drugs, like amphetamines, cocaine, and opiates. Getting rid of those negative feelings may be more important in creating addictions than the good initial feelings produced by certain drugs (Koob & Le Moal, 2001).

Drugs of abuse fall into two categories: those that pharmacologically stimulate the SEEKING system and those that mediate sensory pleasures, including the neurochemical suppression of the PANIC/GRIEF system, which engenders warm feelings of social bonding. Drugs like cocaine and amphetamines primarily enhance the effects of dopamine, which stimulates the SEEKING system, evoking the same sense of enthused anticipation that is afforded by electrical stimulation of the LH. Opiates, like morphine or heroin, are chemically similar to endogenous brain chemicals that mediate sensory pleasures and the formation of positive social relationships (Panksepp, 1981a, 1998a). This is why grooming is rewarding in monkeys (Keverne et al., 1989) and why the company of good friends and loved ones arouses feelings of comfort and relaxation for us. As we will see in Chapter

10, when opiates are administered directly into the brain, they stimulate emotional feelings like those experienced from positive social bonds, as well as many other affectively desirable incentives. Other brain systems, such as those based on oxytocin, have more recently been found to produce similar effects.

But opioid systems are all over the brain. Why should we believe that such good feelings are generated just by brain systems that lie below the neocortex? With animal research one can evaluate such questions directly by infusing opiates into specific brain regions. Animals display preferences for morphine infusions into primitive subneocortical brain regions, such as the periaqueductal gray (PAG) and the VTA—brain regions that send pathways through the MFB-LH—but they do not display preferences for such infusions into other higher brain regions, even though all those regions have abundant opiate receptors (Olmstead & Franklin, 1997). The fact that animals display place preference in response to this drug specifically when it is injected into deep subneocortical regions indicates that these deep structures generate the rewarding affects—that is, affects that animals like to experience. The fact that animals do not show such preferences when the same amount of morphine is injected into many higher regions of the brain, including the cortex, indicates that those regions probably do not have a comparably high capacity to generate rewarding affective feelings.

In addition to displaying place preferences for opiates, animals also display a willingness to work in order to receive doses of morphine and cocaine that are administered directly to deep medial subcortical loci of the SEEKING system (Ikemoto, 2010). Thus, the findings with chemical and electrical stimulation of the brain match up. Similar effects are seen with other drugs placed into other emotional systems. But the overall amount of data diminishes as one goes from neural networks that mediate the SEEKING urge to those for the other emotions. This does not reflect contradictory evidence; it only reflects the fact that much of the necessary research still remains to be done.

Animals can also display their likes and dislikes (their preferences) with vocalizations. We all know from observing our pets that animal vocalizations indicate specific pleasures or displeasures. We recognize the joyful yipping bark of the family dog when we get home from work, and we understand the angry, growling bark when a stranger is nearby. We easily distinguish the contented purr when we stroke our cat from its screech when

we accidentally step on its tail. We have no problem interpreting the pitiful wails when we leave a dog at a kennel and the hissing of a vexed cat. All these emotional vocalizations arise from subcortical regions of the brain, enriched with very similar anatomies and neurochemistries across species (Burgdorf et al., 2007; Brudzynski, 2010; Jürgens, 2002; with extensive summaries of early work in Newman, 1988).

Much of the recent scientific work on emotional vocalizations has been done with rats. For example, when rats play with each other or are tickled, they emit high-pitched (ultrasonic) chirping at 50 kilohertz (kHz). A similar frequency of vocalization is emitted when rats, both males and females, are anticipating sex or any of a variety of other treats (Knutson et al., 2002; Panksepp, Knutson et al., 2002). Accordingly, wherever in the brain 50-kHz calls are artificially induced through electrical brain stimulation, rats will self-stimulate those electrode sites (Burgdorf et al., 2007). In contrast, when the rats are socially defeated or when there is danger around (for instance, a cat is nearby), rats exhibit long 22-kHz "complaints" or "alarm calls." These are especially prominent in between the successive administrations of foot shocks in fear-conditioning studies, and when a safety signal is sounded, indicating no pain is forthcoming, the rats sigh (Soltysik & Jelen, 2005). Surprisingly, following copulation, a male rat also emits 22-kHz vocalizations. Perhaps, just perhaps, this is the vocal report that lets a female know that he is no longer in the mood for socializing. Alternatively, perhaps the animal is sending out a bogus "alarm call" to keep other males at bay (rats are promiscuous) and hence increase the chance (without thinking about it, of course) that he will be the father of the female's next set of "babies."

These facts about self-stimulation, place preference, and the circuits for emotional vocalizations and other instinctual-emotional behaviors allow us to conclude which brain regions are most important for the generation of raw emotional experiences. This kind of evidence is of critical importance for a factually based understanding of how the brain generates all of the primary-process affective states, whether sensory, homeostatic, or emotional.

SENSORY, HOMEOSTATIC, AND EMOTIONAL AFFECTS

Do animals experience primary-process affects other than the basic *emotional* ones? There is adequate evidence to finally consider the nature of sensory pleasures and discomforts (i.e., *sensory affects*, such as the pleasures of taste and the distress of pain), as well as affects arising from imbalances in the body (*homeostatic affects*, such as hunger and thirst). However, we will not focus on sensory and homeostatic affects extensively in this book, even though we think it very likely that animals feel them intensely.

Why? Largely because the database for these affects is less extensive than for the emotional ones, and those systems may not be essential foundations for consciousness itself. Scientists also lack fine manipulations, such as localized electrical and chemical stimulation of relevant brain regions to clearly evoke such states in animals, and thereby to conduct *causal* experiments on the affective qualities of those states. Most of the available evidence is in the *correlative* rather than the *causal* or *constitutive* domain, so we know what kinds of behavioral and brain changes occur when potentially hedonic stimuli are presented to the animal, but we do not know which of those changes actually causes the associated affects. In the absence of such data, one is left with the logical dilemma of arguing for causal links on the basis of correlative observations.

In any event, there is a growing substantive scientific literature on homeostatic affects, as garnered especially with human brain imaging. Brain-imaging evidence from humans highlights that thirst, hunger, and all of the other "bodily-visceral" feelings are elaborated in deep subcortical structures that regulate these same processes in animals (Denton, 2006). Likewise, the fascinating literature on the electrophysiological correlates of taste (Rolls, 2005) highlights, with great subtlety, the possible nature of sensory affects. But it is a category error to assume that these findings will explain emotions.

We are not yet certain where sensory affects are inaugurated. The most likely answer is that they are generated at many levels of the nervous system, perhaps even in the neocortex. Some of the best understood systems are those that mediate taste (Berridge, 2000, 2004; Steiner et al., 2001). Thanks to work on this system it is clear that in laboratory rats positive taste qualities such as sweetness are mediated to some extent by deep brain-stem structures in the basal forebrain and around the globus pallidus. Kent Berridge and Susana Peciña of the University of Michigan have identified

specific regions of the basal forebrain (the ventral pallidum) as the epicenter for neural processing of sweet tastes (Peciña et al.,, 2006). Sometimes, investigators imply that such subcortical regions simply process gustatory information, which is transformed into tasty feelings somewhere higher in the brain, such as the insula, which is clearly important for feelings of disgust (Craig, 2003a, 2003b). It is widely recognized that many incoming sensory systems split in two as they reach the thalamus, where affective aspects of the stimulus diverge into various subcortical systems, while more cognitive information that allows us to thoughtfully discriminate various sensations moves on to the neocortex (see Sewards, 2004 for taste, but the same principle applies to pain, touch, and so on). We suspect that the lower brain regions themselves suffice to generate the raw affective taste experiences they studied. But the question of the brain substrate for most sensory-affective experiences is not as easy to resolve at present as is the question of the brain substrates for primary-process emotional affects.

BRAINMIND EVOLUTION AND HIGHER FEELINGS

As the brain has evolved, newer structures have supplemented the functions of older ones (Figure 2.3), leading to hierarchical controls that shift with development (Figures 1.4 and 1.6). So it is likely that earlier in evolutionary history the affects associated with emotional, homeostatic, and sensory experiences emanated strictly from deep subneocortical regions of the brain, and with brain evolution, they have come to be elaborated by more recently added brain networks. Perhaps the generation of some affects has even been "taken over" by the higher neocortical areas, but we are working in the dark with such suppositions. We can be certain only of the fact that early in infant development all animals are more dependent on the functions of the lower than the higher brain structures (Chugani, 1998).

It is likely that, during maturation, deeper parts of the brain can program —or "teach"—more superficial structures how to function in particular ways (Figure 2.3). So it is possible that certain primary-process affects are initially elaborated subneocortically and that in the course of individual development these functions are refined, and perhaps in some cases, taken over by newly evolved higher brain regions. If so, it is likely that most affects are heavily influenced by more recently evolved functions of the

brain. This is surely especially relevant for certain sensory affects that are highly cognitively mediated (e.g., those cultivated for expert wine tasting). But in allowing subtlety of feelings, this may often be at the expense of intensity of feelings (i.e., cognitive regulatory functions more often dampen primary-process feelings than amplify them). But here we are completely in the land of speculation.

In contrast, we can be confident that emotional feelings are more intense at the lower reaches of the brain than higher ones—for one simple reason: In every mammal that has been studied, including humans, electrical stimulation induces much stronger feelings with much less electrical current in the lower regions of the brain. Thus, stimulation of the amygdala produces less intense emotional feelings in humans and other animals than stimulation of brain-stem areas such as the PAG, which lies at the center of the midbrain (one of the most ancient regions of the brain). Also, as already noted, when the neocortex is missing or removed early in development, both humans and animals grow up to be outwardly more emotional creatures than those that have higher regions of the brain to inhibit primary-process emotionality. It is easier to evoke emotional displays in animals without a neocortex (especially frontal regions) than with an intact brain.

These facts come as nothing less than a blessing for our scientific understanding of emotional affects. The tight relationship between the neural circuits that generate raw affects and the display of instinctual emotional expressions allows us to study something that we cannot see directly (affects) using proxies that we can see (emotional behaviors). Why has this insight been missing from the neural and psychological sciences? Perhaps because we are so accustomed to seeing motor processes as "mere outputs" as opposed to also being integrative processes for the organism as a whole. Unless animals had sophisticated action-schema in their brains, such as basic emotions, they would simply not have any chance to survive. The fact that such complex "motoric" brain functions can constitute emotional feelings seems compelling when one begins to think about the nature of life on earth, and the data now impressively demonstrate the concordance of emotional action and emotional feeling systems within the brain.

This allows a host of testable predictions based on animal brain research, especially research concerning neurochemical factors that can be applied in similar ways to studies of animal affects and human experiences. The

knowledge being gathered will be critically important for the sciences of biological psychiatry and psychotherapy. Obviously, emotional affects have powerful implications for mental health and illness. Regulation of RAGE, developing the capacity to counteract PANIC/GRIEF (by forming warm social attachments), negotiating FEAR adaptively, enjoying a capacity for PLAY, fulfilling one's LUSTful strivings gracefully, and approaching life with optimistic anticipation, compassion, and forgiveness are essential elements for good mental health.

It does not take all that much for emotional systems to go awry—which is why affective dysregulation is, and probably always has been, a common human experience. Only recently have psychologists become intensely interested in positive emotions (for the fullest recent summary, see Sheldon et al., 2011), and even neuroscientists and psychiatrists have started to scratch the surface of positive emotions (Burgdorf & Panksepp, 2006; Vaillant, 2008) beyond just studies of the brain's "reward system" (which is a misnomer, as we have already seen, but which we will further elaborate on in the next chapter). Until the neural nature of primary-process affects is clarified, psychiatry and psychotherapy will remain without a rigorous and transparent scientific foundation. There is still no generally accepted strategy for addressing this dilemma. But the affective neuroscience approach of cross-species triangulation (among behavioral, neural, and psychological lines of evidence) presents an established track record of bringing to light, from the depths of our brains, the sources of our most basic emotional feelings.

The mechanisms of human emotional feelings no longer need to remain a mystery. If we recruit the insights garnered from animal models in our efforts to understand the nature of human emotional affects, perhaps we can begin to fill the empirical gaps *that currently remain quite large*. Until now, mental health professionals have relied on disparate theories, none of which is complete or completely valid. Psychiatry relies on diagnostic categories that have little to do with brain science or our understanding of the emotional brain; they are derived instead from descriptions of outward cognitive signs and symptoms, reported verbally for the most part. Psychiatric medications have been discovered largely by chance—when side effects of medications for other ailments were unexpectedly found to produce beneficial emotional changes. Hardly any new *type* of psychiatric medicine has been discovered in the past 40 years. With clearer

neuroscientific visions of affective brains, new medical discoveries should follow more rapidly (see Burgdorf et al., 2011).

We believe major strides in our empirical and theoretical understanding will be made once we begin to take primary-process emotional action systems seriously as predictably organized affective entities within all mammalian brains. We may then develop new drugs and new therapies on the basis of a unified theoretical framework, rather than piecemeal and by chance. In other words, we can use preclinical (animal) models for psychiatric disorders where we manipulate distinct affective systems of the brain, and monitor how other affective systems are modified (for modelling depression, see Panksepp & Watt, 2011).

In short, gaining a comprehensive understanding of the brain mechanisms underlying the emotional affects seems like an essential project for contemporary psychiatry. Such knowledge can also provide a more solid grounding for the art of psychotherapy. In Chapter 12 Panksepp will explore examples of these novel ideas for psychotherapeutic practice, suggested by our emerging understanding of the neural foundations of emotions and emotional memories. Some findings have been totally unexpected. The discovery of *reconsolidation* (Chapter 6) indicates that we can take old and troublesome memories and then recast them with a less affectively disturbing penumbra.

The neuroscientific study of primary-process affective processes of the mammalian brain can open up the Pandora's Box of phenomenal consciousness—namely, how raw emotional experiences are actually constructed within the brain. It can concurrently do this for humans and many other animals. And the more we know about these processes in other animals, the better we will understand ourselves.

CHAPTER 3

The SEEKING System

Brain Sources of Eager Anticipation, Desire, Euphoria, and the Quest for Everything

Though animals learn many parts of their knowledge from observation, there are also many parts of it, which they derive from the original hand of nature. . . . These we denominate Instinct, and are so apt to admire as something very extraordinary . . . on which the whole conduct of life depends . . . which teaches a man to avoid the fire; as much as that, which teaches a bird, with such exactness, the art of incubation, and the whole economy and order of its nursery.

—David Hume, *An Enquiry Concerning Human Understanding* (1748/1910)

ONE OF THE MOST IMPORTANT instinctual-emotional systems of the brain is the one that allows animals to search for, find, and acquire all of the resources that are needed for survival. Arousal of this SEEKING system produces all kinds of approach behaviors, but it also feels good in a special way. It is not the kind of pleasure that we experience while eating a fine meal, or the satisfaction we feel afterwards. Rather it provides the kind of excited, euphoric anticipation that occurs when we look forward to eating that meal. Haven't you welcomed pangs of hunger when a delicious aroma from the kitchen reaches your nose? A period of separation from one's beloved can likewise hold a special charm, before the joy of reunion. The anticipation of sex is often more arousing than the excitement of

consummation. Even the anticipation of a hot bath may be an exquisite imagined delight, especially when one is enduring the chill of cold weather. And then there is gambling, the thrill of exploration, not to mention many aesthetic delights. This positive feeling (euphoria?) of anticipatory eagerness, this SEEKING urge, is entirely different from the pleasurable release of consummation. And this feeling exists as an emotion within certain subcortical networks of the mammalian brain long before the brain develops exuberant object-relations with the world (such as those described above). Initially, it is just a goad without a goal.

As noted in the previous chapter, the SEEKING system has traditionally been called "the Brain Reward System" because Jim Olds and Peter Milner (1954) discovered that rats would overexcitedly self-stimulate this system until they were exhausted—rats compulsively applied little electrical jolts into this brain region as if there was nothing more important in the world. Figure 2.4 shows an early depiction of this system, anatomically called the medial forebrain bundle (MFB), which courses through the lateral hypothalamus, connecting many regions of the lower brain stem and midbrain to many higher regions of the brain, all the way to the medial frontal cortex. This massive system sends connections to many other brain areas, thus, if this system is damaged on both sides of the brain, animals can no longer take care of themselves. They seem extremely depressed (perhaps the first animal model of depression without investigators recognizing that fact); such animals commonly die without intensive nursing care.

Behavioral neuroscientists are not accustomed to giving this essential network for survival a name like SEEKING, for it implies a level of intentionality in animals, but that is because they have not thought much about the likelihood that the primary-process emotional powers do have a simple mind of their own—a primal mind that makes animals into active agents in their natural environments. These ancestral brain systems automatically mediate "intentions-in-action," which may be essential antecedents for eventual "intentions-to-act" in human beings. The behavioristically oriented psychological tradition has called it the appetitive "Approach Motivation System" and even personality tests have been designed to measure this urge as well as its generic opposite —"withdrawal," or the "Avoidance Motivational System" (Elliot, 2008).

The great personality theorist Hans Eysenck in England first conceptualized these dimensions in a personality test for extraversion and

introversion/neuroticism. His student Jeffrey Gray, with rather more neuroscientific panache, developed his own personality tests for the Behavioral Activation System and the Behavioral Inhibition System (for an overview, see Larsen & Augustine, 2008). Other tests soon followed, with the Positive Affect and Negative Affect Scales (the famous PANAS; Watson et al., 1988). It is wondrous to see scientists say basically the same thing with different words, with terminologies designed to focus on just two facets of a multifaceted process. With the recognition that none of these tests evaluate the basic emotional temperaments, Panksepp and colleagues proceeded to develop the Affective Neuroscience Personality Scales (Davis et al., 2003), in which the statistically distinct SEEKING, CARE, and PLAY scales load together onto a positive-affect super-factor, and FEAR, ANGER, and the GRIEF/SAD scales load onto a negative-affect super-factor.

Thus, this system has been implicated in (i) general behavioral activation; (ii) a "wanting" state that controls "incentive salience" (Berridge & Robinson, 1998); (iii) the "persistence" of behavior (Salamone et al., 2009); (iv) the shifting between behavioral sets (Oades, 1985; Redgrave et al., 1999); (v) simple approach behavior (Ikemoto, 2010); and (vi) perhaps most arcanely "reward prediction error" by those who are enchanted mainly by learning theory (Schultz & Dickinson, 2000; Schultz, 2010), as we will discuss extensively later. Unfortunately those terms do not inform us of the many diverse appetitive behaviors the SEEKING system helps promote, and they do not tell anything about the specific positive affective characteristic this system promotes—anticipatory euphoria—as opposed to any "pleasure" of consumption.

We believe the SEEKING label is currently the best overall name for this primary-process system. This system has been found to participate in an enormous number of behaviors in rats, and some findings have been extended to humans (Knutson & Cooper, 2005). However, many of the examples we use here have not been actually studied by neuroscientists so they are heuristic hypotheses to make our theoretical perspectives crystal clear. We predict that when all the kinds of behaviors we describe have been studied, we will have confirmation after confirmation of the SEEKING system's role in every positive appetitive behavior in which we indulge.

From an affective perspective, one persistent dilemma in the field is that so many scientists interested in such problems (e.g., perhaps most

prominently Damasio, 1994, 1999) seem to believe that all types of good feelings are mediated by our senses. Perhaps they have overlooked that our ancient within-brain instinctual emotional action systems also can elaborate affective qualities of the mind. That important message seems to be missing from most scientific analyses of affective feelings. In any case, the evidence indicates that the emotional action systems generate feelings that can be triggered completely inside the brain, although each has certain sensory trigger spots (for instance, pain arouses FEAR). After learning, these systems typically come to be aroused by many other events.

Although there are many sensory inputs into the brain regions that sustain self-stimulation rewards, we need to consider that each animal's basic emotions, and hence their core-selves (see Panksepp, 1998b), are laid out in motor coordinates. This possibility does not preclude that the experiences of eagerness and euphoria that accompany SEEKING arousal can integrate various sensory feedbacks from the body and the external world; it merely suggests that organismic coherence is anchored to the primal action apparatus—the intrinsic "intentions-in-action"—that lies at the heart of our core-SELF structures in the brain stem (see Chapter 11). In any event, this SEEKING system helps motivate practically every energized thing we do.

THE MANY MANIFESTATIONS OF SEEKING IN THE MODERN WORLD

When the SEEKING system is aroused, animals exhibit an intense, enthused curiosity about the world. Rats, for example, will move about with a sense of purpose, sniffing vigorously and pausing to investigate interesting nooks and crannies. Rats often make little excited sounds that we can't hear without special equipment: ultrasonic 50-kHz chirps that are especially persistent when they are having fun (see Chapter 10 on PLAY). These are the same behaviors that rats exhibit when they are looking for rewards, rather than when they are consuming treats. Human beings report a sense of eager anticipation and an enhanced sense of themselves as effective agents who can make things happen in the world. People and animals clearly like this feeling, although it too can become excessive. They will work relentlessly until they are utterly exhausted (sometimes to the point of death, in the case of laboratory rats that are allowed to eat only one meal a day just at the same time when they are also allowed to self-activate

the brain “euphoria” system). Animals will expend much effort in order to achieve electrical or chemical stimulation of this circuitry (Ikemoto, 2010). We have named this crucial motivational system the SEEKING-EXPECTANCY system, or the SEEKING system for short. This designation makes more sense of the overall function of this system than the classic “reward system” concept. There are many affective reward processes in the brain.

Behavioral scientists have traditionally made the distinction we have already made here, between consummatory and appetitive behaviors. Before animals are able to consume rewards, they must proceed through the appetitive phase: they must search for, find, and take possession of the resources they need. And this does not apply only to seeking consummatory resources. The SEEKING system is probably involved in the appetitive phases of all the other emotional systems, although most of the following have not been studied neuroscientifically. For example, when a child eagerly puts on her bathing suit before going out to play in the pool with her friends, her SEEKING system may help energize her preparations. When we plot revenge against those who have irritated us, it is surely the SEEKING system that prompts us to devise these plans. And thus, some bullies eagerly ache for a fight. When hopeful lovers select the perfect restaurants for big dates, their SEEKING system may be paving the way for a romantic encounter. When you bake a cake for people you care about, the SEEKING system helps anticipate their surprise and delight. When you are scared, you have to seek safety. There are many, many cognitive differences in such experiences, but the anticipatory urgency in all of these activities shares a common positive want-to-do, and can-do feeling. Likewise, on the negative side, when the SEEKING system is chronically underactive, we experience a hopeless form of depression, characterized by lethargy and an absence of get-up-and-go.

SEEKING arousal also keeps us going when the chips are down—when we are hungry, thirsty, cold, or lonely. Perhaps we even feel a bit better because of it. This is because the SEEKING system provides positive, enthused affect that can counteract such negative feelings, at least to a point—a state we commonly call despair. Suppose that an animal is hungry. Hunger feels bad, but the encouraging sense of purpose that emanates from SEEKING arousal still makes the animal curious about its environment and sufficiently optimistic to engage in a focused and energetic search for food.

In other words, the "pleasurable" anticipation of finding food and the positive feeling of being able to do so provide a hopeful sense of expectation that will offset the negative feelings of hunger and, with luck, eventually remove them. However, when every plan fails, eventually despair sets in, and this is the gateway to depression.

All unpleasant states of homeostatic imbalance automatically make the SEEKING system more responsive to rewards (and the cues that predict them). Specialized nerve cells known as interoceptors (or "need detectors"), found in ancient medial regions of the brain and also in some other bodily organs, gauge homeostatic imbalances that lead to thirst and other affective indicators of bodily needs. For example, specific kinds of interoceptors respond when blood water concentration has diminished—whether because of cellular dehydration or reductions in blood volume, and thereby feelings of thirst are aroused. Others jump into action when sugar and body fat levels drop, promoting feelings of hunger. Other systems promote sleepiness, and in the midst of sleep we have dreams that are energized by dopamine-driven SEEKING urges. Do animals also have SEEKING dreams? Hummingbirds must eat abundantly each day, or they will die; evolution has taught them to have mini-hibernations each night to conserve critically needed energy for their morning search for nectar. But we don't know if they have dreams that are energized by hopes and fears as ours are, and we have no way of finding out.

Some internal sensors gauge shifts in sex hormones, which can promote LUSTful feelings. Still other sensors monitor core body temperatures. Although we do not yet know the exact mechanisms involved for all, neuroscientists have made great progress and are beginning to learn how neuropeptides convey such specific homeostatic messages to the SEEKING system, promoting behavioral activation. SEEKING arousal then inspires animals to enthusiastically search for the many types of resources that they need. When animals are hungry, thirsty, or cold, especially when there are indications of available resources in the environment, their SEEKING systems go into overdrive as they forage for food, water, and shelter. Likewise, when they have social needs, they may seek mates or, if very young, their mothers.

In addition to responding to homeostatic imbalances, the SEEKING system is also aroused when animals experience negative affects in relation to more complex social needs. These social needs are not monitored by the

kinds of interoceptors that gauge simple homeostatic needs. Nevertheless, as we shall learn in later chapters, unfulfilled social needs, such as the need for companionship or the need to play, cause affective distress. We do not know the precise mechanism by which unpleasant affects arouse the SEEKING system, but research suggests that many neuropeptides are again involved. For example, feelings of psychological pain and loneliness are promoted by high brain levels of stress-promoting corticotropin-releasing factor (CRF) and a dearth of endorphins, which are the endogenous soothing opioid neuropeptides that the brain itself manufactures. When people (and animals) have abundant levels of endogenous opioids in their brains, they experience positive affect and comfort, very much the kind of feeling one has in the company of good friends and lovers. When these chemicals are low, and CRF is running high, people and animals feel lonely, distressed, and often miserable. These painful affects are relieved when they find companionship, partly because of the release of endogenous opioids, but also partly because of elevated oxytocin and prolactin activity within their brains and many yet undetermined molecules. One additional molecule that has recently been identified to promote SEEKING functions is insulin-like growth factor 1 (IGF-1; Burgdorf et al., 2010).

It may be that a dearth of endogenous opioids alone arouses the SEEKING system, which then urges people and animals to find the social companionship that makes them feel better; but good evidence on such issues currently remains scarce. We also know, seemingly paradoxically, that we can intensify SEEKING activities with tiny doses of opioids placed directly adjacent to the dopamine cells and these doses can energize desire and whet appetites. Perhaps low doses of opioids actually promote SEEKING by inhibiting nearby GABA neurons that normally inhibit the SEEKING urge (Ikemoto, 2010). There are other options. The SEEKING system also participates in alleviating other negative emotions, such as FEAR (Salamone, 1994; Blackburn et al., 1992). When people and animals are in danger, their SEEKING systems prompt them to find safe refuge.

The SEEKING system responds to greed as well as to need. It is initially exquisitely sensitive to any and all rewards that are within one's grasp (Schultz et al., 1993). When someone is very hungry, even a dry crust of bread can be a delight as many prisoners discovered in the gulags and concentration camps of our sometimes extremely cruel social world. But even when bodily needs are satisfied, animals and humans are drawn to

enticing stimuli. For example, if a monkey has just eaten its fill, it still becomes excited if it spots a treat—a banana or some other favorite food. However, when we are hungry, we are enticed even more by treats. We mammals are equally susceptible to all kinds of temptations. Who can resist that extra piece of cake or some other favored food? And when it comes to drugs like alcohol, cocaine, and heroin, it is the SEEKING system that solidifies our addictive desires. And animals become addicted to exactly the same drugs that humans do. Some researchers believe that this happens without the animals having feelings about it. In fact, as we have developed ways to monitor animal feelings, for instance, through their emotional vocalizations, we find that those sounds can spontaneously indicate how animals feel, highlighting the underlying affective nature of addictive urges (Browning et al., 2011; Burgdorf et al., 2001; Panksepp, Burgdorf et al., 2002; Panksepp, Knutson et al., 2002).

Among animals in the wild, it is easy to see the SEEKING system in action. Resources are not readily available and animals must persistently seek them out in order to survive. They must hunt or forage for food and search for water, find twigs or dig holes to fashion sheltering nests. The SEEKING system urges them to nurture their young, to search for a sexual partner, and, when animals live in social communities, to also find nonsexual companions, forming friendships and social alliances. However, the role of the SEEKING system is not as obvious in the comfortable settings of modern human life, so evident in developed countries. We do our "hunting" at a leisurely pace down the aisles of supermarkets. Water is not actively sought so long as it is available on tap. We have easy access to warm comfortable homes. We meet friends and find lovers at arranged gatherings.

But this system remains alert to enticing possibilities even when bodily needs are met. Thus, it is easy to understand how this system can engender various excessive activities in modern societies that offer so many temptations. We are prone to overeat, smoke when it is unwise, and drink to excess. Many of us are workaholics. Drug addiction is rife. We are overeager to check our emails, to gamble, and to indulge in ill-advised sexual dalliances. In short, our SEEKING systems can all too easily urge us to indulge in a wide range of activities without our stopping to carefully consider what we are doing.

Although this system vigorously responds to homeostatic needs, to emotional urges and to enticing temptations, it operates more or less continuously in the background, albeit at much lower levels when people and animals are not in any particular need of resources or troubled by problems that urgently require solutions. This system keeps animals constantly exploring their environments so that they can remember where resources are. In that way they will be prepared to act when they are in need of food, water, company, or safety. The SEEKING system is in more or less continual operation for people as well. We regularly scan our environments, look in storefront windows, flip through magazines and catalogues, and surf the Internet and answer emails. We are always on the lookout for something that we might need or want, or something that might simply interest us and satisfy our curiosity. Our SEEKING systems keep us in a general state of engagement with the world.

In animals that are not as intellectually bright as we are, the SEEKING system operates without the admixture of forethought and strategic planning that is so characteristic of humans. In humans, strategic thinking plays a major role in SEEKING arousal because this system, like all our emotional systems, has abundant connections to the frontal neocortex, the most highly developed part of the cognitive MindBrain. When the SEEKING system arouses the human neocortex, it energizes thinking processes—a kind of virtual world—yielding complex learned behaviors that are not instinctual and may even be counterinstinctual.

Consider firefighters in the midst of battling a blaze. The situation is dangerous and they will feel a measure of fear that will automatically arouse their SEEKING systems. Under ordinary conditions, through this co-activation of the FEAR system, SEEKING arousal would prompt firefighters to find a means of escape. However, because they have been trained to help others and to put out blazes, SEEKING arousal will energize these learned skills, through activation of neocortical thinking and planning abilities. We have noted that when animals are hungry, their SEEKING systems create an urge to enthusiastically search for food. But when the firefighter's SEEKING system is aroused, it helps to counteract her fear and allows her to perform her job with focused vigor. All her training, experience, and ingenuity—all her cognitive and physical powers—will be bent on finding ways to put out the fire and help people to escape.

In addition to promoting the kind of practical strategic thinking in which the firefighter engages, the SEEKING system also arouses purely intellectual capacities of the neocortex. For example, you probably bought this book because you were intellectually curious to learn about the ways that the brain creates affective experience. We have already established that the neocortex does not provide its own motivation; the neocortex is activated by subcortical emotional systems. It is your subcortical SEEKING system that helps energize your neocortex—your intellect—and prompts you to do things like buy this book and also to learn from books, if they are engaging. Similarly, the SEEKING systems of architects, writers, artists, politicians, and scientists urge them to discover new and better ways to solve problems and to express themselves. This system energizes all human creativity—it has been a mental engine for all civilizations.

This is hardly a minor point. It highlights the fact that, in many ways, the neocortex—the source of our human intellect—is the servant of our emotional systems. The SEEKING system impels the neocortex to find ways of meeting our needs and desires: to cultivate farms, breed animals, build comfortable shelters, and weave protective garments. The SEEKING system urges the neocortex to do things that make us feel important and in command of our destinies; we try to manipulate social ties in ways that make us more influential or powerful. We build monuments to ourselves and to our gods and we express ourselves through artistic endeavors. The SEEKING system prompts us to satisfy our liking for novelty. We engage in scientific research that reveals nature's secrets. The SEEKING system also urges the neocortex to devise ways to gratify each and every one of our desires. We don't just farm and milk cows; we also make chocolate. Our clothes are not just for protection but for beauty and sexual allure. Mankind's great and unique achievements, the products of our prodigious neocortices, are firmly rooted in the psychic energy provided by this system.

It is evident that the SEEKING-EXPECTANCY system is a general-purpose system for obtaining all kinds of resources that exist in the world, from nuts to knowledge, so to speak. In short, it participates in all appetitive behaviors that precede consummation; it generates the urge to search for any and all of the "fruits" of the environment; it energizes the dynamic eagerness for positive experiences from tasty food to sexual possibilities to political power; it galvanizes people and animals to overcome dangers

either by opposing them or by escaping to safety; it invigorates humans and prompts us to engage in the grand task of creating civilizations. But in the beginning, at birth, it is just "a goad without a goal" (Panksepp, 1971) that opens up the gateways to engagement with the world, and hence knowledge.

The SEEKING system is driven by brain dopamine, but it is much more than just the creation of that one energizing neurotransmitter. It is a complex knowledge- and belief-generating machine. No wonder this system is still called "the brain reward system." In fact, this is the ancient brain system that allows us and all the other animals to gather all the rewards of the world. This is probably the system that almost brought the world to a second major financial depression in a century, the economic crash of 2008—with selfish greed outstripping broader human and societal concerns. Apparently this system needs to be trained well in order to reduce human tragedies. It has no intrinsic morals. It is just a super-efficient get-up-and-go-get-it system. Human cognitive aspirations, both for good and evil, spring forth from its vast affective "energy."

THE ANATOMY OF THE SEEKING SYSTEM

Anatomically, the trajectory of the SEEKING system runs from the ventral tegmental area (VTA) up to three main destinations: (i) the medial forebrain bundle and lateral hypothalamus (MFB-LH), (ii) up to the nucleus accumbens and (iii) to the medial prefrontal cortex via the mesolimbic and mesocortical dopamine pathways. A general summary of the anatomy is in Figure 3.1. Some of the major neurons of this system, the dopamine ones situated in the VTA, receive abundant inputs from other parts of the brain. As we mentioned, this system also has massive outputs to several higher regions of the brain, especially the nucleus accumbens, which is a major way station for appetitive learning. In certain "lower" mammals like rats, the ascending dopamine pathways that energize this system do not project beyond the frontal cortical regions. In humans, however, this system reaches much further, into the sensory-perceptual cortices concentrated in the back of the brain. This is consistent with the fact that SEEKING in humans arouses cognitive functions that do not have clear homologues in other animals.

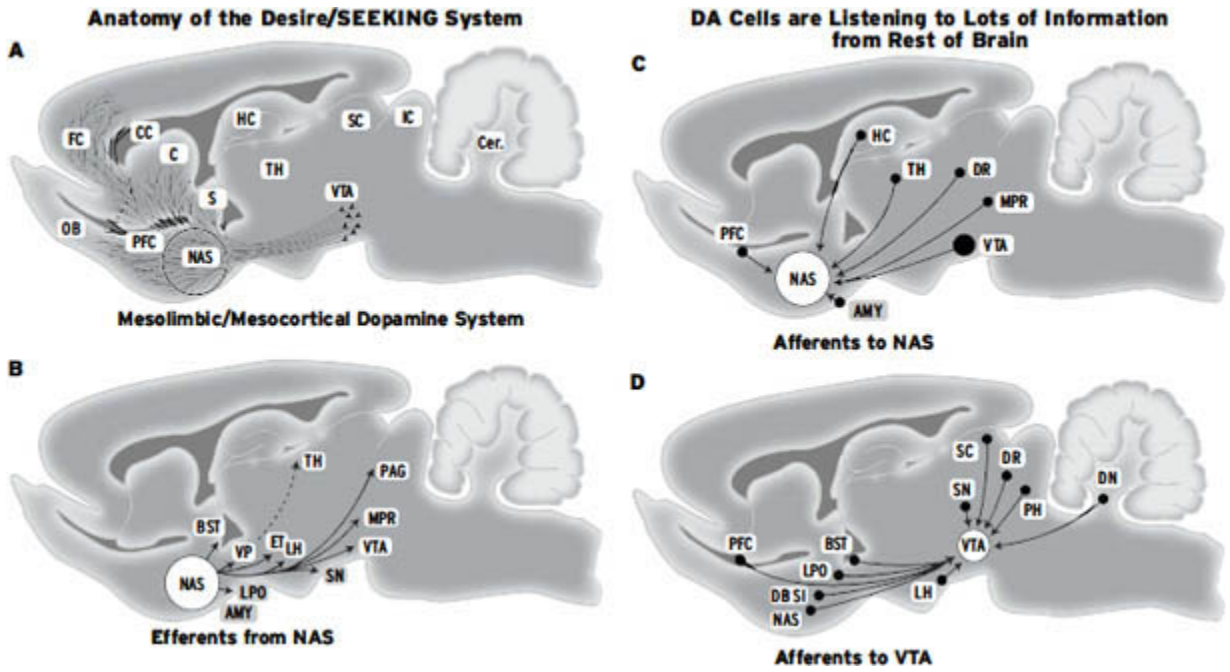

Figure 3.1. Schematic diagrams of the rat brain. **A.** Ascending projections of A10 DA (Dopamine) neurons localized in the VTA, innervating to limbic regions, including the NAS (nucleus accumbens septi), the mesolimbic DA system, as well as cortical regions via the mesocortical DA system. **B.** Major efferent projections from the NAS. **C.** Afferent projections to the NAS. **D.** Afferent projections to the VTA. Abbreviations—AMY, amygdala; BST, bed nucleus of stria terminalis; C, caudate–putamen; CC, corpus callosum; DB, diagonal band of Broca; DN, dentate nucleus; DR, dorsal raphe; ET, entopeduncular nucleus; FC, frontal cortex; HC, hippocampus; IC, inferior colliculus; LH, lateral hypothalamus; LPO, lateral preoptic area; MPR, mesopontine reticular nuclei; OB, olfactory bulb; PAG, periaqueductal gray; PFC, prefrontal cortex; PN, parabrachial nucleus; SC, superior colliculus; SI, substantia innominata; SN, substantia nigra; TH, thalamus; VP, ventral pallidum; VTA, ventral tegmental area (adapted from Ikemoto & Panksepp, 1999).

In all mammals, the nucleus accumbens interacts with the medial frontal cortex to promote simple appetitive learning (and addictions). Because the SEEKING system energizes the frontal neocortical regions, especially medial zones that focus on immediate emotional needs, we are able to devise strategies to obtain life's bounties and to escape its pitfalls. When

experiences are exceptionally pleasurable, we remember them, and this lays the foundations for the possibility of addiction. As already noted, the dopamine part of this system extends further throughout the cortex in humans than it does in most other animals. Of course, this system works in association with many other brain regions (Figure 3.1 B, C, D), including those that control general arousal (globally operating norepinephrine and serotonin systems) as well as more specific brain-attention functions such as those mediated by acetylcholine, GABA, and glutamate. Because the SEEKING system also participates in the enactment of all the other emotions we will discuss in this book, we will not repeat such complexities in each chapter, but we think that readers will appreciate that the discussion of each system is abstracted from the larger brain complexities in which each of those systems is embedded. No emotional system can do much without the help of the rest of the brain.

THE CHEMISTRY OF THE SEEKING SYSTEM

The SEEKING system is fueled heavily, perhaps mainly, by the neurotransmitter dopamine (DA). The role of DA in stimulating this system has been most thoroughly studied, but there are other key chemistries that enable this system to perform all the functions that it does. Neuroscience has amassed a huge wealth of molecular detail about dopamine functioning —enough to make the average reader's head spin. Drugs of abuse, like cocaine or amphetamines, are addictive because they directly enhance the effects of dopamine and thereby arouse the SEEKING urge. If overstimulated, animals' behaviors become stereotyped, and humans become intensely interested in very mundane things. For instance, women may engage in repeatedly reorganizing their handbags—taking things out and then putting them back in, seemingly endlessly, seemingly entranced. If this type of arousal is sustained for too long, individuals can become suspicious and most will develop paranoid tendencies. As we will see, over-activity of this system contributes to psychiatric disorders such as paranoid schizophrenia.

Other brain chemicals, most notably glutamate (Heidbreder et al., 1992; Yeomans et al., 1993), the major excitatory neurotransmitter of the brain, play a major role in the acquired functions (the learning) of the SEEKING system. To a large extent, appetitive learning occurs when the nucleus

accumbens integrates cognitive influences descending from the medial prefrontal cortex with emotional energies that ascend from lower regions of the SEEKING system (Kelley, 1999, 2004). Glutamate is the main brain chemical that fuels the appetitive learning process, just as it fuels learning in all of the other emotional systems.

In addition to dopamine and glutamate, a variety of neuropeptides are also clear chemical participants in regulating the SEEKING system. For instance, the neuropeptide orexin enables homeostatic imbalances, along with other emotional systems (like the FEAR system), to arouse the SEEKING system. Animals are typically enthusiastic about obtaining neuropeptides like neurotensin that activate the SEEKING system, and they usually dislike chemicals like dynorphin that deactivate the system. This underscores the fact that people and animals like the feeling of SEEKING arousal and dislike the feeling of this system winding down too low. It is now clear that when this system crashes, and the aversive feelings produced by dynorphin begin to prevail, people will feel depressed. Investigators are currently developing new antidepressant drugs that might reduce the awful feelings of too much dynorphin along this pathway (Bruchas et al., 2010).

STIMULI THAT INHERENTLY AROUSE THE SEEKING SYSTEM

We noted earlier that only a very few stimuli inherently (unconditionally) arouse most emotional systems. A rat has an inherent fear of the smell of predators, of brightly illuminated open spaces, and so on. Other mammals have different inherent likes and dislikes. However, the SEEKING system is also briefly aroused by all novel events, which means that it is aroused for a short time by a large number of changes in the environment. When a stimulus ceases to be novel (when the animal becomes accustomed to it) the SEEKING system no longer responds. This phenomenon is known as "habituation." The system also inherently responds to unexpected rewarding stimuli, like the delivery of food (Schultz, 2006). And the system continues to respond repeatedly if rewards are delivered sporadically or every once in a while—that is, it develops a sustained anticipatory urge (or a chronic craving). In some animals this might include the smell of prey or the sight of red, ripe fruit.

This already large repertoire of stimuli expands with learning. Suppose that a baby is excited by a shiny mobile hanging over his crib. When the attractive sight moves, the pieces touch and make a tinkling noise. Perhaps on a hot summer afternoon, the baby is in his highchair in the kitchen, having just finished lunch. His mother fixes herself a glass of iced tea and when the baby hears the tinkling of ice in the glass, he becomes excited. Perhaps it sounds like the tinkling of the mobile. When the baby first saw the mobile, it was a novel stimulus that aroused its SEEKING system. Now being used to it, the baby's SEEKING system is somewhat habituated. Nonetheless, the tinkling sound can still arouse the baby's SEEKING system, albeit not as much as when it was new. Now, anything that reminds the baby of the mobile, like the sound of ice in the mother's glass, or perhaps even when the baby might imagine the sound, can arouse his SEEKING system. But we cannot really study such issues in humans. There are always alternatives for every observed behavior. For instance, maybe the sound of the ice arouses the system because it is a novel sound, not because it provokes some memory of the sound of a mobile.

In any event, a variety of such associations occur throughout life, leading to highly individualized patterns of arousal. Animal research can actually track the cascades of causes and effects, and human brain imaging can provide less refined evidence of similar processes. Thus, we have good reason to believe that obsessive gambling and sexual urges are exquisite provocateurs of the SEEKING urge—the nucleus accumbens lights up more and more as one gets ever more excited. It does seem that all desired excitements in life arouse this system. However, some paths lead to excesses, and others guide people to substantive life accomplishments. It is left for a well-educated neocortex to decide which life choices to pursue. But if the conditioning is strong enough, often the higher mind cannot resist the temptations that the lower mind wants to pursue.

SEEKING in Relation to Disappointment and Rage

The SEEKING system is calmed by consuming things that have been desired, but it will not be calm for long if the satisfaction does not last. When a hungry animal forages for food, its SEEKING system is aroused, but when it begins to eat, the SEEKING system becomes quiescent. Still,

the system can be promptly aroused by the possibility of a special treat. However, when the system is thwarted, perhaps by some other critter getting the treat, anger may flare. Consider the common frustration of placing coins in a vending machine that does not fulfill its part of the bargain. People will shake and sometimes kick the machine. In terms of neurophysiology, the SEEKING system has shut down without the benefit of consummation (with no treat) and this then arouses the RAGE system.

PATHOLOGIES OF THE SEEKING SYSTEM

A well-functioning SEEKING system is essential to physical and emotional health. However, when the system is under- or overstimulated it can promote emotional disorders, ranging from depression to psychosis. In his book *Awakenings* (1973), Oliver Sacks wrote about the crushing depression suffered by patients whose SEEKING systems were understimulated due to the depletions of dopamine caused by Parkinson's disease. The drug L-dopa redressed this chemical imbalance, for a time, with dramatic results. Sacks, quoting one of his patients, Leonard L., wrote, "I feel saved . . . resurrected, reborn. I feel a sense of health amounting to Grace. . . . I feel like a man in love. I have broken through the barriers which cut me off from love." Sadly, the abundance of dopamine eventually overstimulated the SEEKING systems of these patients, producing excessive cravings and desires and an unrealistic sense of destiny—in a phrase, psychotic symptoms. As we will see, in such frames of mind, one can begin to see delusional connections between events; animals exhibit similar types of misattributions.

As already noted, depressive feelings emerge when the SEEKING system is chronically underactive, for instance, following repeated frustrations or during withdrawal from addiction to amphetamines and cocaine. On the other hand, schizophrenia, mania, and psychotic delusions arise at the opposite end of the SEEKING spectrum, reflecting excessive psychological tendencies when the system is grossly overstimulated with dopamine (Grace, 1991). Drugs of abuse like amphetamines and cocaine are very effective stimulants of the SEEKING system because they increase the availability of dopamine in the synaptic clefts, the communication channels between neurons. Such drugs are easily abused, and they hypersensitize SEEKING urges, making people even more responsive to addictive drugs. Animals also become more responsive to other treats, from tasty foods to

sexual encounters (Nocjar & Panksepp, 2002). Psychiatrists are well aware that these kinds of drugs, taken for too long and in high doses, eventually cause psychotic symptoms—in anyone. Some succumb quickly; others deteriorate more slowly. But everyone who takes too many of these drugs will eventually tumble toward psychotic, paranoid thinking (Snyder, 1972). And then, during drug withdrawal, depression will rule.

We have mentioned that the SEEKING system is especially effective in arousing cognitive areas of the medial frontal cortex. One of the functions of the neocortex is its ability to generate concepts of cause and effect. When it is overstimulated, the frontal cortex, which elaborates "working memory" (see Chapter 6), will entertain abundant new thoughts about how the world is organized. It will often inspire someone to see causal and other meaningful links where there are only correlations or where there are no meaningful connections at all. When this happens, thinking runs wild, resulting in rampant and often erroneous conclusions. Now the mind is fertile ground for delusions to sprout. The enhanced sense of self, which is also typical of SEEKING arousal, can likewise take on unrealistic proportions, resulting in psychotic delusions of grandeur.

For example, a schizophrenic patient might harbor the delusional belief that his actions, like breaking a favorite mirror, caused an important world event—like the bombing of the World Trade Center towers on 9/11. This would constitute a delusional belief in cause and effect because the patient's personal actions had not caused something in the greater arena of the world. There is also an element of delusional grandeur in this because the patient believes that he has the power to cause these important events to occur. These sorts of psychotic fantasies are generated by a grossly overaroused SEEKING system. It is interesting to note that stress can elevate dopamine activity in the frontal cortex. This may explain how severe stress helps promote paranoid, schizophrenic thinking patterns. Indeed, some have envisioned a relationship between such modes of thought and dreams (Panksepp, 1998a; Solms, 2002), and recent work has confirmed that dopamine neurons in the SEEKING system are firing at very high rates during REM sleep (Dahan et al., 2007). Therefore, it is reasonable to conclude that abundant dopamine activity in the brain occurs both during dreams and in schizophrenia (Léna et al., 2005; Panksepp, 1998a; Solms, 2000).

Antipsychotic Medications

Dopamine is the main chemical that arouses the SEEKING system—although it is not the only one, it is certainly the one that we know the most about. Dopamine arouses this system by being released at synapses in a global way and by binding with molecules known as receptors on receiving neurons (and there are five major types of receptors, clustered into two families, namely D1 and D2, of which we will only consider one here: the D2 receptor, which is especially important in psychiatric disorders such as schizophrenia). Binding occurs in a key and keyhole fashion, where dopamine serves as the key and the receptor as the keyhole. In addition to dopamine, a host of other chemicals (i.e., neuropeptides and other neurotransmitters) can also serve as keys for their own specific receptors.

There are commonly a number of different receptors with which each brain transmitter chemical can bind—each chemical has more than one receptor that it can "talk" to. Receptors, on the other hand, are typically more exclusive; they can "listen" to and only bind with a particular transmitter chemical. A chemical key that fits into a receptor "key hole" but cannot open (or activate) it is called a receptor blocker. When a receptor is blocked, the chemical that normally binds with it cannot do so, and the activity of this brain chemical is thereby reduced. So if one administers dopamine blockers (many such drugs are antipsychotic medications), then the dopamine released at the synapses can no longer bind with receptors in the SEEKING system, and the system becomes underaroused, resulting in depressive symptoms, such as those described above.

Researchers have discovered that the excessive activity of dopamine at one of its receptors, namely the D2 variety, causes (or at least correlates with) some schizophrenic symptoms. Virtually all medications for schizophrenic symptoms, which are medications that quell delusions and hallucinations, will block dopamine activity at D2 receptors. If the patient who broke the mirror, as mentioned above, were put on an antipsychotic D2 blocker, the cognitive aspect of his delusions would not disappear completely, but the delusions' power to motivate would be markedly diminished. He might still think that he had something to do with the catastrophic events, but these thoughts would no longer have the same intensity of conviction. In other words, antipsychotic drugs usually reduce the strength of delusions but do not change their content. This is why

talking therapies are sometimes also useful in helping patients to reconfigure their delusional cognitions. In the case of this patient, if his delusion sprang from excessive anger, then it might be helpful to understand what made him so prone to rage in the first place. Antipsychotic drugs that block dopamine signals also quell an animal's tendency to investigate its environment and hence pick up new information. The tendency to investigate is a normal expression of the SEEKING system. Delusions lie at the pathological, far end of the SEEKING continuum.

Bizarre Cases of Ritualistic Adjunctive Behaviors

When the SEEKING system is less severely overstimulated, it generates adjunctive behaviors, which are compulsive but often serve no obvious outward purpose. Under laboratory conditions, one sees adjunctive behaviors, for instance, when very hungry animals periodically receive small amounts of food. The small bits of food they receive are not enough to satisfy them, and they have no means of procuring more by themselves. Since these animals are in a continuous state of hunger, their SEEKING systems are continuously hyperaroused. While they are waiting for the next small food delivery, these animals commonly engage in adjunctive behaviors. For example, a hungry laboratory rat might run excessively in a running wheel. Another rat may shred paper, gnaw on wood, or drink copious amounts of water. These behaviors are not related to their bodily needs and are therefore called adjunctive. One also sees adjunctive behaviors in everyday life. People who are very hungry tend to pace back and forth. Pacing is an adjunctive behavior that does nothing to nourish the body or to procure food. In fact, it may be counterproductive if it expends scarce energy.

Adjunctive behaviors are often repetitive and appear to be ritualistic. B. F. Skinner, one of the founders of behaviorism, noted that hungry pigeons would engage in a repetitive and predictable strutting, wing-flapping "dance" during long intervals between receiving small bites of food (Skinner, 1948). They did not perform their dance just after receiving a morsel, and they did not perform it during nontesting periods. Rather, the pigeons danced while they waited for the next bits of food, usually in a state of extreme hunger—a state that unconditionally arouses the SEEKING

system. We are not suggesting that pigeons cognitively "think" they can make food appear by dancing. Rather it seems that when the SEEKING system is over-stimulated, it automatically promotes repetitive and ritualistic behaviors. These adjunctive behaviors are markedly diminished by dopamine blockers as well as by lesions on the lateral hypothalamus, manipulations that deactivate or damage the SEEKING system (Wayner et al., 1981).

What is more difficult to understand is why an animal would engage in one sort of repetitive adjunctive behavior instead of another. For example, why would one hungry man pace the floor while another whistles and yet another slams his fist into his palm? In the study of animals, the type of behavior exhibited seemed to be a property of the specific animal being studied—that is, it was a property of its personality. So the man with the more aggressive personality might punch his fist, while a more compliant soul would whistle. Alternatively, adjunctive behaviors can seem more purposeful—they are directed toward stimuli that typically predict rewards. For instance, hungry rats that are periodically given small bits of food will begin to gnaw the food bin into which food is delivered, although this does not affect the rate of food delivery in the least. It almost seems as if certain sorts of behaviors give the animals a focused sense of purpose. In other words, ritualized adjunctive behaviors seem to be fashioned in a way that makes the animal feel that it is doing something productive, even if it isn't. In a similar way, people and animals on high doses of cocaine and amphetamine, both of which strongly stimulate the SEEKING system, show seemingly endless repetitive behaviors. As noted earlier, when humans have such strong stimulation, they often report that doing mundane things like exploring their handbags suddenly becomes very intriguing.

Probably there is an adaptive value to the proclivity to exhibit such repetitious and ritualistic behaviors. Learning a new skill requires repetition, sometimes to the point of its becoming a ritual. When a gymnast learns how to negotiate a double somersault, probably she will take exactly four steps, tuck her head in a characteristic way, and always leap off the same foot, and so on. We have many habits that involve repetition and ritual. We put our keys on the same hook every night, and we fold our clothes in particular ways. Even when we take a shower, we are apt to wash different body parts in a certain order. It appears that SEEKING arousal helps engender these sorts of habits. However, once a behavior has become

habitual, it is laid down in dopamine-controlled brain regions, such as the dorsal striatum just above the nucleus accumbens (e.g., the caudate nucleus) whose arousal is controlled by the nigrostriatal dopamine system just lateral to the VTA. Stimulation of those brain regions is much less rewarding, because habits are just habits. Many are done unconsciously. Under such conditions, one no longer needs to become emotionally aroused when formerly exciting behaviors established through the SEEKING urge have become routine. Thus, it should not be surprising that animals do not exhibit much self-stimulation behavior for activation of those more recent dopamine systems.

The Strange Case of "Autoshaping"— Correlations Are Not Causes, But . . .

Autoshaping refers to a laboratory phenomenon that gradually emerges when an animal is very hungry (which also means that its SEEKING system is highly aroused) and when the animal is also exposed to a short, extraneous stimulus, for instance, lighting up a key above the food tray, just before the delivery of bits of food (Brown & Jenkins, 1968). This predictive stimulus seems causally related to the animal getting its treat. A cool-headed philosopher might simply decide to patiently wait till each treat arrived, without getting all eager, full of anticipation, and thereby beginning to interact with the stimulus that predicts food.[1] Such behaviors can in no way help alleviate hunger; nevertheless, animals will gradually begin to interact with such stimuli, almost as if they believed that such interactions would procure the reward. After repeated exposure to a pairing like this, the animal, in this case a pigeon, begins to peck at the key that would become lit before the food delivery. Pigeons will persist at this activity long after the experimenter stops delivering food, even though the pecking accomplishes nothing. Autoshaping has now been observed in all mammalian species that have been studied. It is clearly a SEEKING behavior because dopamine blocks the effects of autoshaping (Phillips et al., 1981). This has been a bit of a challenge for those who think animals will behave sensibly rather than emotionally.

To our intelligent minds, which think in terms of cause and effect, it appears that the autoshaped pigeon has made a useful but delusional mental connection between pecking at the key and food delivery. Perhaps it has.

However, most behavioral investigators doubt that pigeons are clever enough to make such mental leaps. Then what accounts for the behavior? It may simply be a matter of blind learning. Perhaps the pigeon does not "think" that pecking the key will ensure food delivery, any more than Skinner's dancing pigeons "thought" that their dance would procure food. Of course, we will never know because we have no way to access the thoughts of other mammals, not to mention birds (but see Clayton et al., 2003), at least not as clearly as we can gauge their emotions. How, then, are we to understand this behavior? Why does the pigeon peck at the key? Well, maybe it has become conditioned to be hyperemotional, and generating superstitious behaviors is as good a way to spend its time as any other in a very boring environment, especially when the experimenter is tempting it with tidbits of food every once in a while, treats that are rather consistently predicted by a cue.

When the SEEKING system is aroused, animals become curious about their environments. It appears that when the hungry pigeon sees the illuminated key, its curiosity is aroused, and it explores the key by pecking at it. In other words, SEEKING arousal causes people and animals to take notice of and examine any stimuli that might help them make sense of the world. Animals do not need to "think" that there may be a causal connection between the extraneous stimuli and food delivery. Conditioned SEEKING arousal ensures that they will be curious about the environment in patterned ways. This curiosity is adaptive because sometimes such extraneous stimuli are cues for resources. Indeed, in the world, such "insights" might work as often as not. For example, if a hungry pigeon in the park happened to notice and investigate some shiny paper on the ground, it might find the tasty remnants of potato chips. Thereafter, any shiny paper would serve as a cue that may predict food, and the sight of it will arouse the pigeon's SEEKING system into a focused approach and interaction with such a stimulus.

Autoshaping and adjunctive behaviors take place separately under strict laboratory conditions. In real life, however, autoshaping and adjunctive behaviors usually go hand in hand. Animals engage in repetitive adjunctive behaviors, often using an extraneous object—a conditioned stimulus—on which they perform the adjunctive behavior. For example, the pigeon in the autoshaping experiment pecked repetitively at the key and the pigeon in the park probably pecked at the potato chip packet in a repetitive way. Human

beings also exhibit combinations of adjunctive behaviors and autoshaping. Suppose that your manager has been bossy and unjust, arousing your RAGE system. You wanted to have it out with him, but he put you off until the following week and your anger had to remain in abeyance. After dinner that night you read the paper, hoping to distract yourself from your irritated preoccupation and you notice the crossword puzzle, something you usually ignore. Tonight, however, you try your hand and become unusually engrossed, staying up past your usual bedtime. While you are doing the puzzle, you feel better and may even enjoy the activity. However, once you put the paper away, you may again think about your boss and feel angry.

In neuroscientific terms, your RAGE system is aroused because your boss has given you a hard time. As far as we know, some SEEKING arousal initially accompanies all types of emotional arousal and in this case it may urge you to plan strategies about how to approach your boss. However, because your boss has not been accessible, your predicament is akin to that of the pigeon in the autoshaping experiment. The pigeon wants to satisfy its hunger by eating and you want to satisfy your RAGE by giving your boss a piece of your mind. Neither of you has the means of doing what you want to do. So your SEEKING system and the pigeon's SEEKING system are aroused without the possibility of useful activity. Under these conditions, you and the pigeon perform adjunctive behaviors toward extraneous stimuli. The pigeon pecks at the key and you work eagerly on the crossword puzzle. Perhaps this is a bit of a stretch, but hopefully the point is clear. It would be much more poignant and clinically relevant if we think of a spouse as opposed to an inanimate crossword puzzle. One might easily vent their anger on the wrong person. We like to have a feeling that we are controlling the world, even if we are not. Could this be one reason so many people pray? Or why they go astray in the way they vent their emotions on "innocent bystanders"?

Later in this chapter, when we discuss conditioned learning, we will again consider how autoshaping and adjunctive behaviors play important roles in providing the circumstances that are necessary for learning to occur. The tendency for autoshaping ensures that people and animals take notice of extraneous stimuli that seem to be causally related. This is a necessary prerequisite for conditioned learning. The tendency for adjunctive behaviors causes people and animals to learn how to perform efficient repetitive behaviors that typically also emerge when animals are conditioned. Both

autoshaping and adjunctive behaviors are manifestations of SEEKING arousal, and both may be foundational in the way conditioned learning occurs in the real world.

And there is an aspect of this that is also central to science—the role of *induction* in generating testable hypotheses. Inductive logic is little more than seeing relationships along correlated events along with the "insight" that such correlations imply causality. Of course, this leads to experiments where critical related variables are independently manipulated to see whether there are causal relationships that can be demonstrated. In this way the many potential flaws of seeing correlations as sources of causality, on which autoshaping is based, are avoided. Predictions and testability save science from the many false leads that inductive thinking can lead to—from observations that suggested the earth was the center of our universe to potentially the power of prayer to change physical events in the world. This critical mode of thinking salvaged science from the seemingly endless cycles of false beliefs, developing from uncritical acceptance of surface observations at face value, that have often characterized human thinking and hence cultures.

THE SEEKING SYSTEM AND FAITH

We have seen that SEEKING arousal can produce persistent ritualized behaviors like the pigeon's dance between predictable rewards or the autoshaped key pecking. The SEEKING system does not think about personal matters, but the neocortex does, especially the medial frontal cortex with which the system is connected. When people are ruminating, this is the brain region that usually lights up (Northoff et al., 2010). People have large neocortices and the neocortex has the capacity to interpret and make sense of events in terms of cause and effect. Imagine a tribal people who are suffering through a drought in an era of limited scientific knowledge. In their frustration, the people might engage in ritualistic and adjunctive emotional behaviors. They might persistently walk about, at times with a kick and shout, kicking up the dry ground in a manner that resembles the pigeon's dance. Eventually, rain would come. Noticing the correlation between their adjunctive stomping about and the advent of rain, they might come to believe that a causal relationship exists, which provides the motivation for creating a rain dance in the hopes of precipitating future

downpours. Thereafter, they might regularly use a dance ritual—a form of prayer—in a culturally condoned effort to produce rain.

Most of us in the modern Western world believe that this is a delusional way of thinking. But many of us have a tendency to pray during periods of distress. Some people pray without really believing that it will help. However, it seems to make them feel better because they are taking some sort of action. Because people are generally intelligent enough to know when they cannot control their fates, this action often takes the form of a verbal appeal to a higher power—God—who can control fate. It appears that adjunctive behaviors make people and animals feel better because they provide the illusion that they are effective agents—this too is a feature of SEEKING arousal. Some explicitly enlist God's power, for instance, to find a parking place when there is none in sight—and sometimes it "works"! Could this partly explain why praying is such a popular activity, especially during times of stress? Could praying be an adjunctive behavior that gives human beings the illusion that they are somehow able to magically change their fates?

One can also imagine how autoshaping might be involved in the creation of religious symbols. Suppose that the chief of a tribe whittled aimlessly on a piece of wood during a drought. When the rains came, someone noticed that the haphazard whittling resembled the face of a wolf. This piece of wood might attract the attention of the tribal elders, in much the same way that the lit disc attracted the attention of the pigeon. It would be a novel and significant object and their SEEKING systems would focus on it. Because their large neocortices are able to think in terms of cause and effect and to devise narratives, they might think that wolves had supernatural powers that brought on the rain. Then they might carve wolf faces in wood and use them as religious symbols to which they could pray in times of trouble. Of course, we are just imagining such situations here. However, if prayer can be seen as an adjunctive behavior and if autoshaping plays a role in the creation of religious symbols, the SEEKING system might explain a great deal about the neural roots of religious belief. In this connection, it is perhaps no accident that religiosity is a core feature of many psychotic illnesses.

But there are many other aspects of affective life, tendrils that go deep into religious traditions. In agreement with Thandeka (2009), we believe that one driving force behind human religions is our affective nature,

especially our desperate need for nurturance and understanding, to ward off grief through community, and often with the desire to seek a higher good. We will revisit this revolutionary theme again in the chapter on the PANIC/GRIEF system.

TWO GENERATIONS (AND COUNTING) OF MISUNDERSTANDINGS ABOUT THE SEEKING SYSTEM

As already related, the SEEKING system was first studied in 1953 by Olds and Milner at McGill University, in Canada, although they did not call the system by that name. While looking for other things (i.e., how artificially induced brain arousal/attention might facilitate learning), they stumbled upon the phenomenon that animals would work in order to receive tiny electrical jolts to specific parts of the brain. At times, Jim Olds (1922–1976) called this "the pleasure system." But other investigators were rather more prudish, at least until the 1980s, when the grand exploratory era of self-stimulation research had come to an end, and most investigators started to focus on the dopamine component of this complex system.

From then on, practically everyone was calling it "the brain reward" or even "the reinforcement system." But one must suspect that by the time he was writing his last book (Olds, 1977), Olds had realized that there was much more to this system than the creation of pleasure. He had started to study the classical conditioning of appetitive drives, by pairing a tone with delivery of food to hungry rats, and monitoring the neuronal activities throughout the brain. He discovered that many, many places in the brain learned to anticipate the forthcoming food, but the fastest neuronal conditioning, and the earliest signals indicating that the animal was anticipating the food were coming from neurons along the MFB-LH corridor of what we here call the SEEKING system. The firing of cells typically predicted the forthcoming reward, but Olds never seemed to make up his mind that what he had actually discovered was the brain system that eagerly anticipates rewards rather than just registering the pleasure from consuming the rewards. A few years before Olds's untimely passing, Panksepp had discussed the EXPECTANCY/SEEKING hypothesis with him, on a flight to Europe, and he had been intrigued, noting how his electrophysiology work was consistent with that idea.

The discovery of "brain reward" by Olds and Milner was surely one of the greatest neuroscientific discoveries of the twentieth century, leading eventually to studies that have revealed the neural underpinnings of learning and addiction. Animals would learn all kinds of things with MFB-LH stimulation, from pressing levers to running down particular paths in a maze, which are formally called operant and instrumental conditioning. They called the effect self-stimulation, because the animals played an active role (they worked) in order to receive the electrical jolts of "joy"—one might even suggest they discovered a form of mental masturbation. Animals tickled their brain regions that were evolutionarily designed for getting other goodies. After all, what a person masturbating really wants is an erotic relationship but, for various reasons, ends up taking care of his or her satisfactions alone, closely resembling an addiction (Zellner et al., 2011). And now we know that this general-purpose SEEKING system is critically important for all kinds of addictions from drugs such as cocaine and morphine, to dependence on alcohol and nicotine, and even sex (Wise & Rompre, 1989; Robinson & Berridge, 1993). The system also is a force behind all kinds of creative activities (Reuter et al., 2005).

One big problem remains. Most young investigators who have "inherited" the study of this fascinating brain system hardly question the unitary concepts such as "reward" and "reinforcement" that were handed down to them by previous generations, as if they were unitary phenomena. Indeed, the concept of "reinforcement" may just be a summary term for our ignorance—perhaps the "phlogiston" of behavioral science[2]—that was simply a convenient *procedure* for training animals. However, as a brain *process*, this did little more than to cover (and hide) a mountain of ignorance. Along the way, most investigators of this "brain reward system" have failed to consider the actual "natural" behavior patterns, characteristic exploratory activities, that animals spontaneously exhibit when the SEEKING system is artificially aroused.

Animals will self-stimulate many areas of the brain, the main ones are the septum and the LH, through which courses the MFB that contains the ascending dopamine systems, but also many other neural networks. We already know that self-stimulation of the MFB-LH and of the septal area are experienced quite differently by animals, although sites along the MFB-LH tend to feel the same to animals since they have difficulty discriminating two distant sites along this pathway (Stutz et al., 1974). Because very little

discriminating work like that has been done, we must assume that many other brain sites that mediate self-stimulation also generate distinct types of rewards. In any case, the brain contains many reward systems.

Now there have been several generations of scientific "passing of the buck" about the kind of reward that is engendered by MFB-LH stimulation, and we probably don't have to repeat, that the global concept of the "brain reward system" is rather off the mark, even though people who know better, continue to use the term (Haber & Knutson, 2010). This description simply does not capture the natural behavior patterns that such brain stimulation evokes in animals, and the electrophysiology consistently indicates the system is designed to first get excited about newly found rewards, and then rapidly comes to anticipate them, if more is coming. The bottom line is that animals getting MFB-LH rewards simply do not behave as if they are consuming a delightful treat and experiencing a sensory-affective reward. Stimulation of the septal region produces behavior that is closer to that.

When animals self-stimulate the LH, they do so in a frenetic way. They frantically press levers, with noses sniffing "a mile a minute," almost as if they are trying to see what was behind the lever—to explore it—and they typically work much harder than necessary to get all the "rewarding" electrical jolts. In contrast, animals self-stimulate the septal area with a very different behavioral "attitude"–they work at a methodical pace, usually pressing the lever once for each shock, and without agitation. By various measures, it does not appear that stimulation of the septum is any less pleasurable than LH stimulation, and by human self-reports, septal stimulation actually does evoke feelings of pleasure (Heath, 1996). Why then do animals work so excessively hard to obtain LH stimulation? The most reasonable hypothesis is that the SEEKING system induces a robust mental and behavioral invigoration—the kind of arousal that animals display before they get forthcoming rewards, as they experience some kind of euphoric enthusiasm. Just think of a hungry dog bounding up and down, sometimes in circles, as you bring forth a food bowl.

However, in those early days following the discovery of self-stimulation, behavioral concepts ruled as the only meaningful scientific way to discuss animal behaviors. Even ethologists, who preferred to study the natural behaviors of animals in the wild, constrained their analyses to accurate descriptions of behavior with no hint of any mental constructs, and emotional issues were rarely discussed. The Nobel Prize winning ethologist

Niko Tinbergen noted that since "subjective phenomena cannot be observed objectively in animals, it is idle to claim or deny their existence" (1951, p. 5). There was a taboo against any talk about the subjective aspects of the brain (Wallace, 2000). Thus, it is no surprise that the animal mind was largely neglected by scientists, at least until Donald Griffin (1984, 2001) started to talk forcefully about the possibility of animal consciousness again—an exercise that was quite popular, perhaps rather too in vogue, during the late nineteenth century (e.g., Lindsay, 1880), especially by Darwin's protégé, George Romanes (1882).

When Olds and Milner discovered the phenomenon of self-stimulation, behaviorism was at its zenith. The biggest achievement of the behavioral movement was its discovery that animals can be coaxed to work in specific ways (they will display operant/instrumental behaviors) in highly predictable patterns, when rewards, usually in the form of food or drink, are delivered at particular times (i.e., according to various reward delivery procedures called "schedules of reinforcement"). For instance, under one schedule, when food is delivered after a fixed number of responses (fixed ratio schedules, sort of like chopping wood), animals press a lever as fast as they can, eat the reward, and relax for a bit before beginning another flurry of maximally fast operant behaviors. They work somewhat more slowly but at a steady pace when rewards are delivered unpredictably—after varied numbers of operant lever presses (variable ratio schedules). When rewards are delivered at regular intervals of time, regardless of the number of operant behaviors (fixed-interval schedules), animals press a lever slowly after receiving a reward and press increasingly quickly as the time approaches when the reward is about to be delivered again (it looks like a curve of increasing anticipation). Animals work comparatively slowly but rather steadily if rewards are delivered at various unpredictable times regardless of how often animals press the lever (variable interval schedules). If it is hard to visualize these verbal descriptions, see Figure 3.2 for "cumulative records" of responses when animals are working on these various schedules (Panksepp, 1998a, p. 22).

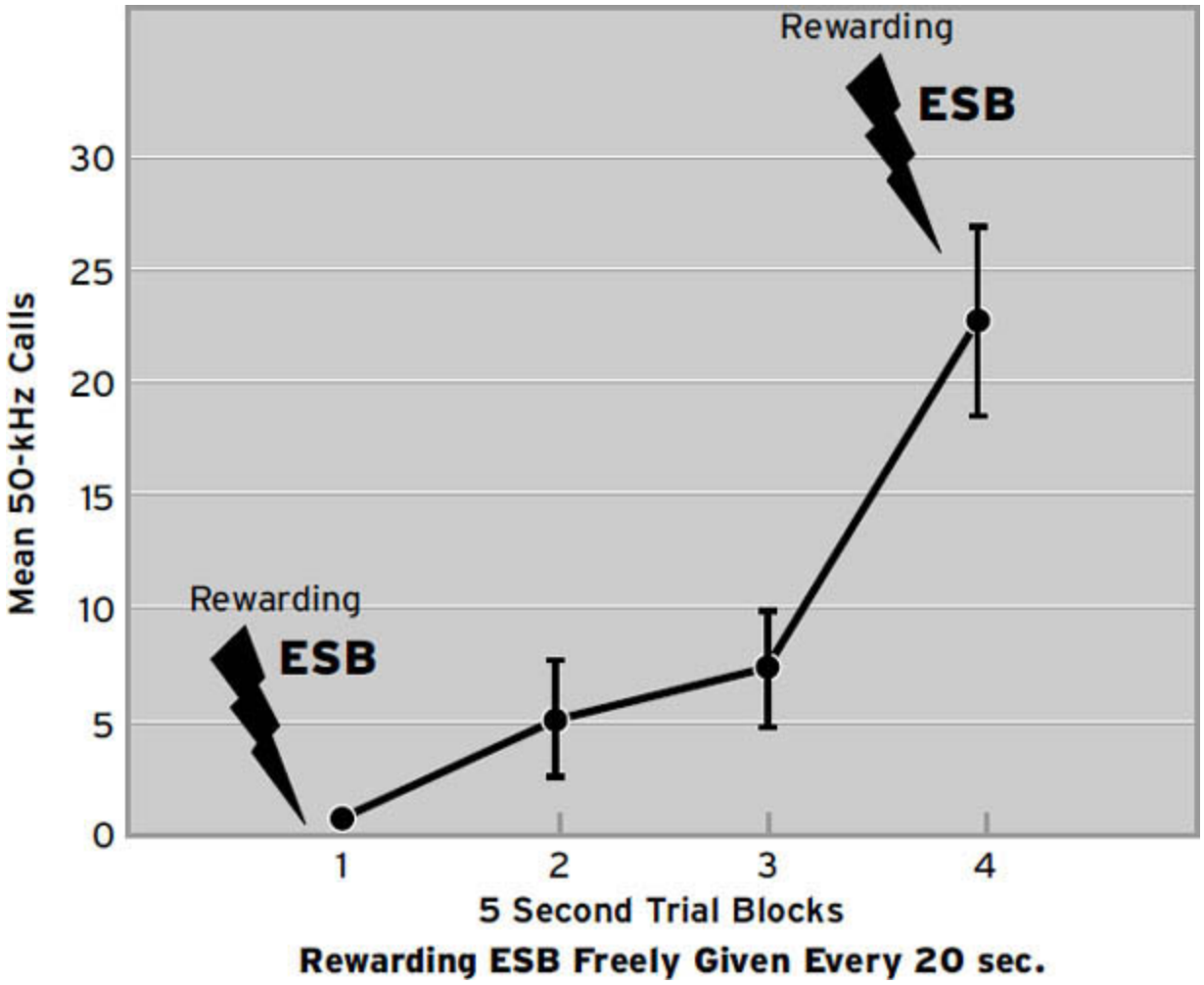

Figure 3.2. The spontaneous generation of positive-affect-indicative 50-kHz ultrasonic vocalizations in rats given a half second of free rewarding lateral hypothalamic electrical stimulation of the brain (ESB) at a fixed interval schedule of one stimulation given every 20 sec. After only modest exposure to this pattern of free brain rewards, animals begin to exhibit an anticipatory curve, which is characteristic of animals working for conventional rewards such as food on a fixed interval "schedule of reinforcement." Very similar patterns are spontaneously also obtained with measures of sniffing behavior, which reflects an instinctual exploratory response mediated by the underlying SEEKING system (data adapted from Burgdorf et al., 2000, Figure 1, p. 321).

Schedules of Reinforcement and the Strange Effects of "Brain Reward"

The use of schedules of reinforcement or reward, many more complex than the basic ones described above, led to highly characteristic and predictable behavior patterns in all animals that were tested, including human beings. This consistency gave behavioral scientists confidence that they were revealing laws about the ways that people and animals respond to rewards in simple learning situations. Of course, being radical behaviorists, they were not interested in the deeper neural nature of emotions and motivation. Instead, they considered only stimuli and responses, associated with rewards, punishments, and a process called "reinforcement" that supposedly glued them all together. The reward stimulus was usually food or water delivered at particular schedules and the response was the animal's patterned behavior. However, food was not the only reward—males would work for access to sex and mothers would work for access to infant rats, and so forth.

Such behavioral discoveries were not lost on people who ran and designed gambling casinos. They programmed one-armed bandits (slot machines) to deliver cash rewards in those patterns that were ultimately most efficient in relieving clientele of their hard-earned cash (namely, variable ratio schedules of reinforcement)! Of course the casino always won, in the long run. Indeed, such money-grubbing activities and mentalities are very effective in lighting up the "reward centers" of the brain when they are monitored with modern brain imaging (Knutson & Cooper, 2005).

Olds and Milner were struck by the fact that when LH stimulation was administered at schedules that mimicked the schedules of food delivery described above (e.g., fixed ratio, variable ratio), animals worked in almost the same predictable patterns that they displayed when they worked to receive food. The difference is that animals are never as persistent in working for LH stimulation reward as hungry animals when working for food rewards. For instance, on a fixed-ratio schedule rats can easily push levers hundreds of times for each morsel of food, but for a brain reward they rarely would exceed a tenth of those levels. If the experimenter stops giving the animal LH stimulation in return for pressing a lever, the pressing also peters out quickly and stops. Often the animal proceeds to engage in relaxed self-care activities like grooming. Self-grooming is something that animals do quite vigorously after they have eaten or had sex—at times when the SEEKING system is relatively quiescent. So when the

experimenter stops giving the animal its brain reward, its SEEKING system deactivates relatively quickly. However, if one stops giving food pellets to a hungry animal, the animal will continue to press the lever for a much longer time. This is because the animal is in a state of homeostatic imbalance and this automatically sensitizes the SEEKING system, prompting the animal to continue to vigorously press the lever. Panksepp published research (Panksepp & Trowill, 1967a, 1967b) in which he found that nonstarved animals working for very high incentive treats (i.e., chocolate milk infused directly into their mouths) would often behave like self-stimulating animals, which indicates that the lack of any bodily need may have had something to do with the unusual behaviors of self-stimulating animals.

Of course, Olds and Milner had to think about the phenomenon of self-stimulation in the restricted set of concepts that were being used by behaviorists at the time. The discovery was exciting enough, perhaps the most important psychological finding of the twentieth century, but there was no incentive to think about radical ideas like the SEEKING-EXPECTANCY system, and eventually investigators paid little more attention to the differences between self-stimulating animals and those working for conventional rewards. Most researchers believed that self-stimulation simply had to reflect, in some way, the pleasures derived from conventional rewards and perhaps also the homeostatic imbalances that led animals to look for rewards. Because there are different ways to restore homeostasis (eating, drinking, or the many behaviors that could be evoked by stimulating the MFB-LH), various researchers also assumed that the LH must contain subsystems for each type of consummatory activity. One subsystem would energize eating, perhaps by inducing momentary feelings of hunger followed by the satisfaction of eating; another would energize drinking; a third, sexual consummation; and so on. A host of experiments, however, indicated that those assumptions were not correct.

If the LH were the part of the brain that registers the pleasure of consummation, then it would be aroused when animals experience the delight of consuming the goodies of the world. Neurons there would fire when animals eat, drink, copulate, and so on. Experimental data, however, do not support this view. Neurons in the LH are typically active when animals search for food, but these neurons promptly shut down when the animals find food and start to eat (Hamburg, 1971). Other experiments yielded similar results, showing that brain structures to which the LH is

strongly connected (structures that compose other parts of the SEEKING system) respond to the anticipation of rewards rather than to the rewards themselves (Blackburn et al., 1992; Fibiger & Phillips, 1986; Schultz & Romo, 1990). Thus, in the real world, namely without artificial brain stimulation, at those precise moments when animals are consuming rewards, the SEEKING system does not seem to be especially active. Instead, the SEEKING system is typically most aroused right before animals get the rewards they are expecting. In fact, as we noted, neurons in the MFB-LH tend to shut down when animals begin to eat.

There is, however, some dopamine that is released during the consummatory phase and some researchers have argued that this means LH arousal is the neural correlate for the pleasures of eating and drinking and other activities. But there is a more plausible interpretation of that fact. The dopamine release may be due to the fact that when we eat something, there is a dovetailed pattern of expectation and consummation. If you are hungry and sit down to eat a hamburger, neurons in your LH stop firing as you start chewing the first mouthful of food. When you swallow, however, you begin looking forward to the next mouthful. During that brief period of anticipation, cells in your SEEKING system start firing again and dopamine is released. Even after you are sated, cells in the LH may fire again at the thought of some apple pie with ice cream.

So it is reasonable to suppose that dopamine release and LH arousal occur in cyclical patterns even when you are in the midst of consuming a treat. Nevertheless, as a general rule, many nerve cells in the LH and in the associated structures that compose the SEEKING system typically fire more robustly before consummation than during consummation. The data are consistent with the possibility that your SEEKING system secreted more dopamine while you were anticipating your hamburger than when you were actually munching on it with a feeling of satisfaction. In addition, we must remember that dopamine is only one part of the complex neural network that makes up the SEEKING system.

And the "Brain Reward" Effects Became Stranger and Stranger

But there were other perplexing observations to be explained. Why would this kind of brain stimulation, freely given (without work), produce all

kinds of consummatory behaviors—eating, drinking, wood gnawing, copulating, and so on? This is rather perplexing behavior if the brain stimulation is producing the satisfaction, or the reward, derived from such behaviors. But this commonly observed fact led to another reasonable hunch: The MFB-LH may contain specific neural subcircuits that correspond to each of these kinds of consummatory activities. But when this was tested, the hunch turned out to be untrue. If the LH contained all these various subsystems, then as one moved an electrode around the LH in the same animal (i.e., a roving stimulation probe), different subsystems should become aroused and the animal would first display one type of consummatory behavior, perhaps drinking, and then if the probe is moved a little further down, the animal would begin another behavior, like eating or copulating. But this is not what happened (Wise, 1971). When such a "roving" electrode is used, an animal perseveres with the first kind of behavior it happens to exhibit, and then it keeps showing that behavior regardless of where you place the electrode in the "active field." If the animal is eating, it will continue to eat as the electrode moves throughout the LH. Furthermore, animals sometimes persist in activities that are not consummatory. Sometimes they gnaw on wood, carry their tails around, gather up their young, nibble obsessively on their feces, and so on. And animals would self-stimulate all of these brain sites in very similar ways. Thus, researchers gradually discovered that the "reward circuit" of the LH did not have separate neural circuits for many different kinds of consummatory activities. Rather, the system was ready to respond to any of the many survival-sustaining activities.

The most important studies that concluded that this system served some kind of general behavioral function were those of Elliot Valenstein and his colleagues, who discovered some very remarkable peculiarities about the various appetitive behaviors that animals exhibited when this system was stimulated. The behaviors were very flexible and interchangeable. If an animal vigorously ate food, in preference to drinking or gnawing available wood blocks, and then overnight the animal was continually stimulated, but now without the animal having access to any food (their originally preferred "goal object"), the next morning the animals were either drinking or gnawing wood just as eagerly as they had been eating food the day before (Valenstein, Cox, et al., 1970). And even more surprising, when Valenstein and his coworkers returned the food, the animals stuck with their newly

found behaviors. They also found many other perplexing behavior patterns. For instance, if animals first started to drink water from a sipper tube during brain stimulation, and the researchers simply placed the water in a dish, the animals were then as likely to pick up eating or wood gnawing as they were to go to the readily available water source. There were many other examples of behavior changes that were equally perplexing (see Panksepp, 1998a, pp. 153–155). The researchers pondered these remarkable findings and concluded that the MFB-LH was simply a very plastic learning system.

During this same general time frame, Panksepp was finding similar patterns, but by using brain stimulation to provoke predatory behaviors (Panksepp, 1971). Valenstein saw those findings as supporting his own conclusions, but Panksepp developed a rather different theoretical view: namely, he saw all this as evidence for a unified emotional system in the MFB-LH, one that mediated general-purpose appetitive eagerness and foraging behaviors. The system was a goad without a fixed goal, which was used for the SEEKING of all rewards and, gradually, with learning, expectancies for all rewards. In more behavioral terms, it was a general-purpose, incentive-motivational, appetitive behavior system. If this kind of system was repeatedly aroused, then animals would eventually settle on any old appetitive response that was handy and would stick with it. This was an emotional system, not just a reward system.

The Troublesome Definitive Experiments

Years later, Valenstein began to wonder whether the LH arousal signaled a generalized, nonspecific pleasure that made many kinds of consummation enjoyable, an idea that had also been advanced by Roy Wise (1982), another pioneer in the field who had originally thought that the evidence supported the existence of many consummatory subsystems running along the MFB-LH. To test this hypothesis, Valenstein asked his young faculty research collaborator, Kent Berridge at the University of Michigan, to do a critical experiment. Berridge had already done his doctoral research on the fascinating phenomenon that one could measure the levels of the pleasant taste of sugar water in rats by carefully observing what the rats were doing with their faces, especially their tongue movements, as sweet water was infused directly into their mouths. As Berridge increased the concentration of sugar, the animals would lick their chops ever more vigorously, with

their tongues lapping ever father out, almost like goofy characters in a cartoon. In short, the greater the pleasure (of "sweetness"—a sensory affect), the more intensely the rats licked their chops.

Valenstein and Berridge reasoned that, instead of increasing the concentration of the sugar water (what would correspond to pleasurable sweetness for humans), one could increase the pleasure simply by applying a little additional electrical stimulation to the LH, a general pleasure substrate. In other words, this jolt should intensify the consummatory "liking" response that Berridge was adept at monitoring. During small "squirts" of such brain stimulation, rats should lick their chops excessively, just as if small squirts of modestly sweet sugar were being infused directly into their mouths. The experiment was well done (Berridge & Valenstein, 1991). Regrettably, for Valenstein's theory, Berridge found just the opposite result. When LH stimulation was applied, the licking of chops did not increase; it diminished rather drastically. Clearly, the LH stimulation was not increasing the rats' consummatory pleasure response. Thus, the response must have been due to some other kind of reward.

Panksepp and colleagues were delighted by the results because they had already recognized that LH stimulation arouses SEEKING urges, which reflect intense foraging that typically occurs before animals find something pleasurable to consume. Indeed, Berridge himself had come to essentially the same conclusion and proceeded to cultivate his own version of the SEEKING-EXPECTANCY hypothesis. He suggested that the system mediates "wanting" rather than "liking" (Berridge, 1996). For quite a while he had a hard time convincing his colleagues of that viewpoint. To this day, the most common view remains that this self-stimulation emotional system is adequately called "the brain reward system," and recently that misnomer has been picked up by most human brain imagers whose intellectual roots go back to cognitive psychology. They are consistently finding that one of the main terminal areas for this system, the nucleus accumbens (see Figure 3.2), lights up like a Christmas tree in response to everything that humans desire and enjoy—from moving music to a good joke (Knutson & Cooper, 2005). Panksepp once asked Brian Knutson why he does not call it the SEEKING system, and he indicated that he would have trouble getting his work published if he used such a radical name. In other words, investigators of appetitive learning prefer to see their animals as passive integrators of sensory information, enhancing 'incentive salience', rather than active

organisms that have brain systems to engage with the world euphorically to meet their needs. These are fundamentally different ways of viewing how organisms were constructed in the cauldron of evolution.

There have been other theories along the way, but they will not be discussed in detail (for a recent summary, see Panksepp & Moskal, 2008). But no theory has been as inclusive, as ethological, and as emotional as the SEEKING system hypothesis. If anyone still feels that this system only mediates the good feelings created by a fine meal or superb sex, he or she has not been paying attention to all of the evidence. The most dramatic observation, one that most investigators in the field still do not focus on, is that animals getting this kind of brain stimulation frantically explore their environments, taking notice of all the new stimuli they encounter. Indeed, by organizing environments in a certain way, stimulated animals tend to become hoarders, picking up all kinds of objects when stimulation comes on and then dropping them whenever the stimulation turns off. Thus, if one arranges one half of a test chamber with piles of items that we would consider junk (corks, bottle caps, etc.), and then arranges SEEKING stimulation to come on when rats entered that side, the animals would carry all that stuff to the other side of the box, and drop it there when the brain stimulation turned off. Just another obsessive, adjunctive behavior!

In sum, LH stimulation does not produce the feeling of distinct homeostatic bodily needs—it does not produce hunger or thirst. Rather, it promotes an emotional "energy" that is conducive to autoshaping and a large number of adjunctive behaviors. The food and drink become the targets of adjunctive urges, yielding a frenzied consummation or interaction with anything sufficiently interesting that is at hand. Thus, the fact that LH stimulation can also lead to avid consumption of food and water did not indicate that any specific feeling of bodily need (i.e., homeostatic affects) had been produced. Rather, it is a substrate for being able to respond to many needs, including the need to explore one's world and to chase down interesting options in the environment. Most of this work was done before the dopamine networks of the brain had ever been seen.

THE DOPAMINE/SEEKING SYSTEM—DOES IT JUST CONTROL BEHAVIOR OR AFFECT ALSO?

In the early 1970s, when all these experiments were carried out, Urban Ungerstedt (1971) discovered an ascending dopamine system that arose from the VTA, conveyed messages through the MFB-LH, and ascended to the nucleus accumbens, all the way up to the medial regions of the frontal cortex (Figure 3.1A). In other words, it was clear that the dopamine pathways were a big part of the massive and complex MFB-LH circuitry, extending from the midbrain up to the neocortex. This circuitry was called the mesolimbic dopamine and mesocortical dopamine pathways, along with many related neural pathways, and we now know a great deal about their rewarding nature (Ikemoto, 2007, 2010). We just don't agree on what they do overall for organisms or how to talk about such a global emotional function of the brain that makes animals and humans spontaneously "active" organisms.

During the 1970s Panksepp formulated the idea that these pathways constituted a SEEKING-EXPECTANCY system. His theory, unlike all the preceding theories that saw the LH as some sort of homeostatic or generalized pleasure-reward substrate, conceptualized it as an emotional brain system that generated expectant behaviors and euphoric-enthusiastic affects that spurred animals to take possession of nature's bounties and to escape from dangers. In this view, adjunctive behaviors and autoshaping were natural consequences of SEEKING overstimulation. This alternative explanation for the frenzied activities that characterized MFB-LH self-stimulation reward recognized that the traditional behavioristic "reward" concept hid the functions of this system under one ambiguous, generic label. If one accepted the concept of a "reward," apparently one no longer had to think about all the paradoxes in the field.

Likewise, Berridge concluded that this system did not generate a sense of consummatory gratification ("liking"), but a rewarding kind of appetitive "wanting" (Berridge & Valenstein 1991; Robinson & Berridge, 1993). He and his colleagues proceeded to argue that this ascending dopamine system increased something called "incentive salience," a slightly ambiguous concept that essentially means the extent to which stimuli in the environment are attention-grabbing. In fact, this is an attribute of many emotional systems—they all help gate sensory and cognitive information into the brain (see Figure 2.1). Thus, this idea resembles just one key aspect of the SEEKING-EXPECTANCY concept—a view which maintains that mammals have an inherent urge to reach out and grab appealing stimuli and

to escape from those stimuli that are threatening. However, the "wanting" terminology tends to focus on how an animal perceives the world, while the SEEKING hypothesis includes how an animal is designed to be an actor in the world—an active agent as opposed to a passive processor of information.

There remains another critical difference between Panksepp's and Berridge's hypotheses. Berridge put the terms "wanting" and "liking" in scare quotes to indicate that they were only metaphors. He did not acknowledge that any real internal emotional experience emerged as a result of activity from the LH-dopamine (SEEKING-"wanting") system. Rather, he focused on the potency/intensity ("salience") of the sensory properties of rewards and the stimuli that predicted rewards. Thus Berridge, for a while, suggested that "liking" was intrinsically a nonexperiential process that might influence psychological experiences in the higher neocortical reaches of the human brain (i.e., it was another "read-out" hypothesis). From this view, it is hard to imagine why animals would self-stimulate, except perhaps because something about this system helped create feelings in higher parts of well-endowed human brains, which, of course, did not explain why animals with no neocortices found brain stimulation "rewarding"—they self-stimulated just fine (Huston & Borbély, 1973).

Berridge chose to envision "liking" in rats as an unconscious antecedent to human affective experiences; that way he seemed to get around the problem of how mental processes, or experience, could exist in other animals. If his interpretation is correct, Berridge is one of the most sophisticated read-out theorists (see Chapter 2). He believes that arousal of the LH "reward" system, especially the dopaminergic part, is a precursor to the conscious experience of "wanting" that arises from the human neocortex. This might be fine if we were focusing on the anticipation for specific objects and aesthetic experiences far in the future, tertiary mental processes, as opposed to desire itself.

Panksepp, on the other hand, much earlier than Berridge, proposed that the raw affective experiences of enthused eagerness—an enhanced pure sense of euphoric anticipation—arise directly from these subcortical structures that are found in all mammalian brains. In other words, he proposed that other animals are fully affective creatures that can experience their SEEKING urges in enthusiastic ways. Animals self-stimulate the LH not because it feels pleasurable, the way a wonderful meal is delightful, but

because it promotes an internal state of SEEKING that not only generates the search for resources but concurrently produces a very special positive feeling that closely resembles how we humans feel when we are full of positive excitement about the good things the world contains. But the system does not initially know what it wants, which makes the "wanting" concept rather too cognitive, and not sufficiently affective. Anyway, practically every investigator implicitly agrees that this system mediates a certain type of positive feeling in the brain, but there is currently little consensus or discussion about what this feeling is like. One key problem is that behavioral neuroscientists, as a community, are not yet ready to agree that animals have emotional experiences. Indeed, most are not yet willing to openly discuss the nature of emotional feelings in animals. This, we believe, needlessly diminishes the other animals, and thereby our own intellectual integrity.

THE SEEKING SYSTEM, CONDITIONED LEARNING, AND THE "REWARD PREDICTION ERROR"

Most neuroscientists today are not much concerned about the affective feelings that animals may have. Few acknowledge that a study of the relevant affective brain mechanisms of other animals is the only clear scientific path to understanding our own basic affective feelings. Most investigators are more intensely interested in how this "reward" system—what we prefer to call the SEEKING system—helps animal brains to learn. Because neuroscientists study the brain, they do not just focus on the rewarding and reinforcing effects of external stimuli in the environment as the behaviorists did. Instead they have focused their attention on brain regions, circuits, and neurochemistries that might mediate rewards and reinforcements. They are finally seeking neuroscientific answers to the questions that should have plagued behaviorists, had they been interested in what the mechanisms for learning are. But most neuroscientists do not recognize that the mechanisms for affective experiences, namely the neural mechanisms of feelings that are aroused by unconditioned stimuli as well as emotional unconditioned responses, are both part and parcel of the "reinforcement" processes that allow brains to learn.

At present, most behavioral neuroscientists agree that the main chemistry of this system, dopamine, is a fundamental substrate for conditioned

learning. Before the neuroscientific revolution, behavioral psychologists proposed a reward/reinforcement model to explain how conditioning happened. In classic experiments, rewards like food or drink are delivered right after animals perform operant behaviors, such as pressing a lever. Behavioral psychologists proposed that food was a reward that reinforced learning. They had no idea what the *process* of reinforcement really was, even though they all knew that the procedure of reinforcement (i.e., a response followed by an external reward) worked very well indeed. The *process* question clearly required brain research, and the self-stimulation "reward" seemed like the most obvious gateway to understanding in the early 1970s.

However, before the discovery of the "brain reward" there was always a major problem inherent in the reward/reinforcement learning theory: nobody was able to meaningfully, in terms of brain activities, explain what a reward or a reinforcement actually was, aside from things that inspired learning. The behaviorists defined a reward as food or drink for which an animal will work. But why will the animal work for food and drink? Because they are 'rewarding'! Reinforcement was defined in the same circular way. A stimulus like food reinforces learning. But how does it do so? Simply saying that animals will learn patterns of behavior in order to obtain a reward will not be sufficient. These kinds of arguments tell us nothing beyond the obvious. The study of neural mechanisms finally became attractive to many behaviorists when the "brain reward" was discovered, but they tried to keep their old terminology. To handle the troublesome concept of emotions, they suggested that emotions were entities generated by learning, namely by reinforcement contingencies (e.g., Gray, 1990), which engendered a bit of a debate: The alternative view that Panksepp advocated was that reinforcements were the manner in which emotional feelings and other affects worked in the brain to promote learning (Panksepp, 1990a).

When the newly minted neurobehaviorists began to think in new ways about learning—in terms of brain circuits and neurochemistries—they continued to cling to their traditional behavioral theory of reward and reinforcement with all of its inherent ambiguities. Many believed they had found the fundamental learning substrate in the LH-dopamine system because dopamine neurons are always active with interesting patterns when animals are conditioned.

The most modern theory in the behaviorist vein, arising from a series of dopamine learning theories, is the "reward prediction error" hypothesis proposed by Wolfram Schultz, a Swiss electrophysiologist now at Cambridge University in England. Schultz probed, with exquisite skill, the firing patterns of dopamine neurons in the brains of hungry monkeys that anticipated the signaled delivery of food. So, for example, if a flashing light signaled the delivery of a favored treat, Schultz could monitor dopamine activity when the monkey was first exposed to the light, and then to the food, and finally when the light predicted the food, and also at times when the light came on but the monkey received nothing (and was no doubt frustrated).

Schultz observed that dopamine neurons in the monkey's brain initially responded to the unpredicted delivery of food, but as the monkey became conditioned to associate a cue, like a flashing light, with food delivery, the dopamine cells gradually stopped firing to the delivery of food, and instead began to fire to the light—in other words, to cues that predicted forthcoming food. However, if the light came on and the food did not arrive, the dopamine cells showed a mild reduction in firing, which supposedly signaled "reward prediction error," which helps refine learning.

Remember that the behaviorists maintained that rewarding stimuli like food and drink would reinforce learning. Schultz maintained that, from a neuroscientific point of view, rewards initially take the form of the rapid firing of dopamine neurons. This rapid firing reinforces learning. So the rates of dopamine firing would teach the monkey that the light is a signal for food. When food is omitted, the reduction in dopamine firing is unrewarding (is a punishment), which further refines the learning. If the experimenter no longer provides any food at all when the light flashes, dopamine neurons fire more slowly and this is how the monkey learns that the light is no longer a consistent signal for food. In this way, Schultz concluded that dopamine cells, in what we call the SEEKING system, constitute a "teaching signal." But it is important to note that the studious monkeys were restrained to sit at their "desks" so they could not exhibit as many interesting behaviors as they surely would have if they were free. This was a behavioristic view of how environments control behavior as opposed to the internal urges of animals.

Schultz assumed that dopamine reinforces learning because changes in dopamine activity always attend the learning process. However, one of the

great lessons of science is that "correlations are not the same as causes." By relating dopamine-neuron firing to specific learned behaviors, one is looking at correlates, not necessarily at causes. When Schultz observed that dopamine-neuron firing changes systematically as animals are conditioned, he assumed that these neurons play a pivotal role in leading the learning process. It was equally likely that they were simply following learning that happened elsewhere in the brain. We believe that is a more correct interpretation of his fine data. Indeed, certain other lines of evidence were available that were inconsistent with the set of assumptions that seem to have led Schultz's theorizing (some are mentioned above in our discussions of autoshaping, adjunctive behaviors, and the remarkable work of Elliot Valenstein's group).

Learning Follows Quickly in the Footsteps of Emotional Arousal

The main concern of the behaviorist was "How does learning occur in the brain?" Much progress has been made on that important question. For instance, recent research on the FEAR system and fear conditioning (LeDoux, 2000), as summarized in Chapters 5 and 6, has revealed that learning relies heavily on the transmitter glutamate. Glutamate provides a gateway that allows information about a neutral (conditioned) stimulus to have access to the FEAR system—access that it did not have before. Suppose that a rat is repeatedly exposed to the ringing of a bell a moment before it receives a painful electrical shock to its paw. The pain of the shock unconditionally arouses the FEAR system. However, prior to conditioning, the ringing of the bell aroused no evident emotion, neither anxiety nor worry, only an attentive orienting response. After conditioning, the rat clearly becomes afraid whenever the bell rings—with an increased probability of pooping and peeing, and autonomic indices (heart rate and blood pressure) flying high.

These experiments demonstrated that, before conditioning, the neural pathway that carried information about the ringing of the bell did not have access to fear behaviors. After conditioning, this pathway did have access to various "fearful" responses. Access was provided by a specific molecular learning mechanism that is a conditional gateway to the behavioral and autonomic output that is, in shorthand, described by the word "fear." This is

why the conditioned rat exhibited fearful responses when the bell rang. And this molecular mechanism is the neural crux of conditioned learning (LeDoux, 2000). Absolutely no place was provided for the neural mechanisms of FEARful feelings in these schemes. That is because we supposedly must be perpetually skeptical about the possibility that animals have experiences, namely minds. But what if the experiential aspects of brain emotional activities are critical in many learning processes?

There is a similar glutamate-mediated learning mechanism in the nucleus accumbens where dopamine systems send their most important "reward" messages—namely SEEKING urges (Kelley, 1999, 2004). Overall, most of the evidence that Schultz has collected is consistent with the simple and straightforward possibility that conditioned cues gain access to a SEEKING circuitry when hungry animals are given predictable access to food. The "reward prediction error" is a complicated way to say something else—namely that animals can discriminate between cues that consistently predict rewards and those that do not. To the best of our knowledge, that distinction occurs in higher regions of the brain rather than at low levels where Schultz recorded dopamine-neuron firings. But many of those higher regions keep dopamine neurons informed of what is going on elsewhere (see Figure 3.1D).

If dopamine activity does not "reinforce" conditioned/emotional learning, then why are they so closely correlated? The short answer, as just noted, is that they receive information about the learning that is happening in other parts of the brain. That learning is setting the SEEKING system in action or inhibiting excitement when no reward is coming. This does not mean that changing dopamine activity is intimately involved in directly mediating the learning process itself, but that is a reasonable hypothesis that needs to be directly evaluated at the terminal fields of dopamine axons, especially in the nucleus accumbens. However, dopamine activity also follows the emergence of higher-order psychologically desirable states such as listening to moving music, gambling, and other everyday "addictions" in higher parts of the brain. These are cognitively mediated anticipatory states that may have originally been constructed by the patterned release of dopamine.

In addition, the initial arousal of the dopamine-energized SEEKING urge when animals are first given food in an appetitive learning situation ensures that animals take notice of conditioned stimuli. The SEEKING system is always aroused during appetitive conditioning because conditioning

requires animals to be emotionally aroused to begin with (i.e., without such unconditioned responses, learning does not happen). Under typical experimental conditions, emotional arousal is prompted by the unconditioned stimuli (i.e., pain arouses FEAR; a treat arouses SEEKING), and it is certainly likely that those unconditioned responses can open gateways to learning elsewhere in the brain. If so, this would highlight how emotional arousals are critical for many types of learning studied in animal models (e.g., see Chapter 6).

This general-purpose SEEKING response not only helps animals spontaneously look for and, with luck and skill, find the resources that they need, but also the means of escaping from danger, which they eventually need to learn to avoid. All this entails looking around and exploring the environment. So if you were in a state of irritated rage, your SEEKING system would also become aroused. In less civilized societies, you might act this out in highly negativistic ways. If your manager asked you to take on more tasks despite your heavy workload, you might very well wish to yell and let him have "a piece of your mind" but you keep quiet regardless. You would hopefully devise a graceful means of verbally sharing what was troubling you—but, of course, that requires self-discipline, which has typically been fostered by past emotional lessons. In any event, in all these situations the simple learning that is usually studied in animals follows automatically from the complexities of brain mechanisms that activate RAGE and SEEKING systems. Those emotional mechanisms may be rather different than the way the "reward prediction error" hypothesis envisions the underlying brain systems. We think the arousal of each primary-emotional process is critical in actually creating a large-scale neurodynamic that "draws" associated stimuli into its network (see Chapter 6). In other words, in emotional learning the unconditioned responses to unconditioned stimuli are as important in setting up the learning process as the unconditioned stimuli, which are selectively favored by many investigators. Remember, primal affective emotional experiences within the brain arise from the arousal of the unconditioned emotional response systems, working in conjunction with related environmental events. Although such appetitive learning mechanisms have not been worked out in great detail, much progress is being made (Alcaro et al., 2007; Kelley, 2004).

We suspect the real emotional learning mechanisms for food "rewards" are similar to those already deciphered for aversive "punishments." For

instance, in recent years, neuroscientific research on FEAR conditioning (see Chapter 6 for more details) has found that the molecular mechanism that gives the conditioned stimulus (i.e., the ringing of a bell just before a foot shock) access to the FEAR system is the crucial learning mechanism, and this requires changes in glutamate transmission so that fear-predictive signals have access to "fear outputs" as most put it, or "the FEAR circuitry" as we claim. In the former view, there is really very little interest in the "output" mechanisms. In contrast, within the affective neuroscience view, since it is the FEAR system itself that has been conditioned, one is intimately concerned with the direct study of the FEAR system itself in order to understand primal emotional learning (Panksepp, 1998a; Panksepp et al., 2011). In other words, it is possible that the conditioning mechanism is critically linked to the unconditional arousal of the FEAR system itself.

If we can translate such knowledge to appetitive learning of the type Schultz has studied, then it would be wiser to conceptualize the lower-level permissive "teaching" processes in affective emotional-system terms rather than cognitive ("reward prediction error") terms. We think the SEEKING system perspective provides a more coherent, overall vision of how the lower regions of the brain, which mediate the euphoric self-stimulation reward, are organized. Many researchers are still looking for a brain process that deserves the label *reinforcement*, independent of affective-emotional functions, but that has not yet been definitively discovered among the robust automatic learning processes of the brain. Perhaps a better way to view simple classical conditioning is to envision how the unconditioned stimulus and unconditioned response tendencies of the brain, both deeply affective, draw external information into their orbit, so that those previously neutral stimuli can come to trigger adaptive emotional responses in ever more patterned and well-structured ways.

When we really understand the neural mechanisms of raw affective experiences, we anticipate that we will have a better overall understanding of what we are talking about when we see animal learning in action. Behaviorists spoke exclusively in terms of stimulus and response. They were not prepared to consider unseen neuropsychological processes. However, a variety of primary-process affective processes do exist in the brain, and unless we conceptualize them properly, we will not understand what is really happening when organisms are learning. According to this more commonsense view, if we could erase affects from the brains of

animals in learning situations, it would not matter how we reward or punish them, because they would not learn. It almost sounds too elementary, but, of course, understanding the true neural nature of affects is hardly that. And a key fact is that all primal emotional systems innervate those basal-ganglia brain areas where learning occurs.

A more accurate understanding about most types of animal learning should entail understanding the affective mechanisms of the brain. It is possible that behavioral neuroscientists seeking to understand a nonaffective "reinforcement" mechanism have surely been hunting a "snark"—a creature that does not exist. To understand learning, we need a much better understanding of what it means to have "rewards" and "punishments" in the brain, and we need to determine how those neural mechanisms promote learning. This strategy is almost a mirror image of how these questions have been traditionally approached. Instead of just "using" rewards and punishments to promote learning, we need to understand the brain mechanisms that make objects and events into rewards and punishments. That takes us directly to the affective nature of the brain, and we postulate that this will eventually contain critical keys for understanding the mechanisms of learning.

Of course, there is not a single reward or punishment process in the brain; they come in many different kinds. But the general principle may be the same: The more primal affective brain mechanisms definitively control the operations of higher brain functions, from learning to thoughts (Figure 2.3). Unfortunately, because of the history of this scientific field, such deeply interesting alternative possibilities remain barely discussed.

SEEKING arousal and learning are intimately intertwined in a number of ways. SEEKING arousal prompts animals to go to new places where they are apt to learn. SEEKING arousal also induces them to take notice of extraneous stimuli, which is usually one of the necessary requirements for conditioned learning. But this aspect may take place completely unconsciously. The SEEKING system also eventually generates repetitive behavior patterns, accompanied by enthusiasm that is now guided and structured by the conditioned learning. However, this intimate relationship with SEEKING arousal and learning does not indicate that dopamine activity is an affectively neutral "teaching signal"; it is the affectively rich neural state that permits learning to occur. Thus, we predict it will be some yet unfathomed aspect of the neurobiology of affective circuits, perhaps

through fluctuating glutamateric transmission, where silent synapses, especially abundant in young brains, get restructured (i.e., become active synapses) in certain brain regions, yielding learning (see Chapter 6). Again, rather than looking for *reinforcement* signals, the more productive vision here may be that primary-process affective circuits "pull" associated informational events into their own "orbits," yielding ever more structured and effective emotional action systems. But this can also lead to various adjunctive behaviors, symptomatic of mania, and the autoshaping of delusions, that is a core symptom of paranoid schizophrenia. And sustained underactivity of this system surely contributes to depression.

It seems that neuroscientists like Schultz still envision the brain as an organ that learns in accordance with some kind of underlying reinforcement principle that is related to stimuli that generate fluctuations of dopamine activity. Then, on the basis of what it has learned, the brain instructs organisms either to engage or disengage with the environment. This is a passive view of the brain as an organ that learns first and only secondarily generates behavior. The SEEKING system is a spontaneous, unconditional behavior generator that takes animals to places, actively and inquisitively, where associated learning mechanisms allow them to develop knowledge structures, to guide their foremost evolutionary action tools (inbuilt emotional systems) to create more structures—more higher mental processes—which facilitate survival.

Thus, as an alternative view, we see the dopamine-energized SEEKING system not just as a learning system but as one that inherently causes people and animals to reach out and actively engage with the world in ways that promote learning. Sometimes this engagement facilitates accurate learning; sometimes it does not. All would agree with Schultz that learning is one of the main functions of the brain—a function that reflects many other interacting functions. However, we see the brain as a more inherently active organ that, before conditioning, prompts organisms to engage inquisitively with the world. Eventually we all come to engage the world on the basis of what we have learned, but the initial proclivity to become engaged, as in babies, is an unconditioned emotional affective response that is fundamentally independent of individual learning. It is an "ancestral memory" that permits learning to occur.

The SEEKING system reflects ancestral learning of such importance, that it was built into our brain organization. In other words, our primary-

process ancestral emotional tools are memories encoded in our genes that construct essential tools for living within our brains. Thus, the affective neuroscience vision is that all mammals are born with an urge to engage the world in various ways, and this is the most fundamental contribution that the SEEKING system brings to the neuroscientific table. Nothing of personal value in the world will move forward without this system. Parents and educational systems need to use this power of the mind more effectively.

According to the classic behaviorist view that Schultz has followed, the mammalian brain is primarily an organ for learning and its spontaneous behavioral and inherent affective and other psychological tendencies seem secondary. In our view, the mammalian brain is hardwired in ways that prompt us to actively interact with the world in various distinct (emotion-specific) ways. These ancestral memories (basic emotions) are refined by experiences but they are not created by them. Accordingly, important as learning is, we do not see it as the primary reason why young people and animals initially engage with the environment. Rather, learning is an automatic, *unconscious* process that enhances and refines our natural proclivity to engage with the world in ever more subtle ways, as our minds mature. Affect, on the other hand, is never unconscious. In the beginning it is *anoetic*—without knowledge; but it rapidly becomes *noetic*—imbued with the imprints of environmental affordances that constitute the beginning of knowledge.

SEEKING arousal is an anticipatory gift of nature that provides seemingly infinite opportunities for learning; with the developmental/epigenetic emergence of higher mental processes, it gradually fine-tunes reasonable expectations, working hypotheses, as in the conduct of science. This is not a subtle distinction. But it takes just a small shift in perspective to envision Schultz's fine neurophysiological data on fluctuating firing patterns of dopamine neurons as direct support for a primary-process SEEKING system.

THE SEEKING SYSTEM AND A SENSE OF TIME

There is, however, one very special way in which the SEEKING system is able to learn spontaneously. It is not the kind of traditional conditioned learning we have been talking about, and it does not appear to involve

thinking. Rather, it reflects the way that this system is able to gauge the passage of time. This system can learn to anticipate spontaneously various events, especially rewarding events that are highly predictable. When we discussed classic "schedules of reinforcement" that are commonly used in behavioral experiments, we mentioned one schedule that is of particular interest to the present discussion. These are the fixed interval experiments, where animals are allowed to obtain rewards by pressing levers, poking their noses into holes (that have photocells to automatically record those investigations), or performing any of a variety of other tasks at fixed intervals of time. Animals press their various "operant buttons"—lever-presses, nose-pokes, and such—quite slowly after just having received a reward on a fixed interval schedule, but they gradually speed up until, during the second half of the interval, they press the lever with ever-increasing frequency. When these patterns of operant behaviors are plotted on a graph, they form a scalloped shape—an apparent upward curve of anticipation. And this happens spontaneously.

Animals also show such scalloped responding when working for a self-stimulation reward. But this type of pattern also emerges spontaneously in animal brains and bodies, when rewards are given freely. Suppose that a rat is given totally free LH stimulation at regular fixed intervals, say at every 20 seconds, so that it has to do nothing at all in order to get each reward. In this experiment, the rat is not given a lever or any other device for performing operant behaviors. All rewards are free. The animal has the option of being "cool as a cucumber" and to sit back like a philosopher, and relax. Still, a remarkable anticipatory pattern emerges. The fixed interval brain "reward" produces spontaneous sniffing behaviors in the same scalloped pattern (Clarke & Trowill, 1971; Panksepp, 1981a). Indeed, aroused sniffing is one of the cardinal unconditioned signs of SEEKING arousal in rats (Ikemoto & Panksepp, 1994; Rossi & Panksepp, 1992). Thus, it appears that some kind of intrinsic learning occurs during highly periodic SEEKING arousal that gradually produces the scalloped pattern of the sniffing response.

As another spontaneously emerging indicator of the same process, more recently we have found that rats also exhibit scalloped patterns of 50-kHz ultrasonic vocalizations (Burgdorf, et al, 2000)—the excited chirping sounds that young rats make when they play (see Chapter 10). These sounds, just like invigorated exploratory sniffing, are known to be

unconditioned responses of the dopamine-energized SEEKING system (Burgdorf, et al., 2001). In other words, in an animal that has experienced this fixed interval schedule of free rewards for a while there is very little sniffing and chirping right after the brain stimulation; but as the fixed interval proceeds, sniffing and chirping rates both go up systematically at an ever-accelerating rate, until the next brain stimulation is received (see Figure 3.2). Then the measures drop down to a very low level again. In other words, the system automatically shapes into an anticipatory curve, with nothing being explicitly "reinforced." Clearly, the brain is an organ that is designed to spontaneously anticipate the future, perhaps because this system mediates "psychological time" as described at the end of this chapter.

Because an aroused SEEKING system produces both elevated sniffing and chirping, and this naturally shapes into an anticipatory pattern, then it would seem that the SEEKING system is somehow intrinsically responsive to the timing of rewarding affects. It becomes ever more aroused as the moment of reward delivery approaches. How might the SEEKING system be able to gauge this passage of time? No one knows for sure, but it is well known that many neurons have self-generated firing patterns. Although some neurons fire only when they are excited by some external influence, other neurons have some background level of activity that arises from some type of "internal pacemaker"—in other words, a clocking mechanism.

The dopamine-containing neurons of the SEEKING system have such endogenous pacemakers that normally keep them firing at a stable monotonous rate, like the ticking of a clock, especially when nothing special is happening to an animal. These neurons even keep firing when animals are asleep, but the background activity is not normally attended by the release of dopamine. The regular activity of dopamine neurons in the SEEKING system almost seems to act like the second hand of a clock, marking reasonably accurate mental time in a methodical fashion. While the system is ticking along in this way, it is in a quiescent, but informative, state. However, when the system is aroused, dopamine neurons start to "burst" and release dopamine as they fire several times in quick succession. Now the animal becomes alert and starts to explore its world. Or if the animal is asleep, it begins to dream, or at least demonstrate a REM pattern, a state characterized by high dopamine activity (Dahan et al., 2007; Solms, 2000).

Although the research has yet to be done, we can suppose that this type of neuronal bursting and increased release of dopamine take place just at the time sniffing and chirping begin to increase spontaneously during fixed interval experiments. If this system has an internal timing mechanism that can help animals predict when to exhibit eager anticipation—to be "first in line for resources," so to speak—it would be of momentous importance for understanding both the basic behavior and psychology of organisms. It is presumably this internal shaping of activity within the SEEKING system that helps explain the scalloped pattern of behavior that animals exhibit when they are required to work for their food on fixed interval schedules. Perhaps this same process is the one that keeps tabs on the passage of psychological time within our minds.

Thus, when the SEEKING system becomes aroused, the regular firing of dopamine neurons shifting into a more rapid bursting pattern may cause the animal's internal sense of time to speed up as well. We have all heard the adage that time flies when you are having fun, and this has now been empirically demonstrated (Droit-Volet & Meck, 2007). When we are happily engaged in an activity, especially when we are profitably employed and working toward a desired goal, time seems to flow freely, with no bumpy boredom. Perhaps this is because during these periods when our SEEKING systems are aroused and our dopamine neurons assume a bursting pattern, our experience of subjective time accelerates—time seems to pass more quickly, and with a mental ease that is a joy to experience.

By the same token, dopamine neurons respond to some aversive events with an inhibition of baseline firing (Schultz, 2006), but this firing also can be increased by various aversive events (Ungless, 2004), which is consistent with the arousal of SEEKING urges when various negative emotions are aroused. Indeed, if an animal is confronted with affectively negative situations, the dopamine terminal fields tend to show plasticities whereby they are more capable of sustaining negative affects, since this system can mediate both "desire and dread" as noted by Kent Berridge and colleagues (Faure et al., 2008, 2010). Bad times strengthen negative affective circuits in the brain.

When we are in pain or beset by worries—when we are having a bad time—our sense of time itself tends to slow down. Likewise, it is well known that people with Parkinson's disease, in which dopamine neurons are degenerated, have an altered sense of time. Without medicines to facilitate

dopamine transmission, these people fall into a waking "sleep"—they feel themselves to be frozen in time and live in a seemingly eventless universe of boredom, ennui, and psychological emptiness (Sacks, 1973).

Beyond these important observations about dopamine-firing patterns, we do not know how the firing of dopamine neurons computes a sense of time. In addition, we do not know how this sense of time can regulate the arousal of the SEEKING system, causing it to lie relatively dormant during the first half of a fixed interval schedule and then to become increasingly active during the second half. Although many aspects of these ideas remain to be formally tested, there are increasing data from rats that their sense of time, as in humans, is controlled by dopamine (Meck et al., 2008).

We are beginning to understand the reasons for why organisms become so marvelously anticipatory during the fixed interval timing of reward delivery, clarifying the profound mysteries about the relationship between the perception of time passing and the arousal of the anticipatory eagerness generated by our SEEKING systems. For now, we can be confident that our feeling of the passage of time is a basic psychological function that allows us to predict changing events in the environment. Whether time is also a fundamental property of the universe is more debatable (Barbour, 2000), but it is clear that we cannot coherently discuss the nature of the universe or our place in it without this evolved mental process.

ON THE PRECIPICE OF REASON: OTHER ASPECTS OF SEEKING IN HUMAN ASPIRATIONS AND DEFEATS

We have only touched on some of the characteristics of this fascinating system. There is much to learn. For example, it has been proposed that REM sleep—dreaming sleep—may generate its parade of hallucinatory events, full of emotionality and excitement, from excessive arousal of the SEEKING system (Panksepp, 1998a; Solms, 2000). In fact, it has recently been shown that dopamine cells exhibit more bursting, and secrete more dopamine, during REM sleep than during quiet waking (Dahan et al., 2007; Léna et al., 2005). It seems that the emotional mindscape of our dreams is energized by the same chemistries as the appetitive excitements of living. This suggests that a function of dreaming is to help anticipate and deal with the emotional challenges that we face. Although great progress is being made, so many mysteries about this system remain unrevealed, including

the precise ways it participates in psychotic delusions, hallucinations, dreams, and the anticipation of the future.

These limitations notwithstanding, our enhanced understanding of this system allows us to usefully contemplate some of the enigmatic aspects of human nature—for instance, the psychological trait known as "sensation seeking" (Daitzman & Zuckerman, 1980; Zuckerman & Kuhlman, 2000). Why do people enjoy engaging in dangerous jobs and sports? Rock climbers report such experiences. Even when they are in danger, they are engrossed in the business of finding the next hold, working out the way to best position their bodies and make their way up a treacherous vertical terrain. It seems that the joys of the SEEKING system keep them energized and distract them from the danger of their sport.

Consider firefighters. Many love this job, even as they voluntarily expose themselves to danger on a regular basis. Fear is a very negative affect and one would expect firefighters to dread going to work. Of course, firefighting provides a valuable service to the community and this might be a source of pride, clearly a tertiary-process emotion that could counteract the distress of facing fear on a daily basis. And fighters share a valuable sense of camaraderie with colleagues. This too could forge social bonds that could compensate for the misery of chronic fear. We will long honor the hundreds of heroes who lost their lives to try to help others on 9/11 and those who risk their lives in rescuing the victims of fires and other disasters every day.

Some creative psychotherapists might say that firefighters are mastering childhood fears and that the pleasure in mastery is one reason they may love their work. There may be merit to such viewpoints: Cognitive mastery over their emotions may help the firefighters overcome their fear of injury or death. But we could also try to understand the firefighter's love of his or her work in terms of the SEEKING system's ability to provide a euphoric affect that counteracts and sometimes even obliterates the gnawing distress of fear. Furthermore, since the SEEKING system arouses the neocortex, prompting it to work out strategies and solutions, firefighters' euphoric affects may easily get tied up in the details of their dangerous work, interspersed as it is with long periods of tedium and repetitive routines. However, when their SEEKING systems are aroused, as they inevitably will be when racing to vigorously battle dangerous blazes, firefighters will be intensely engaged in the business of putting out fires and saving people

from burning buildings. This kind of powerful and concentrated involvement provides an exciting taste of adventure, enhancing firefighters' sense of themselves as an effective and significant force in the world. These are positive affects that are provided by the SEEKING system. But we must wonder to what extent their experiences also strengthen certain negative affective circuits of their minds.

One can also theoretically imagine many other linkages to psychiatric issues. For instance, we wonder whether SEEKING arousal contributes to "narcissistic" complaints. Narcissism refers to the way that people feel about themselves. In ordinary usage, narcissism usually has pejorative connotations: It means that someone is excessively self-involved. However, narcissism can be emotionally healthy so long as one's self-regard is realistically positive. Pathological narcissism typically occurs when early life experiences have damaged one's sense of worth. These people try to make themselves feel better by overcompensating and overvaluing themselves in one way or another. Children can also become narcissistic if they have received too much unrealistic praise from their parents or teachers.

One of the manifestations of SEEKING arousal is an enhanced sense of oneself as an effective agent in the world. In the social world, this entails feeling important, attractive, successful, and superior. Dopamine generates enhanced self-esteem; this might be one neurochemical key to understanding narcissistic complaints, which would also help to explain why narcissistic problems are so difficult to treat. If narcissism is fueled by dopamine, a highly addictive brain chemical that fuels repetition compulsions, then narcissistic symptoms would be particularly gratifying and difficult to relinquish. Narcissism may also bolster a false sense of confidence and dominance that may crash like a house of cards, leading one on a path to a form of depression that could prove especially stubborn, since it strikes at the very seat of one's self-esteem.

It is also interesting to note that some narcissistic patients are apt to engage in marathon bouts of fantasizing about feats of glory in which they are starring players. This is especially apparent, and probably normal, in adolescence. Frequently these patients say that when they are so engaged, they do not know where the time goes. An overaroused SEEKING system may account for the feeling of time flying. If excessive narcissism and the

rapid passage of time are both indications of SEEKING arousal, then these anecdotal reports make new sense.

If these ideas are on the right track, it is possible that, under some circumstances, psychotherapy with narcissistic patients might be facilitated by mild doses of antipsychotic drugs. These drugs might inhibit the exhilarating pleasures of dopamine and render the patient more open to finding real-life solutions to his or her problems. Of course, the dosage of an effective drug would need to be judiciously gauged because too little dopamine activity can also promote depression.

SUMMARY

We could write an entire book about the SEEKING-EXPECTANCY system. In this lengthiest chapter we have tried, instead, to provide some of the broad outlines that describe this remarkable brain system. We believe that its function has been misunderstood for many years. It is still misunderstood by many behavioral scientists who conceive of learning in passive "information-processing" terms. They focus on studying animals in the prisonlike confinement of controlled experiments, rather than in the active framework of "information seeking" in the real world, where all mammals, birds, reptiles and complex invertebrates must proactively take care of their bodily needs as they live natural lives. In the few decades after its discovery in the 1950s, the reward-SEEKING system was seen as a consummatory/homeostatic reward substrate. In recent years it has been seen as a learning reinforcement system.

We believe that it is neither of these. This is a system that urges us to actively—proactively—engage with the world in order to find the resources that we need to thrive as well as to avoid dangers and threats. It automatically promotes appetitive learning, often in delusional ways (e.g., autoshaping). It energizes all our capabilities from the most basic impulses to the highest reaches of abstract thinking. For this reason, the SEEKING system is essential to the health and well-being of all animals, including human beings. However, malfunctions of this system can result in pathological conditions ranging from extreme depression when the system has become chronically underactive to delusional mania and paranoid schizophrenia when it is overactive. Under special conditions, it may even promote negative affects.

This system plays an essential role in the appetitive phase of essentially all other positive feelings, as well as escape from discrete punishments and relief from other bad times that are more sustained. This is one reason that addictive drugs lead to compulsive behavior patterns. For instance, the dysphoria that can be experienced during withdrawal of addictive drugs, a state resembling depression, can be alleviated promptly by taking the missing drugs again (a phenomenon that behaviorists have called "negative reinforcement"—the alleviation of punishments—instead of using the straightforward affective concept of "relief"). In other words, one of the reasons that drug addictions are so hard to treat is because the withdrawal effects are so intensely negative, that people learn to self-medicate.

When we look forward to anything, when we work toward anything, and when we vigorously try to escape from anything, the SEEKING system energizes our behaviors and attitudes. In addition to being the centerpiece of appetitive behaviors, it also creates the conditions that are necessary for many forms of learning, including operant conditioning, because it prompts us to explore new physical and intellectual terrains, and because it turns mundane activities into exciting pursuits, even in the midst of emotional upheavals. This system also promotes behavior patterns that eventually become incorporated into learned anticipatory conditioned responses. New training procedures in animals, such as "clicker training" utilize the natural tendency of animals to want to do things that seem under their own control (Pryor, 2005). In particular, it is fascinating that the dopamine system gauges the passage of psychological time, an essential ingredient for eager anticipation.

The SEEKING system, although commonly still referred to as a "reward system," has become the unacknowledged darling of the recently emerging field of neuroeconomics (Knutson & Cooper, 2005), where analysis of the SEEKING system and disgust responses in the insula, that hidden island of tissue between and under the frontal and temporal lobes, can predict when people choose to purchase items or not to buy them (Grosenick et al., 2008). If the disgust system of the insula lights up, the person will not buy; in contrast, when the SEEKING urges of the nucleus accumbens light up, the person reaches for his wallet. The same terminal region for the mesolimbic dopamine system is aroused when we listen to emotionally moving music (Blood & Zatorre, 2001). This system energizes our dreams (Solms, 2002) and many other psychological delights and, at times, horrors.

As we will see in the next chapter, this system is also very important in predatory behaviors, such as sexual stalking. Surely, our addiction with the Internet reflects the SEEKING system in action. Future research will probably reveal many other specific capabilities of this remarkable general-purpose system that is designed for SEEKING anything and everything.

CHAPTER 4

The Ancestral Sources of RAGE

Anybody can become angry, that is easy; but to be angry with the right person, and to the right degree, and at the right time, and for the right purpose, and in the right way, that is not within everybody's power, that is not easy.

—Aristotle (320 B.C.)

HUMANS HAVE A SEEMINGLY ENDLESS desire for love. If someone has "robbed" us of this emotional treasure, we discover our equally infinite capacity for grief and loneliness—and raw anger (RAGE), which can turn into jealousy and hatred. In the grip of such passions, we experience an intense desire to reach out and strike someone—not just anyone, but the individual who we believe is responsible for unleashing our fury. The outRAGE that we experience welling up into our thoughts is an ancestral treasure that helped protect us, and it still does. But our primary-process capacity for RAGE does not need an intentional object of hatred; it is a pure feeling. Of course our anger (a secondary-process emotion) always has some object that is perceived to be the cause of the RAGE. And with our abundant cerebral space for thought, we incubate hatreds—rich with various schemes for revenge—in the higher reaches of our minds. Sometimes we make realistic plans to punish our enemies. But more often we do so in fantasy, yielding no lasting satisfaction, often poisoning our minds.

Psychologists who are mainly interested in our tertiary-process levels of mind have no trouble enumerating the many nuances of our anger, even to the point where it seems to dissolve completely into a cognitive attitude.

And so it is usually defined. Jim Averill's (2010) definition states that "anger refers to *an emotional state that involves both an attribution of blame for some perceived wrong and an impulse to correct the wrong or prevent its recurrence*; aggression is *an attempt to coerce another into taking, or refraining from, some action against his or her will and not for his or her own good*" (p. 4, emphasis in original).

Averill then mentions ten questions that few have asked. They are well worth reading, from "Can a dog be angry?" to "What are you venting when you vent your anger?" In brief, for the first he suggests "My dog may growl and snap at me if I try to take away his bone; but he is not angry, for he does not know the language and concept of anger. Yet, my dog is experiencing something; he is not an automaton, and his aggressive behavior is reminiscent of anger. If not anger, then, what might we call it?" (p. 8). He proceeds to wisely use the kind of levels of analysis suggested here, and he places the dog's ire in a "secondary-process" learned irritation category, while we humans clearly have "tertiary-process" anger (as defined above). For the other question, he suggests "Nothing, I would argue. Yet, something does change . . . during catharsis, nothing need be lost, and much may be gained, namely, new insights into how things really are, perhaps not absolutely, but potentially. If that is an accurate interpretation of catharsis, it also implies a new view of emotion, one in which emotions are open to the possibilities of creative change" (p. 20). This essentially states the major goal of psychotherapy—to see your emotions clearly and to learn to use them for the betterment of our lives.

But how do we know anything about the cognitive aspects of a dog's "anger"? Do other animals plan and fantasize about the defeat and death of rivals? Do other animals experience hatred the way that humans do? We don't know. But it would be surprising if the minds of big-brained chimps and elephants do not harbor resentments. There is abundant anecdotal evidence of their tendencies to get "even," although we may never really know what they are thinking about. It is much easier scientifically to understand their primary-process feelings than their higher mental activities. We can be confident, based on hard data, that other animals have brain systems that generate both highly irritated behaviors and negative emotional feelings that deserve the label RAGE. As Averill recognized, to call it by the vernacular term "anger" takes us toward confusions that we simply can't resolve. But we can predict that the mechanisms of animal

RAGE do fuel the feelings of human anger, and now we are in mainstream science: our ideas can be falsified. They can also be supported. For instance, brain opioids inhibit RAGE circuits, and we would anticipate the obvious: Opioids should be very effective in reducing human feelings of anger, and thereby should also diminish the power of hatred and desire for revenge.

However, in this chapter we are not primarily concerned with hatred or with wrathful thoughts or plans for revenge. Hatred and revenge are tertiary processes that reflect our capacity to think about the wrongs that we have experienced and to devise detailed schemes for retribution. Perhaps most other mammals do not have the cognitive capacity to engage in such ruminations. Nevertheless they do express RAGE, which is not fundamentally designed to punish but rather to bring others in line, rapidly, with one's implicit (evolutionary) desires. To the best of our knowledge, all mammals experience RAGE toward others who are competing for resources. Because anger and hatred are the ways that RAGE unfolds within our cognitions, it is often hard to keep these interactive concepts distinct in our everyday language. This highlights an important point for all primary-process emotions—we have many emotional terms that are cognitive elaborations built upon and out of the neural energies of our basic emotions. The overall premise of this discourse is that primary-process affective arousals always participate in the diverse experiences of our higher emotional processes, but we can all agree that no one has devised good scientific methods to get at those mental subtleties, which reflect the way our cognitions are modified by our passions.

Human anger always increases in difficult times when there are many frustrations—in times of economic recession, or when certain seemingly essential resources, from gasoline to jobs to loving feelings, are scarce. Tempers are bound to flare more frequently in times of scarcity than in times of abundance. At a cognitive level, irritating disagreements can be a matter of everyday life. Feelings of vengeance flare easily, especially among youngsters who bully each other but have never been friends. All these human issues are well discussed in Pahlavan (2010). But where are the brain sources for our urge to reach out and strike someone, either verbally or physically? Can animal brain research tell us much about such issues? Our answer is a qualified yes. Many of the higher cognitive processes related to human anger and hatred remain neuroscientifically

impenetrable, particularly when the way humans use language, from frustrated and conciliatory tones of voice to accusations and cognitive peacemaking, can amplify or diminish the passion of anger. But the raw state of RAGE can readily be understood, in detail, through difficult animal brain research (Panksepp, 1998a; Siegel, 2005).

Thus, animal brain research will not let us understand the more subtle aspects of enculturated human values—ways of being that can counter our animal instincts. For example, cross-species affective neuroscience cannot tell us much about the quality of appeasement gestures and forgiveness that can quell aroused RAGE. The ability to forgive, like the ability to feel remorse, is based on complex cognitive processes that most animals may not possess. However, animal research can clarify what it means, inside the brain, to have RAGE flare forth. This primary-process feeling can, of course, lead to many reprehensible and hurtful actions among humans; these are behaviors that can, in paradoxical ways, prove to be self-defeating. Negative emotions, within the higher cognitive reaches of the human mind, seem to have a way of backfiring.

Aggression also has many faces. Among human beings, there are self-centered, narcissistic sociopaths and psychopaths, who are simply predatory and do not care whom they hurt. And, worse yet, there are people who actively want to hurt others and who enjoy doing so. We will also look at such predatory urges in this chapter, although most of our coverage will be devoted to discussing the ancestral roots of the capacity for anger. Our knowledge of these roots comes from an understanding of the details of the primary-process RAGE system of the mammalian brain. To understand the roots of human anger, we must study this powerful emotional system in great detail in relevant animal models.

Unfortunately, in recent years brain research on this system has almost disappeared from the neuroscientific scene. Why has RAGE research been cast aside? To some extent the answer is politically motivated. In the early 1990s an insensitive and politically incorrect suggestion was put forth by a chief administrator at the National Institute of Mental Health (in the United States) who was organizing a conference on the biological roots of violence. He suggested, perhaps without thinking through the issues, that inner-city ghettos were akin to jungles and that animal research could therefore help us understand the cultural problems of such ghettos. Implicitly the men who lived in inner cities were being compared to hyper-aggressive primates, was

one interpretation. This implication was seen as being both offensive and racist. The conference was cancelled. And this brouhaha has cast a shadow over neurobiological research on aggression that endures to this day.

Research on aggression also diminished because studies of rage in animals often result in one laboratory animal attacking another, a practice that is understandably objectionable to many people. Thus, practices such as cockfighting and dogfighting have appropriately been outlawed in many states and nations, and in scientific research precautions often have to be made so that one animal does not severely injure another.

But unbridled anger is not limited to any subgroup of humans, or indeed to any mammalian species. We now know enough to confidently assert that a RAGE system exists in all mammalian brains. We know where such circuits are located and we know something about the chemicals that arouse or inhibit aggressive irritability. But there is much still to be learned, including exactly how such feelings play out in the higher cerebral spaces of human minds. If experiments are designed with a degree of care and sensitivity, there is no reason that neuroscientists should ignore the potential for RAGE that is built into mammalian brains, including ours. The more we understand about the neurobiology of such circuits, the more we will understand a critically important natural tool for living that can cause much chaos in family life and society at large. And perhaps we may also generate new ideas for medicines that would control such passions—to help melt feelings of rage that have become a psychiatrically significant problem. When folks become objectionably angry, a common piece of advice is “take a pill”! No such pill really exists, but there are promising leads for medicinal development that are being neglected since it is not an accepted psychiatric indication for such development.

THE RAGEFUL FURIES OF THE MIND

A variety of circumstances unconditionally arouse RAGE: a restriction of physical activity or irritation to the surface of the body can easily provoke this feeling. At a secondary level, people and animals also feel angry if the aspirations of the SEEKING system are thwarted, such as by the sudden withdrawal of anticipated rewards. In the preceding chapter, we mentioned the trivial but common example of how anticipation can rapidly turn to wrath when a vending machine fails to deliver a promised treat. Such

disappointments are relatively mild, and anger soon dissipates. However, if you have placed an offer on your dream house, only to find that it has been snatched away by a higher bidder, perhaps by someone whom you especially dislike, your frustration will be more extreme and you may remain moody and resentful for some time. Although this would typically be called anger, we believe that the evidence suggests that this energized feeling is generated from the RAGE circuit, which we will discuss in this chapter. Of utmost concern is childhood maltreatment or neglect, which can engender anger that lasts a lifetime. RAGE can flare dramatically during times of war and social upheaval. But it is also all too commonplace for couples to endlessly squabble about minor things, and young children may be witnesses to aggression and related injustices within their own homes.

Homeostatic imbalances, such as hunger arising from food deprivation, can also sensitize the RAGE impulse. In Chapter 3, we noted that excessive SEEKING arousal can result in the emergence of adjunctive behaviors, which are useless ritualistic activities. It seems likely that adjunctive behaviors are also, in part, aroused by frustration-induced RAGE. As we discussed in the previous chapter, adjunctive behaviors occur in the lab when animals are very hungry and they cannot easily satisfy their hunger. Instead, the animals are "teased" with small morsels of food that keep them in a sustained SEEKING state. In other words, when people and animals are excessively hungry, thirsty, or sexually frustrated, and they don't have ready access to satisfactions, rage is likely to set in. Even though the SEEKING system is still in a state of arousal and even though SEEKING arousal can produce positive enthused affects, the RAGE system may also concurrently become aroused due to frustration and the two passions can synergize. Although RAGE itself is not cognitive (i.e., it is not a mental state that is created by information processing), it is destined to become intertwined with cognitive influences through learning.

For instance, subtle situations such as the loss of love are not easy to study neuroscientifically, but RAGEfulness readily arises when our social desires are thwarted. Sibling rivalry is perhaps one of the most common examples of this. If older children fear that a new baby will steal away the love of their parents, they may start to hate their new sibling. Sometimes an older child will ask when the baby will be returned to the hospital or else suggest that it might be a good idea to flush it down the toilet! These kinds of fretful cognitive responses, fertile soil for anger and hatred, are not

limited to young children. One of the easiest ways to provoke angry aggression in most adult male mammals living with a sexual partner is to introduce another male into their territories. Jealousy in human adults has given rise to violent acts throughout the ages, sometimes resulting in murder. Given the inevitable vicissitudes of even the happiest lives, it is easy to see why some moments of RAGEful arousal are inevitable features of every life. Nobody, however good-tempered he or she may be, is immune to this affective experience. It is part of our ancestral heritage. However, Aristotelian emotional wisdom (*phronesis*) can eventually make anger a balanced tool—allowing us to choose with whom to be angry, with what intensity, and for how long. Understanding and reconciliation may be the best options to aspire for in the long run.

THE NEURAL SOURCES OF RAGE

The neuroanatomy of aggression has been detailed by Alan Siegel (2005). The RAGE system runs from the medial areas of the amygdala down primarily via the curved pathway of the stria terminalis to the medial hypothalamus and then to specific areas of the periaqueductal gray (PAG) (see Figure 4.1). In all animals that have so far been tested, RAGE can be evoked by electrically stimulating these brain regions. When the current is turned on, animals will rapidly attack, usually biting objects that are in front of them. The attack becomes more intense when the current levels are increased. If these kinds of brain-stimulation procedures are carried out in human beings, people tend to clench their jaws and to report feelings of intense anger (King, 1961; Mark et al., 1972; Hitchcock & Cairns, 1973). But the subjects do not understand why they became angry—they find it hard to provide any rational reasons for their feelings because there is no realistic offensive object in sight. People find this experience disconcerting because under normal conditions human RAGE has an instigating object or event and is automatically elaborated by neocortical tertiary processes of anger and hatred, consistently attended by specific resentments and ideas about whom to blame. But those external precipitating events and thoughts are not always present. As noted, the RAGE response can also be exacerbated by certain bodily changes such as hunger. Increases in blood pressure also tend to sensitize the RAGE system (Mancia & Zanchetti, 1981). Similarly, brain pathologies, such as tumors that impinge on the

relevant circuitry, can irritate the RAGE system, making it increasingly likely that both humans and animals will exhibit spontaneous, seemingly purposeless aggressive behaviors (Blumer, 2000).

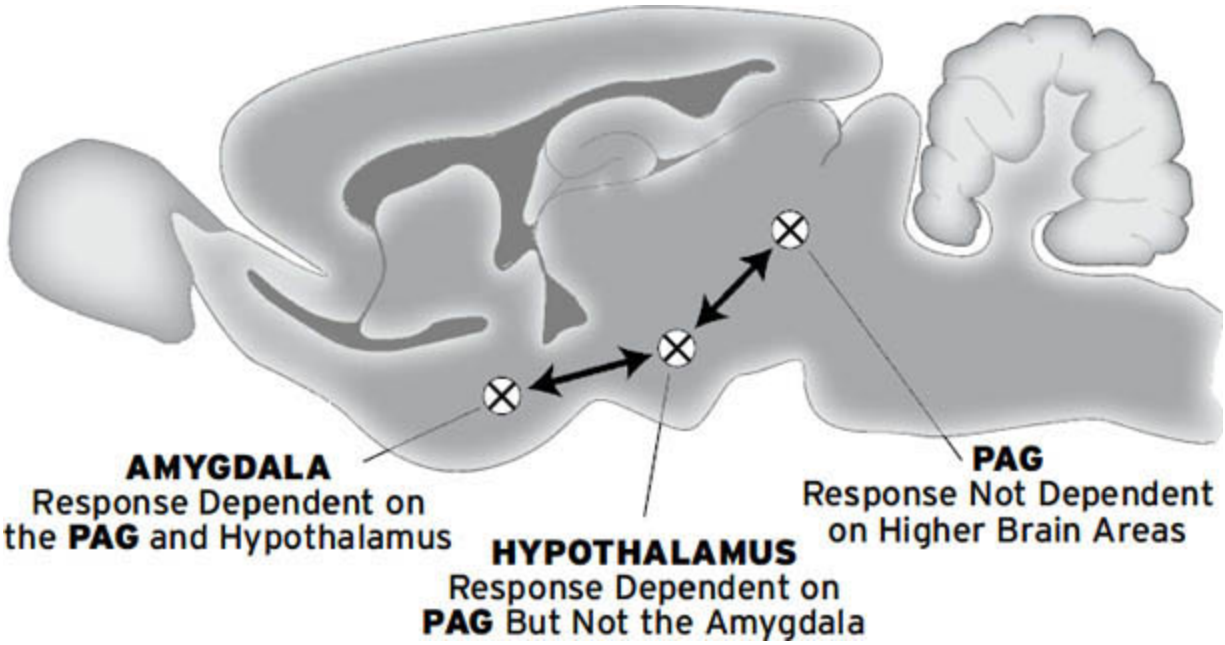

Figure 4.1. Hierarchical control of RAGE in the brain. Circles indicate major brain regions from which RAGE can be evoked with localized brain stimulation. X's indicate lesions, so that damage to the higher areas (e.g., the amygdala) does not diminish responses evoked from lower areas (hypothalamus and periaqueductal gray [PAG]), while damage of lower areas compromises the functions of the higher ones. Hypothalamic damage eliminates responses from the amgydala, but not the PAG, and lesions of the PAG markedly reduce RAGE responses evoked from the higher brain regions (from Panksepp, 1998a; republished with the permission of Oxford University Press).

RAGE circuitry is hierarchically arranged, and the deeper structures are more critically important for the actual generation of the aggressive acts than those that are located higher in the brain. RAGE evoked from the PAG is not diminished by damage to either of the higher brain regions, the medial hypothalamus or the amygdala (DeMolina & Hunsperger, 1962). Damage to the PAG or medial hypothalamus, however, can completely

eliminate rage evoked from the amygdala. And just as one would expect, damage to the middle of the system, the hypothalamus, blocks RAGE from the amygdala but not from the PAG. Thus, it is fair to say that the PAG is critically important for this emotion, with the medial hypothalamus being important, but less so, and the medial amygdala being even less important for the generation of this instinctual emotional response. But the amygdala is more relevant for establishing the cognitive linkages that come to provoke RAGE. Much of that funnels through the medial amygdala from higher brain regions that elaborate spiteful ruminations. But for the full emotional response, the PAG and medial hypothalamic areas still remain critical.

This hierarchical arrangement highlights a general principle for all of the basic emotional systems. The lower, more ancient aspects of each emotional system are more critically important for the coherent emotional responses that they generate, including the raw feeling of RAGE, than the higher brain regions. Such levels of hierarchical control are evident in all emotional networks. Unfortunately, these levels of control have not been well studied for all emotional systems, although there is good corroborative work for both the SEEKING and FEAR systems. For instance, it has long been known that lesions to lower brain regions have larger effects on self-stimulation than lesions in higher brain regions (Huston & Borbély, 1974; Valenstein, 1966).

Presumably, basic physiological “irritations” such as hunger and hormonal/sexual frustrations enter the RAGE system via other parts of the brain, such as the medial hypothalamus, that monitor bodily homeostasis. For instance, hungry animals are always more ready to fight than those that are nutritionally satisfied. How hunger links up with RAGE has so far not received much neuroscientific attention. This is a pity, especially when so many resources are being expended to find out more about brain mechanisms that are “looking for a function.”

None of this should be taken to mean that the higher controls are not important in everyday angers. Of course they are, especially for learned RAGE responses, from cognitively engendered anger to sustained hatred of someone. Lots of cognitive information from the highest brain regions can feed into the RAGE system, providing subtle refinements to the rough-and-ready emotional orchestration that is elaborated within the PAG of the midbrain. For instance, the various environmental irritations perceived by

the cortex are transmitted into the system, in part, via neocortical/cognitive inputs to the medial amygdala regions, which reside at the very top of the RAGE system. People, and presumably some animals, can use these higher controls to master the feelings of RAGE. Once again, as Aristotle highlighted in the epigram at the start of this chapter, to gain a reasoned command over one's anger is to achieve an aspect of wisdom. Sometimes, psychotherapy is of great assistance in that passage to maturity, where one becomes master of his or her emotions as opposed to their slave.

SHAM RAGE?

Several early investigators believed that the electrical stimulation of this system did not produce any real anger-type feelings but instead only generated *sham rage* (the behavioral manifestations of rage without the commensurate subjective affect). This seemed plausible because a small subset of the animals could be petted even as they were hissing and snarling (Masserman, 1941). The electrical sites that were stimulated in that subset, however, appear to have been quite low in the brain stem, namely where the actual final common pathways for the motor displays diverge into the spinal cord, or in the vicinity of the motor nucleus of the trigeminal nerve (also called cranial nerve V), which controls the vigor of biting. This may explain why the full affective RAGE response was not triggered. Such affectively vacuous brain sites are rarely found in the higher regions of the RAGE circuitry.

Now it seems more likely that most electrode placements within and above the midbrain's executive parts of the RAGE network (within the PAG) do evoke a central affective state very similar to raw human anger, except for the fact that humans are normally angry at someone for some kind of perceived transgression. Besides such cognitive components, another difference is that electrically induced RAGE is not sustained for a long time after the electrical offset, probably because there are no thoughts to sustain the feelings, or perhaps because of the sudden release of an *opponent process*,[1] such as the prompt relief when stimulation is stopped, along with the return of balance activity in SEEKING circuitry.

It is noteworthy that Walter Hess, who first discovered the RAGE system in the cat brain in the mid-1930s (he won a Nobel Prize for his work in 1949), using localized stimulation of the hypothalamus, was among the first

to suggest that the behavior was "sham rage." He confessed, however, in writings published after his retirement (as noted in Chapter 2: e.g., *The Biology of Mind* [1964]) that he had always believed that the animals actually experienced true anger. He admitted to having shared sentiments he did not himself believe. Why? He simply did not want to have his work marginalized by the then-dominant behaviorists who had no tolerance for talk about emotional experiences. As a result, we still do not know much about how the RAGE system interacts with other cognitive and affective systems of the brain.

Also, in this context we should emphasize that the hypothalamic portion of the RAGE system (concentrated in the ventral lateral and adjacent basal hypothalamus) is quite close to the SEEKING circuitry (concentrated in dorsolateral regions) as well as FEAR circuitry (concentrated in more ventromedial regions). It is therefore likely that some electrode sites stimulate RAGE simultaneously with one of these other systems. If this is the case, then the positive affect from the SEEKING urge will counteract the negative affect generated by the RAGE response. This may explain why animals sometimes self-stimulate sites that can provoke RAGE-like aggression. In contrast, the concurrent stimulation of RAGE and the nearby FEAR system (detailed in the next chapter) may produce more defensiveness and even more aversion than RAGE alone. Indeed, things can get very confusing with such mixtures, especially since we now have very good reasons to believe that predatory aggression, a topic that we will address below, is promoted by arousal of the SEEKING system.

It is important to keep such issues in mind whenever one is using localized electrical stimulation of the brain, where many systems are contiguously located and often interact in the control of behavior sequences. It is rare that only one system is being stimulated by itself. Perhaps these difficulties concerning the stimulation of adjacent networks can be resolved by emerging neuroscientific technologies. For example, the viral implantation of rhodopsin-generating molecules into specific brain regions (which can make neurons light-sensitive) can be positioned so that one can selectively activate just one brain neurochemical network from among many overlapping ones with specific wavelengths of light. Thus we are now able to more selectively stimulate just one brain system with optic fibers implanted in the correct regions of the brain (Airan et al., 2009). Likewise, local brain stimulation with specific neurochemicals can also provide

selective stimulation of specific systems, to a degree that is not possible using electrical stimulation (Ikemoto, 2010). These advances should lead to substantial refinements in our knowledge of the functional details of the basic affective systems.

THE NEUROCHEMISTRIES OF RAGE

Neuroscientists know much about the brain chemicals that influence RAGE (Guerra et al., 2010; Siegel, 2005). Chemicals that can promote RAGE, usually in the presence of other supporting stimuli, are testosterone, Substance P, norepinephrine (NE), glutamate, acetylcholine, and nitric oxide synthases. Many of these influences can be inhibited with drugs. For instance, because brain norepinephrine can facilitate anger, propranolol (which blocks *beta*-NE receptors) can diminish irritability, but this applies to other kinds of arousal as well. Other chemicals that diminish RAGE are serotonin, as highlighted especially by eltoprazine (a serotonin agonist, sometimes called a *serenic* drug, that enhances the effects of serotonin), but again this effect is not specific. Serotonin tends to reduce all forms of emotional arousal. The list of RAGE inhibitors goes on and on. Perhaps the most prominent one is gamma-aminobutyric acid (GABA), the universal inhibitory transmitter of the brain. GABA reduces RAGE activity but it also reduces rates of neural firing in a wide range of other brain activities. In other words, GABA also tends to mute every emotion, inhibit epileptic seizures, and it is quite effective in promoting sleep. Thus, just like serotonin, it is not *specific* to the RAGE system.

We only list these chemicals to highlight that every brain system is controlled by multiple chemistries. But as we will see, there appear to be some, such as the neuropeptide, Substance P, that do more specifically activate RAGE in certain higher regions of the brain (although in lower regions they promote quite different brain functions, such as nausea). Other neuropeptides such as endogenous opioids (brain morphine-mimics) as well as oxytocin (another brain social-comfort and confidence-building chemical) can also quite effectively quell RAGE. But again, they also do many other things in the brain. All this indicates that neurochemical control of certain emotions may be quite precise at the level of individual brain circuits, but they may also have different effects in other brain systems.

This is one reason it has been difficult to design more precise "mind medicines" for psychiatric practice.

Partly because each animal shows characteristic neurochemical strengths and weaknesses, emotional temperaments are bound to vary widely across individuals as well as species. Emotion-based personality scales have been developed for identification of human temperaments (Davis et al., 2003) but these would be more difficult to devise for animals. However, we can usually breed for emotional-trait differences in animals quite easily through selective breeding (i.e., through the application of "behavior genetics" techniques).

We also know that males and females have different sensitivities in practically all emotional systems. Abundant animal research suggests that, in general, females are biologically less prone to anger than males. Differences in circulating sex hormones, even in humans, are at least part of the reason for such gender differences. Testosterone clearly makes males more assertive and aggressive than females. Indeed, when human females are infused with testosterone, they soon become more aggressive and less tolerant of others (Hermans et al., 2008). Because testosterone also promotes male dominance tendencies, it seems to positively influence several distinct forms of aggression.

Of course, the testosterone/aggression link only pertains to physical aggression. There are other ways to be wrathful and other ways to inflict injury, the most egregious of which may be social rejection (MacDonald & Jensen-Campbell, 2011). When people or animals are deprived of love and acceptance, when they are spurned and forced into lower echelons of a social hierarchy where they have few rights and fewer pleasures, this is often emotionally damaging. Although social rejection does not inflict immediate physical injury, who is to say that psychological injury is not equally pernicious in the long run? After all, stress can be a killer, and social rejection induces great stress. It seems that females of a species, and certainly females of the human species, are more than capable of inflicting these kinds of emotional and social injuries on others. So although physical injury generally appears to be the domain of males, females are often more adept at meting out more subtle injuries to the psyche rather than to the body, with comparable adverse health implications (Knack et al., 2011). If anyone doubts the aggressive intent of girls, they need only delve into the social politics between girls in any classroom. A key question is whether

this tendency reflects differences in the underlying primary-process emotional systems or in the higher cognitive processes that are much more permeable to learning and culture. There is little research on such issues, but we expect that it has more to do with the tertiary processes of the mind rather than with primary ones, which means that social and cultural interventions are bound to be more important than biological ones in many cases of excessive aggression.

MULTIPLE RAGE CONTROLS IN THE BRAIN, WITH MANY UNANSWERED QUESTIONS

RAGE, just like every basic emotion, is regulated by many psychological processes and many brain regions. We will summarize some striking, albeit at times perplexing, findings, mainly to highlight how complex the overall regulation of each emotional system is and how much still remains to be learned.

For instance, certain restricted lesions to parts of the brain that are not included in the RAGE system can dramatically elevate aggression. Ventromedial hypothalamic (VMH) lesions (which make animals overeat and become massively obese, and which dampen female sexual behaviors) can also make animals chronically irritable—simply savage—and almost incapable of being handled without protective gear. And this change is lasting, hardly diminishing with subsequent repeated gentle handling. We do not know for sure why these lesions aggravate RAGE, but perhaps scar tissue from such brain damage is chronically irritating the adjacent RAGE circuitry. In humans who have epileptic foci near RAGE circuits in the medial amygdala, one sees a similar kind of chronic irritability (Mirsky & Siegel, 1994). Also, neural networks from the nearby arcuate nucleus at the very medial base of the hypothalamus, which sends signals of body energy repletion to the rest of the brain, may regulate aggressive circuits directly, inhibiting them when energy resources of the body are abundant.

On the other hand, angry irritation in animals can be ameliorated by stimulating certain higher circuits such as those in the lateral septum. This has led researchers to speculate that the lateral septum can modulate and inhibit the RAGE system (Brayley & Albert, 1977). This may explain the dramatic phenomenon of “septal rage,” whereby lesions to this midline brain region can dramatically elevate aggressiveness for up to several

weeks. The animals that have these lesions are excessively sensitive to touch and many other stimuli. This irritability can be promptly reduced by making additional lesions to the RAGE and perhaps FEAR circuits in the amygdala (Jonason et al., 1973). Likewise, with time and a lot of gentle handling, these septally lesioned animals gradually become very placid and unaggressive on their own, eventually becoming even more prosocial than normal. The simple passage of time along with nonthreatening life experiences can tone down the overactive RAGE response that follows septal damage. No comparable recovery is evident in animals with VMH lesions. The septal area sits at the crossroads of many important emotional and cognitive systems, which indicates that it is especially important for interactions between higher, cognitive and lower, emotional systems. Indeed, it is another major emotional/cognitive crossroad in the brain such as we have already seen with the nucleus accumbens for the SEEKING system, and as we shall see with lateral regions of the amygdala when we discuss the FEAR system. Thus, when the septum is damaged, cortical inhibition is curtailed, resulting in more emotional acting out at least for a while. It is by no means clear why septally damaged animals eventually become even more calm and social than they were before. But apparently they become more responsive to social rewards.

As we have noted earlier, removal of the neocortex, especially the frontal executive regions, can increase emotionality, and one of the first phenomena of this type that was discovered was “decorticate rage”—dogs and cats would become very temperamental if frontal cortical regions were surgically removed. In addition, anger is also controlled by ‘the little brain’ connected to the brain stem, the cerebellum, that was once thought to control only the smooth coordination of our movements. The deepest and most ancient nuclei of the cerebellum, the *fastigial* and *interpositus* nuclei, can generate aggressive behaviors when they are electrically stimulated. Some have thought that, perhaps just like the neocortex inhibits and regulates emotions for more measured behavioral and psychological responses, the cerebellar cortex—the outer rim of the cerebellum—might regulate aggressive behavioral tendencies. Indeed, this may be the case. For instance, Robert Heath, a neurosurgeon who did much human brain work on emotions during the era of psychosurgery (especially in the 1950s), thought he might be able to inhibit aggression in violent patients by stimulating their cerebellar cortical regions. This procedure was indeed

reported to be remarkably effective (Heath et al., 1980). But it was never adopted as common practice, perhaps due primarily to ethical concerns about such direct technological manipulations of the human brain.

To this day, we do not have highly effective ways to control pathological violence, except perhaps by drugs that produce extreme sedation. Despite all the research on aggression, psychiatry has not yet developed a viable medication that can adequately subdue persistent rage/anger, either in people or in animals. Therefore, society remains vulnerable to dangerous individuals who live in the corrosive grip of mental and emotional irritability. Because Substance P has been shown to intensify RAGE in cats (Gregg & Siegel, 2003; Siegel, 2005), we have long advocated that Substance P receptor antagonists, such as *aprepitant* (which is now medically approved for the treatment of nausea) might be quite effective as anti-anger, anti-irritability agents. However, this proposal remains to be evaluated in humans. Nevertheless, an ever-increasing number of studies show how consistently such agents can reduce angry types of aggression in animals (Halasz et al., 2008). And receptor variants within this system have also been implicated in human aggressive and suicidal tendencies (Giegling et al., 2007). In this context, it is important to note that most neurochemical receptor systems in the brain have several variants. For instance, in the case of Substance P, there are the NK1, NK2, and NK3 receptors (NK stands for neurokinin, the family of neuropeptides to which Substance P belongs). It is only the NK1 receptor that promotes aggression. Suffice it to say that, in this regard, any neurochemical that is released into the nervous system will only have a specific effect if a corresponding specific type of receptor is available. While this is an important point to keep in mind, we will avoid belaboring the details in this book, which is intended to be accessible to nonspecialist readers.

Psychiatrists will need to understand that mental health cannot be achieved simply by inhibiting an overactive RAGE system. RAGE is normally quelled by an understanding of social consequences and by the arousal of positive social relationships. If and when pharmacological "cures" for excessive RAGE are available, such medications should be accompanied by psychotherapeutic interventions that enhance a patient's ability to enjoy positive ties to friends and family. In other words, when searching for pharmacological medications, psychiatrists should not simply try to eradicate RAGE as an undesirable type of behavior. This is a general

rule: Psychiatrists should be aware of the affective interaction of the different emotional primes and should search for ways to maximize well-being, characterized by abundant positive affects that promote happiness and social harmony. Obviously, social policies are also effective tools for achieving such ends.

BRAIN IMAGES OF ANGER

Our knowledge about all the inbuilt emotional systems of the brain is far from complete. At present, human investigators, even those who perform brain imaging, have not yet visualized the ways that the RAGE system and other primary-process emotional systems work. Partly this is because they have no routine experimental methods to do causal work on these ancient emotional systems. Functional magnetic resonance imaging (fMRI) is much better at visualizing higher cognitive brain functions than lower affective ones, because the rates of neural activities in the former are much greater than in the ancient brain networks that control our emotions. These limitations mean that we cannot easily visualize, even with modern brain imaging, the intensity of the RAGE networks when they flare into action. We also cannot yet readily monitor the amounts of anger-promoting chemistries that are released in the human brain.

Further, it is rather difficult to provoke intense RAGE within the confines of human brain-imaging technologies. fMRI scans trace blood flow in the brain with the assumption that more blood will flow to areas of increased neural activity. However, in fMRI scans, subjects are required to keep their heads immobile. Thus, if strong feelings were provoked, the human subjects would actively need to inhibit the urge to express them (i.e., they would need to suppress instinctual actions) if the technology is to work properly. For this reason chemical PET imaging of brain functions is bound to be more effective for understanding lower RAGE circuits in humans.

Still, a few good studies have imaged anger. In one of the first studies, feelings of anger did lead to arousal in various midline subcortical regions, but, in particular, arousal was evident in the frontal cortical regions, especially on the left side of the brain. These brain regions also “lit up” quite a bit when people felt anxious. Inhibition of neural activity (reduced blood flow) was more evident in various higher brain regions that mediate cognitions, especially on the right side of the brain. When people were

anxious, frontal cortical areas of the brain were inhibited. When people were angry, areas farther back in the brain, including parietal regions that map the body surface, were inhibited (Kimbrell et al., 1999). Other researchers have observed comparable levels of arousal in anterior and posterior cingulate regions during facial expressions of anger and sadness, with some unique responses to sadness in the amygdala, and to anger in the orbitofrontal cortex—the cortex just above the eye sockets that participates in several affective feelings (Berlin et al., 2004; Blair et al., 1999). How many of these arousals can be argued to be primary-process manifestations of emotions, as opposed to sensory and homeostatic affects? How many are related to secondary and tertiary regulatory processes? It is impossible to know these answers in such studies; indeed, most brain imagers do not concern themselves with such important distinctions. We do not expect these lists of brain changes to be especially enlightening to the average reader, because it is by no means clear to brain imagers themselves how the scans should be interpreted.

As a good example of the ambiguity of the imaging data, the orbitofrontal cortex that lights up in imaging studies during feelings of anger probably tends to inhibit anger more than to excite it, because damage to this area often increases irritability and impulsivity in humans (Berlin et al., 2004). Also, when there is damage to nearby medial brain areas slightly farther back in the brain, such as the ventral striatum, which is part of the SEEKING system, people have great difficulty recognizing that others are angry (Calder et al., 2004). Why this is the case remains unclear, but it may again indicate how many emotions the SEEKING system is involved in regulating. Certainly if one is eager to lash out at someone, parts of the SEEKING system are bound to become aroused. If so, one can imagine that with damage to this system one might have difficulty perceiving anger-induced arousal. But why was the SEEKING system not aroused in other studies that attempted to capture anger within scans of the human brain? Perhaps it is because of the limits of fMRI technology, wherein neuronal firing has to change rather dramatically if the changes are to be detected. And, of course, the experimental conditions in the confines of an MRI scanner are simply not conducive to the experience of strong emotions. We must again remember that neurons in most subcortical regions that mediate emotions fire quite slowly in comparison to higher brain regions like the thalamus and neocortex, which fire incredibly rapidly as they mediate

perceptions and cognitions. Thus, small changes, especially in subcortical emotional regions, are hard to detect with fMRI. Different technologies that can take pictures with much longer exposures, such as positron emission tomography (PET) imaging, along with more refined experimental approaches, might be called for in the effort to adequately image emotional processes.

Indeed, by using such alternative technologies for brain imaging (e.g., PET scans), some researchers have observed strong blood-flow changes (suggesting neural arousals) in very low brain regions. During anger, Damasio et al. (2000) found strongly increased blood flow deep in the medial brain stem where the PAG, the epicenter of emotionality, is situated as well as in some adjacent brain areas such as the locus coeruleus that control overall brain arousal. This superlative PET study highlights that when anger and most other primary-process emotions (fear, joy, and sadness) are aroused, higher cortical regions tend to shut down. This suggests that strong emotional feelings can impair or narrow cognitive processing, phenomena that have long been recognized by scholars of the mind. The fact that when emotions are intensely experienced, many areas of the neocortex shut down, once again highlights where in the brain we feel our emotions most intensely, namely the ancient subcortical emotional networks that we share with other animals.

It is important to emphasize that most of the knowledge about the location of RAGE networks in the mammalian brain has been culled from animal studies, with only occasional relevant data available from humans. Thus, it is premature to conclude exactly what happens in human brains during everyday angers and resentments. However, we expect that humans would have difficulty becoming hatefully irritated if they did not have the RAGE systems in their brains.

RAGE AND WAR

It is tempting to believe that human anger contributes to the motivation to wage war, but that would be a gross overgeneralization. Even in the heat of battle, tactically effective soldiers are not usually enraged, even though such passions surely emerge in the midst of hand-to-hand combat. Obviously, a great number of sociological, political, and historical considerations play a great part in waging war. And probably the SEEKING system, as reflected

in higher emotional urges such as greed and dominance, is more influential in human warfare than are primary-process RAGE circuits. Furthermore, if our capacity for anger accounted for all wars, then we might expect to see other species engage in more collective combat; yet few other animals exhibit such group aggression. Kindred species like chimpanzees occasionally engage in communal skirmishes against other groups (Goodall, 1986) but analogies to teenage or older hoodlum gangs may be more appropriate comparisons than warlike conflict. In any event, at present it is quite impossible to say how much RAGE impulses as opposed to predatory urges have contributed to various forms of group aggression. Perhaps certain types of rage only flare once animals are actually engaged in the passionate throes of aggression when primary emotions may blaze and shift very rapidly.

Thus, very little of what we say here can highlight the causes for war in the human species. Of course, warlike tendencies in humans are ultimately accompanied by many hateful emotions, including avarice, spite, and triumph, not to mention behaviors such as raping and pillaging, but to the best of our meager knowledge, most of these complex feelings, just like our jealousies, resentments, and hatreds, are not instinctual primary-process potentials of the ancient emotional part of the mammalian brain. They probably arise from higher brain areas through developmental and social learning. Other animals are not capable of the neocortical sophistication that we possess. As a result, most other animals are simply not able to have complex thoughts and feelings about such matters in the way that we do. But this is not to say that they are incapable of more simple-minded proto-resentments, proto-jealousies, and proto-hatreds. Still, elemental emotions like FEAR and RAGE surely flare on every battlefield, and these affects stem from the emotional systems that we share with all other mammals.

THE HIGHER NEURAL REGULATION OF RAGE

In earlier chapters, we emphasized that in general the neocortex inhibits emotional systems that arouse the neocortex. We also noted that, when aroused, the neocortex, and especially the dorsolateral regions that support working memory (the ability to think strategically), can trigger and sustain emotions (for a more in-depth discussion, see Chapter 6). RAGE demonstrates these principles with special clarity. It is easy to see how the

neocortex can spark off and sustain RAGE. In his "warts and all" autobiography, the famous, neuroscientifically informed psychoanalyst John Gedo (1997) describes how he responded to his supervisor's telling him that his new course at the Chicago Institute for Psychoanalysis had not been approved because the curriculum committee had decided he was not "a mature-enough instructor to have such a privilege." On the basis of observations and deductions (all of which are cognitive/neocortical functions), Gedo became convinced that the supervisor "had personally engineered this outcome" because of past grievances about a course they had taught together. These thoughts sparked off Gedo's RAGE and he let loose. As he put it—"unrestrained by any need to appease him any longer, I told him in a voice loud enough to be heard throughout the Institute's premises that he could shove his fuckin' course up his arse! I have seldom been so angry in my life" (p. 107). Even experts on the human mind occasionally need to vent their animal instincts. But is catharsis good for you? That, no doubt, depends on whether it brings you what you wanted, and the most important things that you should want, in the long term, are mindfulness and wisdom.

Clearly, rather minor cognitive triggers can precipitate a RAGE attack, even in incredibly bright people, and at times for rather trivial reasons. Perhaps Gedo spent his wrath in this outburst; however, one can imagine that he might have remained resentful for some time, thinking about opportunities for revenge and possibly making real plans to undermine the hated supervisor. In this way, his neocortex (his thoughts) would have kept his anger alive and would have sustained the arousal of his RAGE system. However, if circumstances had been different, Gedo's neocortex might have prompted him to keep his anger in check. There are ways to distance yourself from feelings that you don't want to have, and two major ways to restore your composure are taking a few deep breaths and reflecting on who you want to be. The neocortex is always concerned with ideas about what may increase *rewards* and life satisfactions and how *punishments* will reduce well-being. Gedo vented his anger because he had already been punished and there was nothing to lose. Suppose, however, that some of his senior colleagues at the Chicago Institute had approved his course and had overridden his supervisor's opposition. Gedo might have still resented his supervisor, but he might have reasoned that any expressions of wrath might alienate other colleagues who were his allies. For this reason he might have

held his tongue and his neocortex could have inhibited his RAGE system, if he so wished.

These kinds of neocortical calculations also influence the ways that primitive RAGE plays out in the real-life interactions of other animals. For instance, if one electrically arouses the RAGE systems of more complex and cognitively sophisticated creatures like monkeys, the aroused animals usually tend to vent their rage on more submissive animals and to avoid confronting more dominant ones. However, perhaps the neocortex can take into account the fact that enraged animals are apt to gain in social status in the long run. Indeed, in a colony of monkeys, if one repeatedly stimulates the RAGE system of a particular monkey for sustained periods of time, the animal may ascend in rank within existing dominance hierarchies (Delgado, 1969; Alexander & Perachio, 1973). Perhaps having a sustained irritable mood can help an animal to overcome established dominance relationships. However, in nature, it is often the females that choose which of the powerful show-off males is allowed to ascend to the very pinnacle of perceived power. If such a male loses the favor of most of the females, he will soon be defeated by the many eager suitors waiting in the wings.

THE AFFECTIVE COMPONENT OF RAGE

We know that RAGE is an unpleasant affect not only because people say so, but also because both animals and humans will try to avoid electrical stimulation of this system. When stimulation is unavoidable, animals display escape behaviors, indicating that they wish to terminate this affective experience. Nevertheless, it does seem that some people display an appetite for RAGE and seem to enjoy feeling angry. Probably animals and people can sometimes enjoy RAGE if it inevitably leads to success (victory) in interpersonal encounters. One can easily imagine that a boxer in the ring might suffer a number of damaging blows that arouse his wrath and that he might then more thoroughly enjoy knocking out his opponent. In other words, there can be many secondary benefits to displays of anger. In a similar way, people may enjoy the experience of FEAR if they know they are in the safety of a movie theater or swinging on some carnival contraption where the body is tossed about in ways that would otherwise provoke intense negative affect.

Anger can also provide relief if it is used defensively. All defenses offer some pleasure, or at least they lessen pain and distress. For example, it feels better to hate an abandoning lover than to helplessly endure the pangs of rejected love. But for the most part, pure RAGE feels bad and this is an important consideration for psychotherapists and counselors to remember. We often see people suffering from persistent rage who at first glance appear to enjoy feeling angry. But this is probably because anger engenders a vehement demeanor that one can mistake for enthusiasm (indeed, a state of SEEKING could be aroused in the recounting of an anger episode). People may seem to enjoy being angry simply because they actively look for trouble and provoke arguments in irrational and unjust ways, possibly getting secondary benefits that only they may understand (e.g., feelings of power). Perhaps they enjoy moments of wrathful victory, but no person or animal enjoys the experience of persistent RAGE, because the affective feeling simply is not pleasant. In the vast majority of cases, chronically angry people cannot easily control their rage; some seem as if they cannot help looking for a fight, perhaps because at some stage in their lives something or someone has made them helplessly angry. It must also be remembered that under some very unusual medical circumstances, such as when people have brain tumors that irritate RAGE circuitry, people can become chronically irritable although they have no legitimate external reason to be angry.

Therapists should know that anger is a fundamentally unpleasant emotion. Chronically angry people are troubled and unhappy. They may have been angry all their lives and have never known the inner peace of having truly resolved a disagreement. They go round and round in angry altercations that commonly end in an unsatisfactory emotional stalemate. If we know that anger feels miserable and if we convey this knowledge to patients, this in itself can provide relief because angry people usually do not even consider the possibility that they are angry for a reason. Usually they simply think that they are inherently angry and therefore bad.

Years ago, August Aichhorn (1925) wrote that the young delinquent patient should always know that the therapist is on his or her side. The classical neutral stance will not do with these young people (if indeed it is ever appropriate—but that is another discussion!). Neuroscience can provide a key to establishing this therapeutic relationship because it tells us that RAGE arousal feels bad. If a patient suffers from chronic rage, a

therapist can honestly tell him that although it might sometimes feel good to gratify anger, in general it is a miserable way to feel and nobody chooses to feel angry. So if the patient feels angry most of the time, there must be a reason why. Something or somebody has sparked this anger. He did not simply choose to be angry because he is a bad person. This is one way that neuroscientific insights can help to forge an honest treatment alliance. It can provide an understanding about the nature of affective life. This understanding can serve as a basis for an empathic but honest exploration of the patient's feelings and state of mind.

At the same time it must be emphasized that aggression is not simply the RAGE system in action. It is especially important to focus on the fact that one form of aggression, so-called predatory aggression, arises largely from the SEEKING system, and people can easily confuse aggression and anger. Indeed, neuroscientists have had a difficult time accepting that the "quiet-biting" predatory attack of animals, just like our human urge for hunting, emerges more from the psychic energies of the SEEKING system than from the RAGE system. In a sense, the SEEKING system is always searching for satisfying endpoints, whether it is a predator chasing down a meal-on-the-hoof, or humans aspiring to win a contentious argument. Aggression comes in many forms, as will be discussed in the following section.

PREDATORY AGGRESSION IS NOT DUE TO RAGE

There are two major types of aggressive behavior in animals that are not pure manifestations of the RAGE system. The first of these is *predatory aggression*, which occurs when animals hunt for food. Food comes in the form of other animals that a predator kills, and we generally think of killing as an aggressive act. However, current neuroscientific evidence indicates that predatory aggression is a manifestation of the SEEKING urge. When predatory animals stalk and kill their prey, they appear to experience anticipatory pleasure rather than the harsh barbs of RAGE. Of course if the prey fights back vigorously or should happen to escape, then the animal would reasonably feel frustrated and irritable, but this would be because the SEEKING system had been thwarted without benefit of homeostatic gratification, namely a good meal.

Modern society offers few examples of predatory aggression in the human species because food is abundantly available, at least in the developed world. So there is no need for us to hunt. Foraging in supermarkets most often suffices. Predatory sexual aggression, however, is still rife in many modern societies. The extent to which some forms of rape, for example, are driven by SEEKING rather than RAGE energies is bound to arouse controversy, but in the current state of our knowledge it cannot be determined scientifically.

Most carnivores do hunt for food, and neuroscientists have carried out a number of studies on cats and rats, which demonstrate decisive differences between RAGE and predatory aggression. Virtually all cats engage in a quiet-biting predatory attack, a relatively well-controlled, if not calm, behavior of stalking, killing, and methodically biting their prey (Bandler, 1988; Flynn, 1976; Siegel, 2005). Both the stalking and the quiet biting can be generated by electrically stimulating the medial forebrain bundle of the lateral hypothalamic area, which lies at the heart of the SEEKING system. Arousal of a cat's RAGE system, on the other hand, produces dramatically different behaviors. Enraged cats growl and hiss. Their fur stands on end and they exhibit autonomic arousal (such as a rapid heartbeat, increased blood pressure, higher blood flow to muscles, and an increased body temperature). This is not the way that cats behave when they stalk and capture their prey. These data about cats indicate that predatory aggression is governed by SEEKING urges and not by RAGE.

Further evidence can be seen in laboratory rats, most of which do not exhibit strong predatory tendencies, as do wild rats. Perhaps these tendencies have been bred out of the laboratory populations. However, a substantial proportion of lab rats are clearly predatory (they readily attack smaller animals), while some are almost predatory (they show a lot of interest in potential prey such as mice, but fail to bite them). Neuroscientists have found that such *almost* full-predatory animals could be shifted into a quiet-biting mode of attack by stimulating their SEEKING systems, which again indicates that predatory aggression is clearly a reflection of an aroused SEEKING system rather than of RAGE. Indeed, these animals would self-stimulate their SEEKING systems to a point where they would exhibit a full predatory-type attack on mice. In other words, the animals had amplified their own SEEKING urge to a point where it motivated them to become predatory mouse-killers. This fully completed behavior pattern had

not been observed without the additional self-imposed artificial arousal of the SEEKING system (Panksepp, 1971).

Rats exhibit another behavioral difference that distinguishes SEEKING from RAGE. When they exhibit the quiet-biting predatory attack, generated by SEEKING arousal, they will bite both live and dead mice. However, when their RAGE systems are aroused, rats will only attack live animals. They will simply walk over dead mice (Panksepp, 1971). Apparently when animals are angry, they need a living target on which to vent their rage. Enraged animals will also attack *conspecifics* (others of the same species), but they do not regard them as prey (i.e., conspecifics are typically not appropriate targets for predatory activities).

This might be an interesting point for parents and therapists to bear in mind. When children are angry, they are sometimes urged to vent their rage on inanimate objects such as pillows or punching bags. This may, however, be an ineffective therapy because RAGE appears only to be aimed at living targets; it might even increase a child's frustration to take revenge on an inanimate object. Perhaps if the child makes an effort to fantasize that the pillow is, for example, a hated sibling, this might provide a true expression for aggression. However, this approach is ill advised. We have said that all emotional systems can be sensitized if they are overaroused. If one uses such ploys to artificially arouse the RAGE system, the result will probably not be cathartic. It would be more likely to sensitize an already precariously overaroused system. These facts have implications for violent television shows and computer games as well. Still, a sincere expression of anger in a therapeutic setting, can help establish a relevant therapeutic dialog, and also short periods of simulated acting out of anger impulses, as in simulated choking of a pillow could, with therapeutic guidance, be used effectively to move toward affective resolution of a repressed emotional urge.

In addition to behavioral differences, RAGE also differs from predatory aggression in a variety of anatomical and pharmacological ways. By stimulating different areas of the brain, one can selectively modulate either predatory attack or affective attack (Siegel, 2005). Minor tranquilizers reduce RAGE and increase the chance of a quiet-biting attack. On the other hand, amphetamines (psychostimulants) can increase RAGE while having little effect on predatory attacks. As already noted, Substance P facilitates RAGE and moderate doses of opioids inhibit RAGE, whereas low doses of opioids can facilitate SEEKING (as can several other neuropeptides—e.g.,

neurotensin, oxytocin, and orexin). Still other very general excitatory and inhibitory controls, such as glutamate and GABA, facilitate and inhibit both systems, as well as all other primary-process emotional systems. The effects of the neurochemical interactions on the various types of aggression are so complex that it would require a great deal of space to describe the enormous amount that has been discovered (Miczek, 1987; Siegel, 2005).

Most important is the fact that animals are eager to self-stimulate (e.g., to press a lever), in order to achieve electrical stimulation of the SEEKING brain sites that induce quiet-biting attacks. This indicates that animals *like* the affective feelings generated by SEEKING arousal that promotes predation. But if one stimulates the brain sites that induce pure RAGE, animals will invariably exhibit escape behaviors. Thus, RAGE generates an unpleasant affect while SEEKING feels good. So, predatory animals enjoy going in for the kill. But they don't enjoy feelings brought on by excessive arousal of the RAGE system. Of course, in all this we must remember that humans, who have much more cognitively intentional minds, may also act more impulsively than they wish, for instance, picking up a handgun or other weapon, all too commonly leading to actions that they later regret.

In sum, all these experimental findings demonstrate that RAGE and predatory aggression produce different physiological responses, behaviors, and affects. It is important to re-emphasize that abundant evidence about differences in behavior, neuroanatomy, brain chemistry, psychopharmacology, and affective experience has indicated that predatory aggression is a function of the SEEKING system rather than being an expression of RAGE (Panksepp, 1971). The predatory urge in humans, however, can often be expressed in the most antisocial ways. For instance, we have already mentioned that it is not too far-fetched to suppose that some reprehensible behaviors such as sexual stalking are partly energized by a cognitively poorly directed SEEKING urge, but we will not develop such contentious ideas here (but see Panksepp & Zellner, 2004).

INFANTICIDE AND THE SEEKING SYSTEM

There are a vast number of observations in the aggression literature that are hard to classify in terms of the types of emotions that participate. One especially fascinating finding is the case of infanticide, so common in nature, and not all that unusual in our species. In practically all species that

have been studied in the wild, though not necessarily in humans, males tend to indulge in infanticide much more than females do. There is often a reproductive advantage to this behavior: nursing females tend not to ovulate, and killing off their brood rapidly restores sexual receptivity.

When new male lions take over as dominant males in a pride of lionesses, one of their first acts is to "murder" the young offspring of the previous males; this brings the females back into heat more rapidly, helping ensure the new male's own line of descent. As we will note in the LUST chapter, the mere act of sex tends to make an infanticidal male rat much less likely to indulge in the killing of young rat pups (Mennella & Moltz, 1988). And this killing urge diminishes systematically as the time for the birth of the rat's own pups draws nearer. This is truly a remarkable fact that has been studied under well-controlled laboratory conditions, and it occurs even if the male is no longer in the presence of the female with which it copulated. We suspect that this growing peaceful tendency may be mediated by some kind of long-term, experience-dependent epigenetic effect, perhaps the facilitation of oxytocin transmission in their brains, although no definitive answer is currently available. Our point in this context, however, is that infanticide in males seems also to be an expression of the SEEKING system. Males that engage in infanticide do so in order to have sex with females, which is incidentally also one of the reasons why males engage in aggression against other males.

We are not sure if any of this relates to human behavior. Probably it does. In families, natural fathers are much less likely to abuse and kill their biological children than are stepfathers (Daly & Wilson, 2001). Maybe this happens because in the absence of stable social bonding, strange males are more liable to find the previous children of their new mate irritating, and this increased incidence of anger leads to regrettable behaviors. We simply don't know. In any event, infanticide in animals, like the inter-male aggression that leads to dominance hierarchies, seems to be an expression of the SEEKING system rather than of the RAGE system. It also remains possible that there is a distinct DOMINANCE system in the brain, but it is just as likely that dominance emerges through learning under the auspices of other primal emotional systems such as SEEKING, RAGE, FEAR and PLAY.

THE AMBIGUOUS CASE OF SOCIAL DOMINANCE

Let us consider this poorly understood psychobehavioral process that is so prevalent in most species. In addition to predatory aggression and infanticide, there is another type of aggressive/assertive behavior that is not a manifestation of pure RAGE. This is the urge for *social dominance*. The most common expression of this urge is seen between males, especially when they establish territorial rights and struggle against each other for sexual supremacy. Some people believe that the urge to dominate is an expression of specific types of brain aggression circuitry, and RAGE is the main one that we know exists. Although RAGE is often employed in the service of social dominance in general and inter-male aggression in particular, one should not assume that the urge for dominance is a direct expression of the RAGE system. Even though RAGE can surely be aroused in the midst of aggressive inter-male "tournaments" for "property" rights—be it physical access to consumable resources, territory or sexual access to females—there is evidence to suggest that inter-male aggression and the urge to dominate are quite distinct from RAGE.

Some of the brain regions that regulate inter-male aggression are also those that convey RAGE impulses (e.g., the medial amygdala and the PAG of the midbrain), but damage to others (including the preoptic area of the anterior hypothalamus, the lateral septum, the nucleus accumbens, and the raphe) can diminish inter-male aggressiveness but intensify RAGE. Inter-male aggression and RAGE can also be differentiated on chemical grounds. Most of the brain areas that support inter-male aggression have high levels of receptors for testosterone, and males without testosterone exhibit a much lower urge for dominance. We have noted that RAGE is an unpleasant affect, but recent evidence indicates that, in humans, testosterone makes men feel better than placebos but at the same time the men are less trusting and more suspicious (van Honk et al., 2010). Thus, it appears that RAGE feels bad and that the testosterone-fueled urge for intermale aggression feels good. So while RAGE and inter-male aggression may be highly interactive, it seems unlikely that testosterone is critically important in arousing the RAGE system (although it certainly promotes it to some extent).

Others have asserted that the presence of dominance tendencies in so many animals indicates that there simply has to be an evolutionarily

provided DOMINANCE system in the mammalian brain (Ellis & Toronchuk, 2005). But we do not accept the luxury of mere conceptual analysis. We do not have sufficient evidence to conclude that the urge for social dominance emerges from a single emotional system. Probably social dominance reflects learning that occurs when a variety of basic emotion systems are aroused. In other words, it is largely a secondary, emotional process with some primary-process biological disposing factors. Contributing factors include SEEKING and RAGE, as well as FEAR, and surely early experiences with the rough-and-tumble PLAY system are involved as well.

For instance, during rough-and-tumble play, juvenile rats exhibit all the behavior patterns that one might see in social-dominance encounters in adults (see Chapter 11). But when animals play, the activities are conducted in the context of positive affect, at least initially. One sees similar dominance tendencies when human children engage in rough-and-tumble play (just think of "King of the Mountain"). Indeed, in tournament species, like deer, the adult bucks approach each other just as foals do when they have an appetite for play. Of course the bucks joust in order to establish male supremacy. But given the similar behaviors, one wonders if adult jousting might be an adult variant of PLAY—behavior of the types that one sees in human "professional wrestling" and other martial arts that are currently popular entertainments for many.

Unfortunately, we know very little about the neurology of such adult behavior patterns. In particular, we lack the requisite data to demonstrate coherent emotional patterns evoked by localized brain stimulation, including reward and punishment qualities, which is our gold standard for the existence of primary-process emotions.

Still, let us pursue the alternative argument for the sake of sharing some suggestive evidence for such a system. One chemical trail promoting heightened inter-male aggression involves a molecular cascade starting with testosterone, which induces genetic expression of the activation of genes that produce vasopressin, a neuropeptide that promotes aggression and sexuality in males (Pedersen, 2004; Veenema & Neumann, 2008). Castrated male rats that have half the normal amount of vasopressin are far less aggressive and less sexually active than rats that have a normal amount of vasopressin. Injections of testosterone into preoptic regions of the hypothalamus will restore the rats' normal levels of aggression and

sexuality. These injections are also rewarding, because the animals exhibit place preferences for testosterone.

There is an old saying "them that has gets," which appears to be true of the testosterone system: Victorious experiences (whether winning a wrestling or tennis match or graduating from law school) generate an increased secretion of testosterone and a consequent elevation in male assertiveness and sexuality (Gleason et al., 2009; Strüber et al., 2008). Thus, there is no doubt that the brain has neurally based dominance type aggressive urges; in our estimation this seems to arise from the interactions of several primary-process brain emotional systems, rather than from an emotional "prime" of the type that we are discussing in this book. Still, the role of testosterone, especially in young adolescent males, is unambiguous and impressive (Lumia & McGinnis, 2010; van Honk et al., 2010). It is important to note that female hormones (estrogen and progesterone, as well as oxytocin) often inhibit aggression, so it is tempting to believe that females are, in general, temperamentally more peaceful while men are more pugnacious. However, as we noted above, females can be wrathful without engaging in physical attacks, for example, by imposing social exclusion on a rival. It is plausible to believe that females can exert their social dominance using more social rather than physical means. However, given testosterone, female personalities tend to shift toward a more male-typical spectrum (increased aggression, suspiciousness, and heightened sexuality). Indeed, as we will see in Chapter 7, female sexual eagerness and pleasure has a strong testosterone-mediated component (Tuiten et al., 2000), with those "male" hormones presumably normally being supplied by adrenal testosterone production.

We must provisionally conclude this: We do not know of any distinct brain mechanisms that substantiate primary-process forces that promote dominant behaviors in females, even though testosterone clearly can do so. In the world, this is quite evident in some species. Consider female spotted hyenas, whose testosterone levels are unusually high (further discussed in Chapter 7). Female hyenas are more aggressive than males, and they use their enlarged, penis-like, clitoris primarily for purposes of sociosexual communication, especially for dominance displays. The high testosterone levels in females appear to account for their aggressive and dominant behaviors. Perhaps high levels of testosterone also promote similar aggressive/dominant behaviors in newborn hyenas, which are commonly

born as twins. They are born with a fighting mood, and one of the two usually dies before they enter the gentler phase of youth that is characterized by friendly play fighting. It is also possible that this aggressive behavior is a manifestation of the RAGE system, but we must leave open the possibility that it reflects an early expression of an urge to dominate. Clearly, we have a lot to learn about the way the various forms of aggression unfurl in the nervous system.

The Interaction of Human RAGE, Predatory Aggression, and Social Dominance

It is not difficult to imagine how RAGE, predatory aggression, and social dominance might be dovetailed in the tertiary-process levels of the human psyche. Consider professional tennis players, who travel together from one match to another throughout much of the year. They get to know each other well. Some become friends and others less so—but sooner or later they are adversaries on the court. When friends play against each other (or even sisters, in the case of Serena and Venus Williams), they are struggling for a form of social dominance. Perhaps the adversary might also fulfill the role of prey, highlighting the role of SEEKING in such encounters. Of course, other animals are enthusiastic about killing prey in order to enjoy a good meal while athletes are enthusiastic about defeating rivals in order to enjoy a social victory. Nevertheless, in both cases, the SEEKING system is surely aroused.

Perhaps in the heat of an uphill battle, one might feel moments of RAGE against one's adversary, even if the opponent is a friend or sister. One often hears athletes speak about the "killer instinct" that is necessary to win. To some extent, the killer instinct is an expression of RAGE, especially at moments of frustration and imminent victory. However, the killer instinct is probably also derived from predatory aggression, the learned urge for social dominance, and the cognitively mediated wish to emerge as the alpha player. So it is easy to see how all three biologically promoted forms of aggression—RAGE, predatory aggression, and social dominance—can become merged in the higher mind at the tertiary-process level. Probably this is why we have difficulty understanding all of these feelings as distinct, basic emotional concepts. Reliance on primary-process emotional concepts is scientifically valid only when the concepts have been substantiated by an

abundance of robust neuroscientific evidence. Although current research indicates that RAGE, predatory aggression, and the urge for social dominance are neurobiologically distinct to some extent, only the first of them seems to be a distinct emotional system that is dedicated to a primary-process form of aggression.

SUMMARY

We have described the RAGE system in terms of its anatomy and chemistry. We have also described how RAGEful behavior is manifested in people and animals. Of special importance is the fact that the RAGE system produces unpleasant affects, even though there might be immediate pleasure in defeating a rival. This is a point that everyone in the mental health professions should bear in mind. Although males tend to be more aggressive than females, this pertains only to physical aggression. And we understand far less about the subtle aspects of psychosocial aggression in which females abundantly engage.

We have distinguished RAGE from predatory aggression and from infanticide, which both appear to be manifestations of the SEEKING system. We also discussed the urge for social dominance, the neural bases of which are not entirely clear. However, we do not think that the urge for social dominance reflects the existence of a single primary-process system. Dominance behaviors probably result from learning that occurs when a number of emotional systems are aroused. Certainly, dominance behaviors emerge when children play, and in our ancestral environments, when hunter-gatherers consisted of extended families, this type of activity among the young could easily have led to natural dominance hierarchies that lasted into adulthood. There is also the consideration that inter-male dominance seems to be propelled by testosterone and vasopressin, linking it to the LUST system.

One thing is abundantly clear. Violent crime is a social problem of mammoth proportions, and this highlights the need for medications that can suppress RAGE (at present Substance P antagonists such as aprepitant need to be evaluated in humans). However, psychotherapists and psychiatrists should also keep in mind the interplay of emotional systems and should understand that RAGE is sensitized when people, especially as children, are subjected to abuse and neglect. A key to recovering from pathological

RAGE is to establish or re-establish a person's capacity to form and sustain warm trusting relationships. Consistently friendly and positive interactions can have a wonderful soothing effect on angry souls (just think of those animals with septal lesions that became tame with time and pro-social experiences). Likewise, positive emotional experiences in therapeutic contexts can probably help dull the edges of many kinds of troublesome memories. Psychotherapy can help patients to rid themselves of issues that would otherwise fester as negativistic and irritating ruminations. William Blake (1793) noted this in his deeply passionate and humanistic poetry; for instance, in *Poison Tree* he reflected:

> I was angry with my friend.
> I told my wrath, my wrath did end.
> I was angry with my foe:
> I told it not, my wrath did grow.

This is still true today—a seemingly universal human experience—reflecting how our higher mind interacts with our primary-process potentials for RAGE.

CHAPTER 5

The Ancestral Roots of FEAR

Never, in his brief cave life, had he encountered anything of which to be afraid. Yet fear was in him. It had come down to him from a remote ancestry through a thousand lives. It was a heritage he had received directly . . . through all the generations of wolves that had gone before. Fear!—that legacy of the Wild which no animal may escape. . . . So the gray cub knew fear, though he knew not, the stuff of which fear was made.

—Jack London, *White Fang* (p. 52)

IN THIS CHAPTER WE WILL discuss the nature of FEAR—the primal terror that President Franklin D. Roosevelt highlighted in a famous speech on March 4, 1933 when he advised the nation: "The only thing we have to fear is fear itself—nameless, unreasoning, unjustified terror which paralyzes needed efforts to convert retreat into advance." These prophetic words were uttered in the year that America started to crawl out of the Great Depression . . . and it was the year that Adolf Hitler came to power in Germany (which led to profound fear and misery for millions).

We learn to dread fear itself if we have already endured terrifying experiences. Through all the wars of history, young warriors have felt the fear of death around them. The longer it has lasted, the more deeply that fear becomes engraved in the synapses of their brains, sometimes rendering them pointlessly, painfully, and perpetually fearful of everything—and in a sense, of nothing at all. The objectless fear of chronic anxiety then emerges directly from their overactive primary-process FEAR system, rather than from the actual reality of their current situation. It is hard for most of us to imagine such an "objectless" fearfulness, but this is the kind of free-floating chronic anxiety our FEAR system can produce. This system, like all

emotional systems, behaves like the sinews and muscles of our bodies. The more they are used, the stronger they become; the less they are used, the weaker they become. Many soldiers in the great wars of the twentieth century experienced "shell shock," now known as Post-traumatic Stress Disorder (PTSD)—the gradual penetration of fearfulness as an ever-present irritation of the soul, with many horrific images engraved into the memorial surfaces of minds. All mammals can be afflicted with PTSD because we all have very similar ancient FEAR systems that can become sensitized and full of trepidation within the cognitive darkness of our core affective consciousness.

The FEAR system can become hypersensitized when we have been frightened badly enough or for long enough. From birth, this capacity for free-floating fear is built into our brains; initially it can be activated by only a few *unconditioned stimuli*, but experience can create fearful memories that henceforth can be triggered by previously neutral events of the world. FEAR, like every other emotional system, is born essentially "objectless," and, like all other emotional systems of the BrainMind, it becomes connected to the real world through learning. Obviously, it is not enough for a mouse just to be capable of feeling afraid. It has to learn to fear various specific objects and situations. So do we. Evolution created the capacity for fearfulness in the brain, but it did not (and could not) inform us of all the things we might need to fear and avoid. Practically all that has to be learned. And because we are so intelligent, we humans can learn to fear more things, past and future, than a little mouse can (see the epigraph for the next chapter, where Robert Burns poignantly depicts the differences of fears in mice and men). In multiple senses, we humans are the most fearful creatures on the face of the earth. We can create fears for ourselves beyond the imagination of any other species. Because of our neocortical capacities, we even come to fear insubstantial phantoms of the mind. But we do not quite know how that kind of intrinsic learning happens. As we will discuss in the next chapter, we do, however, know a great deal about how the simplest forms of fear learning occur in the brain.

We do not have to think deeply to find moments in our lives when we were consumed by fearfulness, especially when we were young. We frequently have endured such states even when our higher cognitive minds could easily have coaxed us into recognizing that we faced no real threat. We can even be anxious about becoming anxious. We do not know whether

other animals are capable of this sort of second-order, self-generated anticipatory anxiety. But their FEAR systems—like ours—were designed to anticipate bad things in the future, and they surely become sensitized and overactive in various intimidating situations if they have been repeatedly traumatized. In other words, we know that the FEAR networks of the brain can be over-responsive in all mammalian species, just like all the other basic emotional processes of our brains.

So imagine that you are alone, lost in the woods, in the darkness of night (see Figure 5.1). You have carelessly lost your way on a hiking trip and have little confidence in your ability to find a way out. The moon filters through racing clouds on the heels of a chilly wind. The branches above sway menacingly. Your imagination runs wild, envisioning all manner of horrors, from predators to the ghouls of your dreams. These visions are as terrifying as the monsters that populated the landscape of your childhood imagination when the lights went out, even when safe in bed . . . but too often alone (since sleeping with a mother, which is what all other primates do, has gone out of fashion for us humans). Suddenly, a branch cracks and falls behind you. If you had heard this sound in the safety of your backyard, you might only have turned toward it in mild surprise. But because you are already frightened, you experience a violent startle. You hold very still for a moment, frozen in one position, as your mind fills with dread. All your senses are riveted on the location of the sound as you rapidly analyze its possible sources. Is a mountain lion about to pounce? Are bats swarming overhead? In your fearful delirium you might even envision a mythical werewolf. If you feel in imminent danger, you may explode into a vigorous flight pattern, running faster than you thought your legs would ever carry you. If you are fortunate enough to find a place of safety (perhaps an abandoned cabin in the woods), you will hide, trembling with a throbbing heart (and not just from your physical exertions; FEAR is always accompanied by an aroused autonomic nervous system). You may have wet your pants, or worse, along the way. You remain alert for a long time in a cold sweat as you vigilantly evaluate each new sound, each shadow that might indicate danger.

Figure 5.1. A cartoon of a prototypical FEAR sequence: Lost in the woods in the dark of night, one tends to freeze at any sudden sound, as goblins of the imagination generate many scary possibilities leading to flight. Even finding the safe haven of an abandoned cabin leaves one in a state of anxious arousal, which may recur in dreams on many subsequent nights (this cartoon was originally drawn for this book by Sandra Paulsen, and was fine-tuned by Lonnie Rosenberg).

Fortunately, at daybreak you find your way out. On future occasions you will be more careful not to get lost. You may dream about the episode for several nights. Had you really encountered a mountain lion or a wolverine, your fearful behaviors might have been adaptive. If you had screamed and run about like a raving lunatic, especially flailing your hands back and forth, you might have scared the animal off. Following your ordeal, mixtures of pure FEAR and various associated thoughts might incubate in the neural substrates of your psyche—perhaps for years to come. And you might even develop a mild form of PTSD. When such emotional systems have become oversensitized, you might experience the debilitating agony of "FEAR itself," even when you cognitively know that you are safe.

Fear is agonizing in all its forms. It is horrible to be stricken by sudden terror. It is also terrible to be continually consumed by persistent feelings of anxiety that gnaw away at you, destroying your sense of security in the world. Such feelings are generated by a coherently operating primal brain system, running from the periaqueductal gray (PAG) to the amygdala and back again. This system produces terror when it is precipitously aroused, and it promotes chronic anxiety in response to milder, more sustained arousal. When fear stimuli are far away, the higher cognitive parts of the brain, such as the medial frontal cortex and amygdala, are also aroused; you

may hide and be still. But when a fearful predator is at your heels, then the lower regions of the FEAR circuitry, especially down in the midbrain PAG, take over (Mobbs et al., 2007). Those unconditional fear circuits absolutely compel you to take flight.

THE INTRINSIC FEAR SYSTEM OF THE BRAIN

Many people still believe that the capacity for fear is learned and that both people and animals learn to fear by anticipating danger. If this were true, we would not be afraid of anything at birth. Only after being hurt in some way would we know what it means to be afraid. But animals exhibit an innate capacity to be afraid even when they have never experienced pain or danger. We know this because electrical stimulation of specific parts of the brain, as described in the next section, can generate the full spectrum of fear responses in animals that have been reared in complete safety. The electrical stimulation does not carry any information about danger in the environment or about the physical sting of pain. Direct stimulation simply arouses the intrinsic affective potential of the FEAR system—it arouses fear itself.

This is a point that researchers should have appreciated more than half a century ago, in the 1950s and 1960s, when they initially found areas in the brain not only that animals would voluntarily self-stimulate (Chapter 3), but also other nearby areas where stimulation would make them flee as from a psychological plague—areas that would even motivate learned escape behaviors (Delgado et al., 1954). In the course of their investigations these researchers had stumbled across the primal FEAR system, which courses near many of the brain regions that animals self-stimulate. When these FEAR structures were inadvertently stimulated, animals exhibited various fearful behaviors, freezing at low current levels and precipitous flight when the current was increased. Thus, long before the FEAR system had been formally conceptualized (Panksepp, 1982), it could have been surmised that laboratory rats had innate FEAR systems. This was evident, because the rats were afraid of certain types of brain stimulation when no learning about peripheral aversive events, such as commonly used foot shocks, was involved. All mammals stimulated in these areas behaved fearfully. Clearly, this system had been laid down by evolution rather than by the life experiences of animals.

Long ago, during early vertebrate brain evolution, recognition of certain threatening external stimuli became encoded in the brain-building DNA of our ancestors, yielding innate fears of certain stimuli that consistently caused pain or forewarned of danger. For instance, rats are innately afraid of the smells of certain predators, such as cats, ferrets, and foxes. They are not initially afraid of the appearance of these predators, only of certain aspects of their odors. If one places hair from such creatures in the cages of rats or mice that had grown up in the complete safety of a controlled laboratory setting—animals that have never encountered any predators in their lives—they would nevertheless exhibit FEAR responses. Many animals will simply freeze; others exhibit a generalized wariness (increased "risk assessment" as investigators labeled the cognitive worry-type aspects of such emotions). Even after these scary odors are removed, the rats and mice will remain timid for a long time, due to a symphony of fearful neurochemistries that have been released within their brains. Rats' social activities will be inhibited for quite a while, and they will engage less in play, feeding, grooming, sexuality, and other positive behaviors (for a depiction of some relevant data, see Figure 6.1 in the next chapter). If the animals are subjected to such stressors for too long, they begin to exhibit depressive symptoms. This innate capacity to fear the smell of predators promotes survival because the inherited FEAR system motivates animals to freeze and hide when such predators are nearby, and to flee if the predators get too close. One fearless encounter with such a predator is one too many, from the evolutionary point of view.

The fear of odors emanating from predators helps animals avoid locations where predators dwell rather than being a signal that a predator is near. We can conclude this because predatory odors enter rodent brains via their vomeronasal organ, which detects large nonvolatile molecules, rather than their main olfactory bulbs that monitor relatively faraway odors that are "on the breeze" so to speak (Panksepp & Crepeau, 1990). It is the same with mice. The molecular composition of this offensive smell has recently been identified in cat saliva; it turned out to be a single molecule belonging to the major urinary protein family, known as Feld4 (Papes, et al., 2010), which had previously been identified by this same group of investigators to intensify inter-male fighting among mice. Presumably, mice fight readily with strangers, in part, because the smells they carry make animals wary of each other.

In addition to fearing pain and the smell of predators, rats inherently fear well-lit open spaces, sudden movements, and loud noises. All these stimuli indicate possible danger, and they have been handed down as evolutionary memories (i.e., hard-wired sensory inputs into the FEAR system), because it is adaptive for the rats to innately fear them all. A few stimuli that arouse innate FEAR exist in all species of mammals. Pain is the universal provocation. Most animals also become afraid when they hear loud noises. Human infants can become anxious when they are not securely held, and as they grow older, many babies tend to cry when left alone in the dark. It is possible that these negative feelings arise as much from the social PANIC/GRIEF system (Chapter 9) as from FEAR. In fact, without brain research, it may be hard to distinguish when one or the other of these "anxiety" systems is more active. It is possible that they can be active concurrently, but such issues have yet to be studied by neuroscientists. In this chapter we will focus on the FEAR system only. Like all the other primary-process emotional systems, it is born relatively "objectless," but mammals can rapidly learn to respond to many stimuli that predict FEAR-invoking conditions.

All young animals initially only have a few intrinsic inputs into their FEAR systems, with pain being the most well understood. Pain stimuli enter the PAG directly, and as a result there are also pain-inhibitory mechanisms there. Given stimulation at the right place within the PAG, one can alleviate fairly severe pain in humans (Mayer et al., 1971; Richardson & Akil, 1977), because of the release of endogenous opioids (Hosobuchi et al., 1979; Herman & Panksepp, 1981). However, if one hits a fearful site in the PAG, animals show a full FEAR response. This FEAR state probably promotes learning, and it is probably the way that animals quickly develop acquired fear responses to the visual and auditory stimuli associated with predators (see the next chapter, which is devoted to fear-learning and memory). In this way, the FEAR system is brought under the control of a great number of life events, at both the simple learning (secondary-process) and the more complex cognitive (tertiary-process) levels.

The fact that people and other animals exhibit free-floating anxieties indicates that they have an inherent capacity to experience FEAR. In other words, the capacity to become anxious is part of the evolved emotional toolbox of the brain. As we have already mentioned, the proof of this is the simple fact that one can easily provoke a full set of behavioral and

physiological FEAR responses merely by electrically or chemically stimulating specific brain regions. Such responses are evident across all mammals that have been studied. Animals dislike such feelings by practically all measures that have been taken—they try to escape from stimulation, avoid places where such stimulation has occurred, and so on. Of course, learning can add much to the FEAR system (see the next chapter), but our key point is that learning does not by itself account for the capacity to fear. This basic capacity is provided by an intrinsic emotional system in the brain.

The capacity for fear can only be eliminated or attenuated if the FEAR system itself is destroyed in some way or if access of sensory inputs to the system is blocked in some manner. This can happen through injury or disease. For example, there are parasites (e.g., *Toxoplasma gondii*, a parasite commonly found in cats) that can attack the rodent's FEAR system and render rats less afraid of cats (Vyas et al., 2007). This facilitates feline predation. Cats eat more rats because their prey don't hide and run away as readily as usual. So the infected rodents enter into the stomachs of cats. And the cat's body is the perfect environment for the protozoans to finish their reproductive cycle.

THE BRAIN TRAJECTORY OF THE FEAR SYSTEM

The existence of an intrinsic FEAR system is most directly supported by experiments that use direct electrical stimulation of the brain (for a recent overview, see Panksepp et al., 2011). When electrical stimulation is applied to specific parts of the mammalian brain, in the deep subcortical regions that all mammals share, the animals exhibit innate FEAR responses even when there is no frightening stimulus in the environment. Different strengths of electrical stimulation produce different levels of fear. Mild electrical stimulation produces wary, subdued activity, with occasional bouts of frozen immobility similar to the kinds of inhibited behavior exhibited by rats when cat fur is placed in their cage—these behaviors are common when predators may still be far away. If the current is turned up still further, at the same brain site, the animals will flee, just as rats do when a cat gets too close and is ready to attack (Panksepp, 1991). With these

progressions, the cascade of neural arousal moves from milder forms of fearfulness (freezing, worry) to more intense forms (flight, terror).

In humans, increased feelings of fearfulness have been observed when the FEAR system is aroused by internal physical stimuli, such as epileptic activity in the parts of the limbic system where this emotional network is situated. Epilepsy is an electrical storm in the brain. When this storm encroaches on the FEAR system, the person (or animal) exhibits an intense internal fear, perhaps in a way that feels similar to PTSD (Adamec, 2001; Pincus, 1981, 2001). So the electrical current generated by the epileptic fit can act in a way that is very similar to direct electrical stimulation of FEAR circuitry in the laboratory.

Electrical stimulation experiments have revealed that the innate FEAR system is a two-way pathway that runs from the central zones of the amygdala to the anterior and medial hypothalamus, surrounding the third ventricles, and from there to specific (dorsally situated) areas of the PAG within the midbrain (see Figure 5.2). The FEAR system includes specific autonomic and behavioral outputs that control the physiological symptoms of fear (such as sweaty palms, rapid heartbeat, freezing, or running away). Pharmacological and surgical dampening of activity along this system can make both animals and humans placid. In short, the many *unconditioned*—instinctual—expressions of FEAR emerge directly from this neural system.

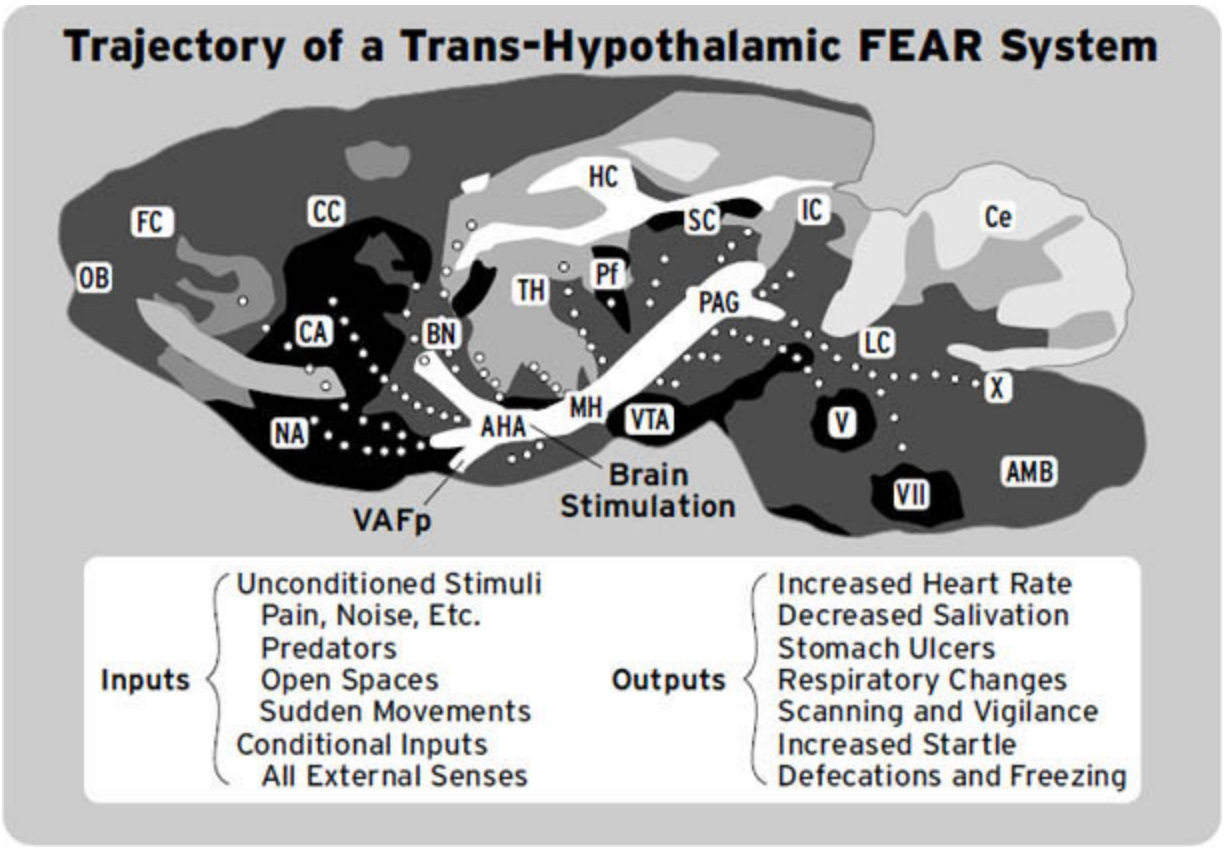

Figure 5.2. Schematic summary of the trajectory of the FEAR system and the various symptoms induced by stimulation of this emotional system (adapted from Panksepp, 1990b; see original for anatomical designations). The darkness of the brain regions approximates the levels of acetylcholine in the brain.

Many experiments have shown how much animals dislike this kind of brain arousal. Animals try to escape from it. And they quickly learn to turn off such stimulation if given a chance, by pressing levers or simply by moving to locations where the stimulation never occurs (Panksepp, 1991). They also exhibit conditioned place avoidance: they avoid places where they have received the stimulation (Roberts & Cox, 1987). If animals are exposed to environments where such brain stimulation occurs, they will avoid going back to those locations if given an opportunity to choose. But even when they are in a nearby safe area, they will still appear to be nervous, often freezing and pooping more than normal—thus, still

exhibiting the behavioral and autonomic symptoms of fearfulness (Panksepp, 1991).

Thus, the objective correlate of the FEARful affective state is the visually evident freezing and flight that such stimulation produces. Once again, the evident behavior evoked by brain stimulation is an objective equivalent; it is the external indicator of the mental state that we cannot as readily observe. However, in line with the affective neurosciences strategy, comparable brain stimulation should evoke the spontaneous verbal indicators of the aroused emotional state within humans. Indeed, that has consistently been observed.

Humans stimulated in such brain areas consistently report a sudden onset of fearfulness and anxiety. For instance, when stimulation to the PAG was turned on, one of the original subjects said, "I'm scared to death" (Nashold et al., 1969). In another study that observed psychological changes following electrical stimulation of the periventricular gray in humans (Amano et al., 1979), patients reported "an abrupt feeling of uncertainty just like entering into a long, dark tunnel", a sense of being by the sea with "surf coming from all directions" and, "Somebody is now chasing me. I am trying to escape from him." The arousal of the FEAR system quickly triggered anxiety-ridden scenarios in the cortex, perhaps from real past occurrences or maybe just from remembered stories. The speed of this interaction makes it easy to understand why people would think that the feelings are created in the cortex—that the ability to picture such scenarios is necessary to have the feeling. But remember, the initial feeling of fear came from stimulation of the deeper emotional system, and this system is shared by all mammals, regardless of their cognitive endowment.

PAIN AND THE FEAR SYSTEM

Pain always arouses the FEAR system to some extent, but the reverse is not true. Fearfulness can actually diminish the perception of pain (Miczek, 1991). When the FEAR system is electrically stimulated in the human brain, people report fear but not pain. When this system is electrically stimulated in animals, they exhibit fear but rarely screech or yelp as they do when they are actually hurt. However, intense fear can often inhibit the experience of pain, because during fearful episodes the brain secretes analgesic brain chemicals, such as the brain's own opioids, that temporarily

reduce the sensation of pain (Miczek, 1991). This is an adaptive mechanism that allows injured animals to ignore pain, increasing the likelihood that they might escape from predators. However, it can also cause the numbing that accompanies PTSD. There is some evidence that the blockade of opiate receptors can actually reduce such numbing and psychological dissociations, helping people with borderline personality disorders respond more positively to psychotherapy (Bohus et al., 1999). The same applies to PTSD (Pitman et al., 1990).

Although sudden pain is one kind of stimulus that can usually arouse the FEAR system, we have just seen that the system can also be easily aroused by stimuli that do not cause physical pain. The smell of a predator does not cause physical pain in a rat. Well-lit open spaces cause no bodily pain. Similarly if a human baby is not well supported physically, it may eventually fall and be hurt, but the seeming lack of support arouses fear long before the experience of any physical pain has occurred. Loud noises may be unpleasant, but they are rarely painful. Nevertheless, babies and most animals are afraid of thunderous or piercing sounds, because those "startle" stimuli have often heralded dangerous events in the evolutionary history of most mammalian species. Indeed, the startle response is amplified if animals are already anxious. It has been long known that the temperamental trait of anxiousness can be easily bred into animals by using behavioral-genetic selection procedures. Investigators are beginning to detail the brain changes that arise from such inherited temperaments (Harro, 2010; Harro et al., 2011; Kanarik et al., 2010; Singewald, 2007).

Physical pain is often used in fear-conditioning experiments because it is so easy to inflict on laboratory animals, most commonly through the application of electrical shocks. In fear-conditioning, animals learn to become afraid of *conditioned* (previously neutral) stimuli, such as an auditory tone or a light, when the presentation is paired with an *unconditional* stimulus, like an electrical shock, that always arouses the animal's FEAR system, as it does in humans. Quite rapidly, animals learn to fear the tone or the light even when it is not accompanied by the shock. In other words, cues that predict painful events always begin to generate fearful responses in practically all animals that have been studied. Such rapid development of fear responses to conditioned stimuli is the hallmark of successful fear-conditioning (see the next chapter).

Very few behavioral neuroscientists doing such work are willing to acknowledge, or even talk about, whether their animals experience anything awful. They claim, at times surely opportunistically, that such internal feelings cannot be directly observed and hence should be excluded from scientific discussions. However, this seems short-sighted. Indeed, if aversive feelings are a critical reason that animal brains learn to become behaviorally fearful, these scientists can never understand how the fear conditioning, in which they are so interested, really works. Scientists are supposed to go with the "weight of evidence" but in this arena that standard value of scientists seems to be neglected. This is why we have highlighted a *dual-aspect monism* strategy (Panksepp, 2005b, 2007a) that has the power to translate emotional feelings in animals to concrete psychological predictions in humans.

In addition to becoming afraid of the conditioned stimulus, rats easily become afraid of a variety of *contextual* (extraneous) stimuli that happen to be present during conditioning experiences. For example, rats readily learn to become afraid of tones that are paired with shocks, but they also become afraid of the walls of the conditioning chamber and perhaps of the unique smell of the sawdust used in those test cages. The rats may also become afraid of the sight, sound, and smell of the experimenter who puts them into the test chambers. These are all contextual stimuli that are also brought under the conditioning umbrella during systematic fear-conditioning experiments.

VARIETIES OF FEAR EXPERIMENTS AND THE CHEMISTRY OF FEAR

Researchers have been keen to learn about the chemistry of FEAR largely for psychiatric reasons. For example, many people who suffer from PTSD could be helped by a medication that alleviates their consuming feelings of fear. Four general types of experiments have been used to study the chemistry of FEAR. Each involves a means of inducing fear and a means of measuring the reduction of fearful behavior in response to particular drugs. It is assumed that any drug that diminishes the *latency* (duration) or intensity of fear responses in animals might also reduce fearful affect in humans. This experimental work has revealed that in most cases *benzodiazepines*[1] (BZs) quell most kinds of fear.

We will not examine the various experimental procedures that have been devised to study fear in animals in any detail here. If readers are interested, they can consult the previous detailed coverage of those issues (see Panksepp, 1998a, pp. 209–212) on which this condensed version is based. Understanding these methodologies is more important for people working in the field than for general readers. Some readers may, however, wish to pursue the details of the effects of various drugs in ameliorating fear in experiments involving *conditioned emotional responses* (CER), *potentiated startle responses*, and intrinsically scary environments such as *elevated mazes*, all of which take advantage of an animal's innate defensive behaviors. For example, one might place a probe with an electrical shock in a rat's cage. Sooner or later, in the course of its explorations, the rat will touch the probe, usually with its nose, and will receive an unwelcome shock. Typically, the rat will pile up the sawdust or other bedding in its cage in an effort to cover the probe. (Whether this is an instinctual defensive behavior in rats or one based on previous learning is not yet clear.) If, as a result of medication, a rat takes longer to build up a barrier over the offending probe, the medication is seen to reduce the rat's fear. Again, antianxiety agents such as BZs are effective in diminishing these and many other defensive behaviors. However, as we shall see, they are not especially effective in reducing separation-PANIC responses, which is only one of many lines of evidence indicating that it is a distinct negative emotional system that provokes a different kind of "anxiety" (see Chapter 9).

We will not engage in a thorough examination of the drug-related experimental research, but we would like to discuss two topics in some detail. The first is the way that the FEAR system influences the startle response. All animals exhibit a startle response to loud noises. The vigor of this response, however, can vary. If the FEAR system is already aroused, the startle reflex will be much stronger. For example, if you subject an experimentally naive rat to a loud noise, the rat will probably be moderately startled by that unconditioned stimulus. Suppose, however, that you had previously conditioned the rat to associate a light with a shock to the foot. That animal would have learned to be afraid of the light. If you exposed the conditioned rat to the light, thereby arousing a background level of arousal in the FEAR system, and then shortly thereafter exposed the rat to the same loud noise, its startle response would be far greater than it would be if it had not been trained to fear the light. This, incidentally, is why you can often

elicit a "*potentiated startle*," or an extreme reaction, when sneaking up on someone who is watching a scary movie. Because she was already afraid, the person's startle response is more vigorous than it would have been if she had been watching a comedy.

The neurological details of this "potentiation" have been worked out. Basically, the startle reflex itself is organized very low in the nervous system, as a very rapidly acting reflex, well below the FEAR circuitry. However, outputs of the FEAR circuitry do descend that far, and if FEAR has been aroused, it facilitates the intensity of that ancient reflex within the brain stem. This is an excellent way to see how the FEAR system potentiates a specific reflex. Similar procedures have been used in humans with a fear-potentiated blinking response evoked by a small puff of air applied to the eye (Davis & Lang, 2003).

The second topic on which we will briefly dwell concerns a number of conceptual and methodological problems associated with some of these drug experiments. These problems have generated some confusion about the efficacy of certain drugs. It is a mistake to believe that any chemical capable of reducing apparent fearful *behavior* necessarily decreases fearful *affect*. Suppose that a lever in an animal's cage delivered a painful electric shock. After the first or second shock, the animal would avoid touching the bar. If you gave the animal a drug that induced amnesia, it would no longer avoid the bar because it would forget that the bar was the source of pain. So this drug would increase punished behavior, but it would not do so by decreasing the animal's affective distress after each shock. This is one instance where the animal's willingness to engage in punished behaviors does not reflect a reduction in fear. Other drugs may simply disinhibit animals, so they are more active and willing to do many more things. Such drugs may also increase random lever pressing. When one tests a particular drug that increases an animal's willingness to engage in punished behaviors, investigators always need to consider that the drug might affect brain processes other than anxiety reduction. Such qualifications apply to the study of all of the other emotional systems. Such are the dangers of excluding affective feelings from scientific discourse.

One area where general disinhibition was mistaken for attenuation of fear occurred in serotonin research. Between the 1950s and the 1970s, some scientists were persuaded that increased serotonin activity in the brain was responsible for anxiety. This conclusion was reached because serotonin

receptor antagonists, which reduced serotonin activity in the brain, caused animals to engage in more punished behaviors, like pressing a bar that delivers food, even when a CER stimulus is presented that predicts a forthcoming foot shock. So these researchers believed that a decrease in serotonin surely reduced anxiety and they concluded that high levels of serotonin cause people and animals to feel anxious.

It is now clear, however, that a reduction of serotonin in the brain makes animals more manic and impulsive in general. Serotonin acts globally through most of the brain. Animals whose serotonin has been reduced tend to be disinhibited in a broad range of circumstances, and they will tend to overrespond in anxiety causing situations because of their impulsivity rather than because of any real decrease in anxiety. In fact, serotonin-depleted animals are prone to become more anxious than normal ones and they are generally hyperemotional in all realms; for instance, they tend to show much more aggression than normal and are often hypersexual. Accordingly, increased behavior in the face of punishment could simply reflect a generalized release, or a disinhibition, of active behavioral tendencies, not a reduction of anxious feelings.

Although serotonin modulates the intensity of anxiety, it does that to no greater extent than it modulates other negative emotions. Serotonin regulates the intensity of *all* emotions. Elevated brain serotonin activity generally inhibits emotions, including fear, while less serotonin arouses emotion, including fear. Thus when serotonin-deprived animals exhibited increases in punished behaviors, it was because all their emotions were aroused and they were overactive. There is presently little empirical reason to believe that global elevation of serotonin activity in the brain plays a major part in promoting the experiences of anxiety or fear. Currently, most of the available data are more consistent with the alternate conclusion, namely that an overall *increase* of serotonin activity in the synapses between neurons decreases anxiety and produces feelings of relaxation—serotonin can dampen every emotional and motivational urge in the brain. This is why selective serotonin reuptake inhibitor (SSRI) antidepressants, which increase the availability of serotonin in synapses, are quite effective in relaxing overstressed people and making them less irritable (Knutson, Wolkowitz et al., 1998).

However, during the past few decades a horde of serotonin receptor types have been identified (15 are presently known) and it looks as if one or two

of these receptors may actually promote negative feelings of some sort. Still, how precisely different serotonin receptors participate in the generation of relaxation as well as in promoting a negative affect is by no means well understood. For instance, a comparatively new antianxiety drug, buspirone (its brand name is BuSpar) is known to operate on serotonin receptors that can both increase and diminish brain serotonin activity, depending on the placement of the receptors in regards to the synapse. Initially, buspirone was thought to reduce anxiety by reducing serotonin release from presynaptic terminals, but it now seems more likely that it is doing so by increasing serotonin activity at one type of postsynaptic receptor (for details, see Panksepp, 2004, p. 501). Thus, even though it is evident that global facilitation of serotonin activity reduces anxiety, there is still much to learn about the effects of serotonin at individual receptors.

In sum, there are many ways to monitor fearfulness in animals—from timid behaviors in large test arenas called "open fields" to "social interaction tests" to "elevated plus mazes" to "contextual freezing." How these environments and behaviors all connect up to the FEAR system remains uncertain. Overall though, at present, there is an enormous amount of work on fear-learning (as described in the next chapter) and very little work on the evolutionarily provided FEAR circuit of the brain. So, while neuroscientists know a lot about the neurochemistries (e.g., glutamate synapses) that allow conditioned stimuli access to the FEAR system, they know comparatively little about the way that the FEAR system itself works. Still, during the past few decades, a few groups in Brazil have been intensively studying the various neurochemistries in the PAG that regulate defensive behaviors, and in our terminology the FEAR system (e.g., Brandão, et al., 2003, 2008; Del-Ben & Graeff, 2009). Some of these details are difficult to summarize succinctly, but readers should be assured that the neurochemical understanding of this system will offer many possibilities for medicinal developments, including simple maneuvers such as reducing inflammatory cascades in the circuits that mediate a form of anxiety, perhaps feelings akin to social separation distress (see Chapter 9) that are precipitated by morphine withdrawal following addiction (e.g., Hao et al., 2010).

VARIETIES OF ANXIETY IN THE MINDBRAIN

Not every form of anxiety emerges from the FEAR system. We use the word "anxiety" in several contexts, but we now know that "separation anxiety" is a very different kind of process in the brain than the various emotional trepidations we have described in this chapter so far. It is important for psychotherapists and scientific psychiatric experts to recognize that there are several distinct negative emotion systems in the brain and that more than one may be aroused at any given time. How these systems interact is still unknown. Successful therapy, however, may well rely on an understanding of which system is dominant in each patient. For example, the PANIC/GRIEF system, detailed in Chapter 9, is probably more important for the often intense feelings of social insecurity and loss that people have when experiencing "panic attacks" than for the anticipatory anxiety that occurs in response to scary nonsocial events.

We will devote an entire chapter to the PANIC/GRIEF system, but to expand on a comment made earlier, we note that there are two good reasons to distinguish the PANIC/GRIEF system from the FEAR system. First, they are supported by different brain structures and are therefore anatomically different. Second, the FEAR and PANIC/GRIEF systems are controlled to some extent by different brain chemistries and have different reactions to drugs. As we have seen, BZs are generally effective in quelling FEAR, but they have little effect in eliminating the cries of distress that young animals make when they are separated from their parents. The original BZs (Librium and Valium) also had little effect in quelling panic attacks in humans, even though some of the modern high-potency BZs, such as alprazolam, are quite effective. On the other hand, the original tricyclic antidepressant imipramine can, at low doses, ameliorate panic disorder. Indeed, imipramine was the first drug discovered to have clear antipanic effects in people and also to reduce separation cries in animals (Klein & Rabkin, 1981; J. Scott, 1974).

One can also distinguish between PANIC/GRIEF and FEAR on clinical grounds because they mobilize different autonomic responses. There are two major branches in the autonomic nervous system. The *sympathetic* branch readies an animal for an active response. So, for example, the sympathetic nervous system may elevate heart rate and respiration, thereby providing oxygen for burning elevated levels of blood sugar that are necessary for taking flight. It may likewise dilate pupils in order to increase vigilance. The *parasympathetic* branch, on the other hand, takes over when

animals are in a more passive state. Under the influence of the parasympathetic nervous system, the heart rate slows, breathing is regular, and pupils remain undilated. The parasympathetic nervous system is also sensitive to emotional changes, and it promotes tears, salivation, and sexual arousal.

Anticipatory anxiety (conditioned FEAR) is characterized by generalized apprehensive tension, with a tendency toward various symptoms stemming from the sympathetic arm of the autonomic nervous system. So symptoms like a rapid heartbeat, sweating, gastrointestinal upset, and increased muscle tension characterize FEAR. Manifestations of PANIC/GRIEF, however, are accompanied by feelings of weakness and depressive lassitude, with more autonomic symptoms of a parasympathetic nature, such as a strong urge to cry, often accompanied by tightness in the chest and the feeling of having a lump in the throat. While FEAR beckons one to escape from situations that intensify anxiety, PANIC/GRIEF prompts thoughts about lost objects of affection and impels one to seek the company of the people one loves.

Although there are distinct emotion systems, each characterized by specific affects and behaviors, they frequently interact in complicated ways. There is an abundant psychotherapeutic literature on attachment disorders, which are manifestations of the PANIC/GRIEF system. Children with severe attachment disorders are unable to trust, will reject feelings of dependency in themselves, and cannot empathize with others. They are apt to be needy, greedy, and inappropriately demanding, often turning to drugs, especially opiates and alcohol, in adolescence and adulthood.

People with attachment disorders also frequently suffer from persistent fears, stemming from childhood experiences of neglect or abuse. It is this complex picture that one often sees when examining the histories of young people in custodial penal institutions. Such children grow up to be highly aggressive and are often antisocial. At the same time, they often suffer from a sense of hopelessness about themselves. Clearly they have problems with several basic emotional systems. The complexity of their emotional needs and limitations render them difficult to rehabilitate. A full understanding of the brain emotional systems involved in these behaviors is vitally necessary for the development of therapeutic techniques and effective medications to treat both the persistent fears and the attachment disorders of these unfortunate young people.

Post-traumatic Stress Disorder (PTSD) is another complex condition that involves several different emotion systems. In addition to chronically overactive manifestations of the FEAR and PANIC/GRIEF systems, PTSD is a state of terror that is often accompanied by anger, which we mentioned in our opening vignette as a possible aftermath of repeated trauma (as soldiers experience during wartime). An aspect of PTSD, distinct from straightforward PANIC/GRIEF or FEAR, lies in the fact that PTSD can be diminished with antiseizure medications such as carbamazepine, an agent that is not consistently effective in the control of either panic attacks or anticipatory anxiety (Berlin, 2007). This suggests that there is an additional seizure-type process that can elaborate several negative emotions toward a full-blown PTSD state (Agrawal et al., 2006). Although PTSD has not yet been unambiguously linked to an emotional anxiety-type system, like FEAR or PANIC/GRIEF, it appears to be another way that the brain can be traumatized, probably with several emotional systems participating, such as both FEAR and PANIC/GRIEF as well as RAGE.

Indeed, the vicissitudes of life being what they are, with each of us bombarded by a diverse set of emotional challenges, it will be next to impossible to prove that any emotional disorder is due simply to a single emotional system, not to mention a single chemical imbalance. Most people will reflect several emotional imbalances, explaining why the concept of "comorbidity" is so common in psychiatry. This essentially means that more than one psychiatric syndrome occurs at the same time. Take depression, which is often accompanied by excessive psychological pain, anxiety, angry irritability, as well as diminished urges to seek and pursue other life interests. Indeed, the term "depression" is very ambiguous, implying both generalized malaise and sickness. A more accurate description would need to address the emotional systems involved and the ways that their over- or underarousal contribute to the clinical symptoms, including the increasing possibility that inflammatory cascades that characterize many types of sickness are overactive in depressed individuals (Dantzer et al., 2008). We suspect that scientific psychiatrists, at some time in the future, may have little need for the diagnostic categories presently used, as we begin to understand emotional problems in terms of better descriptions of imbalanced brain emotional systems and an understanding of the many neurochemical changes that can lead to affective distress.

We are just beginning to understand the massive complexities of the underlying neuroanatomies and neurochemistries. A future biological psychiatry that works well along more specific affective psychotherapeutic interventions will probably be based on knowledge that more readily links to the actual emotional experiences of patients. One reason this is not happening as rapidly as it could (and perhaps should) is because many investigators still believe that psychology is a soft science and that it is better to link psychiatric diagnostic categories directly to changes in brain facts, with no intervening emotional analysis. The existence of distinct emotional systems in the brain may facilitate a more comprehensive psychobiological approach than currently exists (Panksepp, 2004, 2006a, 2009a, 2009b).

The Fear Chemistries in the BrainMind

Until the middle of the last century, the only drugs available for the treatment of fear were opioids, alcohol, barbiturates, and meprobamate (the last, known as Miltown, was once very popular, but it has dropped completely from therapeutic practice because people who took it were prone to commit suicide). These early drugs had many drawbacks, the worst of which was the poor safety margin commonly leading to accidental overdoses or suicide.

Because anxiety is often accompanied by autonomic arousal, including increased heart rate and blood pressure, one strategy that has been useful is treatment with drugs that reduce the action of brain and body arousal chemicals known as *endogenous catecholamines*—epinephrine (adrenalin) and norepinephrine (noradrenaline), in particular. These brain chemicals, in a group called *biogenic amines*, activate the sympathetic nervous system that goes into hyperdrive during intense emotional arousals—the "fight or flight" responses that conflate RAGE and FEAR mechanisms of the brain. In any case, blocking their activity exerts a calming effect. Beta blockers (which inhibit one type of norepinephrine receptor) are helpful in the symptomatic control of anxiety, such as palpitations and sweating. Indeed, long-approved drugs such as propranolol are sometimes used to inhibit anxiety during public presentations or performances. It is not uncommon for artistic performers and public speakers to take this agent to minimize the "nerves" that can hinder peak performances.

The more specific treatment of anxiety was revolutionized by the serendipitous discovery of the drug chlordiazepoxide (CDP). The efficacy of CDP was identified in 1960 during the final phase of research, just prior to the scheduled termination of a relatively unfruitful research program on BZs at Hoffman-LaRoche Laboratories. Almost as a last resort it was found that one of the BZ molecules, CDP, was very effective in taming wild animals at a local zoo. CDP was soon marketed under the trade name Librium, and it became a great success in controlling many anxiety disorders. It could reduce anxiety at much less than a hundredth of the lethal dose. Soon many more potent BZ drugs such as diazepam (Valium) became available, with many more to follow. These have been best-sellers for decades.

The mild sedative effects that are commonly observed at the beginning of BZ therapy tend to abate rapidly, while antianxiety effects are sustained during long-term use. Initially, these drugs seemed to produce no apparent physical dependence when used occasionally. However, it soon became common practice for anxious patients to take higher and higher doses of BZs over long periods of time. In patients who had become dependent on these drugs, withdrawal could produce a syndrome resembling delirium tremens (DTs), the confused, agitated, hallucinatory state that often accompanies alcohol withdrawal. For such reasons, certain BZs have fallen into disfavor in the medical community, while at the same time they have become a very useful treatment for those wishing to get off alcohol.

For a long while neuroscientists and psychiatrists did not know why BZs were effective in treating anxiety. Only when the BZ receptor was discovered in 1979 could this research be carried out. Usually when external agents like BZs exert an effect on the brain, one expects to find similar endogenous brain chemicals that are naturally secreted by the brain. For example, the PANIC/GRIEF system can be calmed by the administration of opiates, and the brain produces similar chemicals in the form of endogenous opioids. Neuroscientists assumed that the brain produced a BZ-like endogenous chemical that would bind with BZ receptors, producing a calming effect. But the situation does not appear to be so straightforward. When researchers administered a BZ receptor antagonist, they expected that anxiety would increase, but this did not happen. BZ antagonists had no effect in either augmenting or decreasing anxiety. They were essentially psychologically neutral.

Researchers subsequently discovered that BZs do not independently reduce anxiety. Instead of acting alone in calming the FEAR system, BZs act by enhancing the effect of gamma-aminobutyric acid (GABA), a neurotransmitter that inhibits the activity of neurons, reducing their rate of firing. BZs have their own binding site on the A type receptor for GABA. BZs, in combination with GABA, will slow down the activity of the FEAR system (along with various other affective systems). This enhanced effect of GABA transmission is what keeps people and animals in the state of placid serenity characteristic of treatment with BZs. Researchers subsequently discovered that other older antianxiety agents, including alcohol and barbiturates, also quell anxiety by promoting GABA-mediated inhibition in the brain. In effect, the GABA receptor can be envisioned as a lock with multiple key holes into which different keys can be inserted simultaneously. Each added key enhances the effect of the primary key, GABA. Many who have found relief in the compounded inhibitory effects of alcohol along with other drugs at the GABA receptors have, unfortunately, lost their lives in the bargain. The combination can reduce many bodily functions.

BZ receptors are concentrated along the trajectory of the FEAR system from the central amygdala down to the PAG, and even farther to the *nucleus reticularis pontis caudalis*, where fear modulates the startle reflex (M. Davis, 1992). BZ receptors are also found in many areas of the neocortex, and this may be why they are effective in reducing upsetting ideation. So BZs are effective in diminishing the activity of all levels of fearful anxiety, from the startle response to distressing thoughts.

Although neuroscientists discovered how BZs work in conjunction with GABA secretion, the search for an endogenous BZ molecule has not been straightforward. Researchers have not as yet been able definitively to identify a brain chemical that performs this same function, but there have been and still are many candidates. Furthermore most researchers agree that if there is an endogenous chemical that binds with BZ receptors, it probably does not perform the same role that BZs do—it does not enhance the inhibitory effects of GABA. Most researchers believe that this endogenous chemical acts rather as an *inverse agonist* at BZ-binding sites, acting on GABA receptors in a way that reduces rather than enhances the inhibitory effects of GABA. The results of reduced GABA inhibition would include elevated activity of the FEAR system, making animals more anxious. A key candidate for this role as an endogenous inverse agonist for BZ receptors

has been *diazepam binding inhibitor (DBI)*, a neuropeptide that appears to promote anxiety when it binds to BZ-binding sites at GABA receptors. But despite years of work, there is still no conclusive evidence that DBI is in fact a commanding anxiety-generating transmitter of the brain (Möhler, 2011). Other neuropeptides such as corticotropin releasing factor (CRF) have much more evidence for being powerful anxiety- and stress-promoting systems in the brain.

At present, various neuropeptides are promising targets for specific pharmacological control of subtypes of anxiety. When administered in the brain, a number of neuropeptides arouse the FEAR system. For example, CRF causes agitated arousal while reducing a variety of positively motivated behaviors: feeding, sexuality, grooming, play, and so on. Animals also tend to freeze in environments where they previously received CRF, indicating that these environments contain a number of contextual stimuli that the animal has learned to fear. Conversely, freezing that is induced by the administration of a shock to the foot is diminished by CRF receptor antagonists. However, it may not be feasible to use a CRF antagonist to treat pathological fear because usually CRF is also a useful hormone that travels through the bloodstream, leading the brain and body to effectively respond to stress and danger. For instance, such drugs may diminish immune defenses and worsen bodily disorders such as irritable bowel syndrome (Stengel & Taché, 2010). In any event, at present, CRF antagonists are mainly being targeted as potential treatments for depression, a disorder that is not uniformly controlled by existing antidepressants. Even though CRF antagonists have been clinically effective, problematic side effects have been observed, such as liver toxicity.

In addition to CRF and the catecholamines, a number of other neuropeptides can activate the FEAR system. The neuropeptide *alpha-MSH* promotes camouflage-type pigmentary changes in many fish and reptiles. When these animals are scared, their skin tends to turn black. Although this peptide does not control skin pigmentation in higher vertebrates, a vigorous freezing/hiding pattern can be evoked in chicks by the administration of this peptide into the brain. *Adrenocorticotrophic hormone (ACTH)*, which comes from the same segment of the same gene that creates alpha-MSH, has similar effects. Injection of ACTH can precipitate vigorous flight, as well as freezing in rats and other animals. An especially well-studied peptide is *cholecystokinin (CCK)*, which can precipitate a broad range of

anxiety symptoms emanating from both the FEAR and PANIC/GRIEF systems. *Neuropeptide Y (NPY)* also seems to be able to calm the FEAR system because NPY antagonists can evoke anxiety in animal models (Panksepp & Harro, 2004). If such findings are supported by further research, an especially useful category of drugs may result.

The brain contains a number of other chemicals that activate the FEAR system. An excitatory neurotransmitter, glutamate, is key to the transmission of unconditional FEAR signals, such as a rat's innate emotional aversion to the odor of cats. Glutamate also controls the unconditioned FEAR response. If one administers glutamate agonists into medial brain-stem regions where FEAR circuits are concentrated, animals begin to exhibit spontaneous bouts of flight (often while in semicrouched postures) accompanied by apparent psychic anguish. Visually oriented animals such as birds exhibit rapid head scanning, persistent vocalization, and bulging eyes suggestive of profound terror. These episodes can be inhibited by glutamate receptor antagonists. However, since glutamate receptors are widespread in the brain, controlling learning and much of our higher cognitive mind, it is unlikely that its direct pharmacological manipulation would yield a useful antianxiety agent. Nevertheless, milder stimulation through a glycine receptor "side-knob" on glutamate receptors may be a very safe and useful treatment for both anxiety and depression. Such drugs are currently undergoing development and clinical testing (Burgdorf et al., 2011).

A FEW SPECULATIVE DEVELOPMENTAL THOUGHTS ABOUT FEAR AND THE AMYGDALA

Since 1939, it has been known that extensive damage to the temporal lobe yields dramatic fear deficits, known as the Kluver-Bucy syndrome. This was followed by the localization of many of the deficits to the amygdala (Rosvold et al., 1954), which sits at the center of the temporal pole (Figure 1.1). Thus, it is commonly believed that the amygdala lies at the hub of the FEAR system. The amygdala consists of about a dozen nuclei or sections, several of which, known as the *basolateral amygdaloid* (BLA) complex are involved in fear-conditioning (LeDoux, 2000; M. Davis, 1992; Maren & Quirk, 2004). This has been reinforced by the fact that amygdala arousal is seen in practically every brain-imaging study that has anything to do with

anxiety or negative emotions (and occasionally positive emotions too). As we shall discuss in more detail in the next chapter, which is devoted to learning and memory, the BLA serves as a conduit for relaying fear cues into the *central nucleus* of the amygdala (LeDoux, 2000). The central nucleus is at the very top of the intrinsic, primordial FEAR system, but nuclei in the BLA are not part of that primary-process emotional system. So, even though the BLA nuclei in the amygdala play a crucial role in classical conditioning, their important role seems to lie in the ability to conduct information into the FEAR system rather than in the ability to generate fear by themselves. Therefore, while the central nucleus of the amygdala is part of the unconditional (instinctual) FEAR system, the other nuclei are not.

So is the central nucleus of the amygdala the heart of the FEAR system? Given that the FEAR system also consists of many deeper structures that evolved long before the amygdala, it is unlikely that the central nucleus is the most important part of this system. Indeed, humans who have the condition known as Urbach-Wiethe disease, wherein parts of the amygdaloid nuclei on both sides of the brain, especially the basolateral complex, slowly degenerate completely, still have abundant internal worries and rich emotional lives. Although people with deficits in these amygdaloid nuclei have commonly been reported to be deficient in detecting the static fearful faces that are commonly used in brain-imaging experiments, as investigators are looking more closely at the fear deficits, the results are not as clear as early studies had suggested (e.g., Talmi et al., 2010; Wiest et al., 2006). Similarly, brain imaging done on individuals with PTSD, a learned type of fear, often finds stronger than normal arousal of the amygdala, but sometimes it does not (Lanius et al., 2005). This too suggests that fear can emanate from brain areas other than from the amygdala. There is also the fact that young animals that have been surgically deprived of all the neurons within their amygdalae, while leaving the fiber pathways that course through the area intact, are still able to exhibit fear and anxious temperaments, which probably are created by deeper structures in the FEAR system (Amaral et al., 1992; Kalin et al., 2001). Thus, it does seem likely that during development, the fearful capacities of many regions of the upper brain are programmed by lower brain regions.

With respect to the programming of fear in higher brain regions, investigators need to consider that early in life even the amygdala and

related temporal lobe structures may all need to be programmed by deeper structures in the FEAR system, such as the PAG and the hypothalamus, which only then allow higher brain systems to better evaluate fearful stimuli and situations. Likewise, learned anxieties in adult animals may be critically dependent on the influences of lower structures, for example, the amygdala influencing frontal and cingulate cortices. However, at present, these possibilities are largely speculative.

In any event, contrary to abundant press reports, encouraged by scientists working on fear-learning, the amygdala is not absolutely essential for the creation of anxious feelings. In contrast, the PAG and hypothalamus surely are. That is because the FEAR system, just like the RAGE system (Figure 4.1), is hierarchically organized, where the higher emotional functions, like those emanating from the central amygdala, are completely dependent on the lower brain functions (e.g., the hypothalamus, whose emotional functioning is dependent on an intact PAG).

If fear can be generated by structures deeper than the amygdala, and if the amygdala is not essential for the instinctual generation of FEAR in the very young, then even the amygdala may obtain much of its fear-generating capacities because it is *programmed* (taught) by lower structures in the FEAR system. This kind of programming of higher brain regions, such as the amygdala, by lower brain structures is increasingly well established for other emotional systems, especially the SEEKING system. And we will focus on this major concern in the next chapter, because it has been too neglected by fear conditioners. This bottom-up control of learning probably also applies to emotional learning in the cortex, even though there is not abundant data on such issues. In any event, the thesis here is that deeper parts of the emotional brain teach the cortical structures to perform a variety of cognitive strategies related to emotion regulation.

A famous example of such programming in the cognitive-perceptual realm was elucidated by the Nobel laureates David Hubel and Torsten Wiesel (1979), who were the first to demonstrate that neurons in the visual cortex are programmed by the retina to discriminate between specific types of visual information like the orientations of lines and edges, and their movement in specific directions. These highly tuned sensitivities are thought to constitute the basic neuronal grammar of vision, which has to be developmentally programmed and learned by the neocortex. Indeed, it is now known that the visual cortex is not intrinsically programmed by the

genes, but rather by the typical projection of thalamic visual pathways into those higher brain regions that become the visual cortex. If that region of cortex is destroyed in fetal mice before birth, they develop a fine visual cortex in nearby neocortical regions that would normally serve to process touch (Sur & Rubinstein, 2005). In fact, if normal human adults are not allowed to see for a week, and are taught to read Braille, their visual systems begin to respecialize for the fine discrimination of touch (Elbert & Rockstroh, 2004). This means that many cortical functions can remain flexible for a lifetime and can adapt to other skilled processes when the ones they typically mediate are no longer needed.

Thus, all areas of the neocortex tend to acquire their functions through early conditioning, which again highlights the importance of education, as well as the recognition that the tertiary-process involvement of emotions in higher brain regions is largely elaborated through learning. One might take the position that the earlier that good emotional habits are established in children, the better off their minds will be, although this surely remains a highly debatable concept, since there is rather little good empirical work on the topic. However, the ever-growing body of research that demonstrates the flexibility of the BrainMind across the life span certainly gives us increasing hope that early learning is final only in specific instances. In many aspects of life, healthful maturation through learning and adaptive processes may be viable at any age, especially in children, who can be remarkably resilient in the face of adversities.

However, certain brain systems do rapidly lose their early capacity to fully take on the functions we normally expect to see in human beings. Again, if we take the visual system as the best-studied example, there is a window of opportunity for programming of the visual cortex. This programming must occur early in life. If the visual cortex has not been programmed before this window closes, the visual cortex will never function normally, and animals will remain visually impaired, even blind, for the rest of their lives. Biological windows of opportunity are also not uncommon in the development of higher neuroemotional processes. We have learned to manipulate some of these processes.

For example, postpartum female herd animals, like sheep, have a short window of opportunity for bonding with their young. If the mother has not had access to her lamb within 2–4 hours following birth, she will reject it. Normally, during the bonding window, the mother learns to recognize the

scent of her own lamb and to single it out for preferential treatment over other lambs. However, if the bonding opportunity is missed, the window can be opened once more for a short period through the manipulation of brain chemistry, either through direct administration of maternal neurochemicals (i.e., infusion of oxytocin into the brain) or by way of physical and/or social interventions that achieve the same desired chemical and emotional outcomes (these topics will be addressed in more detail in Chapter 8 when we come to the CARE system). An important question in the realm of emotional learning, adaptation, and maturation, which we will also address in later chapters devoted to fundamental social processes (LUST, CARE, PANIC/GRIEF, and PLAY), will be the extent to which we can develop social structures that promote the development of prosocial networks in higher regions of human brains. One could imagine that these positive social forces would be able to very substantially counter the influences of FEAR.

EXAMPLES OF FEAR IN CHILD CLINICAL SITUATIONS

In real-life situations, we often see how the primary affective and tertiary cognitive processes blend in apparently seamless layers of influence and counterinfluence. In the case of FEAR, the expressions can be subtle and vast. It is very hard to decipher what is going on, especially in children's minds as they try to integrate the many affective forces that are guiding their development.

It is worth noting that one runs into conundrums about the expression of fear in a clinical situation, especially when parents report that their children do not seem to be afraid of anything and that they put themselves in dangerous situations without a second thought. Sometimes parents suppose that their children really can experience no fear; however, this is not likely in children whose brains are intact, with the full complement of PAG, hypothalamic and amygdala circuitries. Current brain evidence, some presented in the previous section, suggests that only damage to the lower core of the FEAR systems of the brain can render a person truly "fearless." Thus, children who appear to be fearless may in fact be quite fearful in their lower brain regions, but their maturing higher brain regions have not yet integrated those messages, and the neocortex can exert inhibitory control

over lower brain functions. And in childhood, it is not preordained that the top and bottom of the mind—the tertiary and primary BrainMind processes—will work well together.

Indeed, apparently fearless children are often preoccupied with internal anxieties and ways to avoid such negative feelings. One such fearless 6-year-old, who had recently seen the film *Jaws*, spoke about sharks that can grow teeth back and about starfish that can grow back an "arm." He laughed loudly, saying that people can do that too, and he then picked up a paintbrush, brandishing it like a sword at an invisible enemy. Once or twice in the course of this imaginary duel, he said, "I'm not afraid of you!" It seems likely he was actually very afraid at some level of his brain, but his willful shows of aggression made him feel a bit better.

Children, of course, sometimes put themselves in dangerous situations, such as climbing high ladders or running across busy streets without looking. This is usually because they just haven't really understood that particular danger yet. Some children, though, deliberately endanger themselves in order to frighten and punish their parents. In these cases, it is not that the FEAR system isn't working. Instead, another emotion system, perhaps RAGE, is holding sway. In contrast, perhaps the PANIC/GRIEF system is at work when children who suffer neglect endanger themselves in an effort to win the love and attention that they cannot get any other way. Worse yet, when children are abused, they sometimes engage in dangerous activities because they have concluded that they are naughty and deserve to be punished. In a sense they are punishing themselves in the ways that their parents could punish them. These apparently fearless behaviors are really an effort to integrate and perhaps accept the demands of abusive parents, and thereby win their love. This too may be a distorted expression of the PANIC/GRIEF system.

There is also a close relationship between RAGE and FEAR that one encounters in human psychology. The two systems are closely intertwined, which accounts for the intimate dovetailing of fight and flight responses. The two systems are anatomically and chemically intertwined but also distinct, so they often work in tandem. The ascendancy of one system over the other depends on the kind of danger in the environment. For example, if it is possible to avoid danger, FEAR may predominate and an animal will freeze in the hopes of being overlooked. Otherwise, if danger is too close, too imminent, the animal will run for its life. If, however, the predator is not

so powerful (if it can be successfully attacked) or if no escape is possible, the RAGE system will come to the fore. Then the intended prey will assault its attacker, hoping to inflict an injury or create a diversion that will allow it to flee.

These two systems can often be difficult to distinguish in the clinical setting, especially when working with young children, who see and interact with the world in very different ways than do adults. When a child throws a tantrum, she may be furiously angry. Alternatively she might be terrified. If you think back to the example of encountering a large and dangerous predator, your terrified screaming and running about might have frightened it off. Fear behaviors are often not so different from enraged behaviors. When you are furious, you yell, shake your fist, and perhaps pace about. If you are terrorized, you are bound to exhibit slightly different but equally energetic acting-out behaviors. Adults are rarely terrorized. But a child's life is not so emotionally tranquil. It is often difficult to tell whether a child is very angry or very frightened. Only when the child calms down enough to speak about his or her feelings, can the truth be discovered, but this will take patient and understanding communications.

Take the case of the 4-year-old girl who was uncharacteristically reluctant to go into a therapy room. Once in the room she began to throw toys around, shouting unprovoked words of protest and abuse at the therapist. She appeared to be expressing great anger but the reason why was unclear. Eventually her therapist was able to coax an explanation from her. It so happened that the night before, the girl's teenaged brother had babysat and had allowed her to play a frightening video game featuring a villain who had worn dark, wraparound sunglasses. Then, just prior to the therapy session, the therapist had encountered his young patient with her mother in the car park. It was a sunny day and the therapist had been wearing sunglasses that happened to be similar to those worn by last night's villain. The little girl was frightened all over again and as her fear went out of control, she threw a tantrum. Probably her tantrum expressed both rage and fear. She was angry with her therapist for scaring her when he was the one who had promised to help her. However, her predominant affect was fear, which only appeared to be rage on the surface.

They say that attack is the best form of defense, however. And expressions of RAGE, when modulated, can sometimes have a positive effect in allowing children to overcome their fears. A 2-year-old girl had

been frightened by a rambunctious puppy and thereafter was afraid of all dogs, especially when she heard them barking at night when she was trying to go to sleep. Her father helped her cope with her fear by using PLAY to counteract the negative emotions. He sat with her one night and when the dogs barked, he waved his hand in a disparaging way, saying that "the doggies are stupid" and adding in a loud voice, "Be quiet, you stupid doggies! We don't like you!" Then he laughed conspiratorially, telling her that the doggies were so silly because they could not understand and kept barking. "Do we care about those doggies?" he asked, shaking his head. She shook her head. He said, "Do you know what I think about doggies?" His little daughter shook her head. He made a raspberry sound with his mouth, which made his daughter erupt in peals of laughter. After a while, the little girl joined in the game, saying that doggies were stupid and that they should "be quiet," making a raspberry sound of her own. After a few days of playing with her dad, she began to play the game alone in her cot. When she heard barking, she shouted, "Be quiet, stupid doggies!" She followed this with a vehement raspberry. When she played this game with her father, it seemed to be a lighthearted activity, a cause for mirth. But when she shouted alone at night, she seemed genuinely angry. At first this was a nightly ritual, but after a few weeks it was intermittent and finally disappeared. In this way she used an understated expression of anger to overcome her fear. In due course, her fear of dogs disappeared and she was able to pet a neighbor's dog without any signs of distress.

Of course, the use of neuroscience data in this way is dependent completely on clinical hunches. There is no way to really know which systems are active in children's brains, except through an accurate reading of their instinctual displays. This goes to show that one can only estimate the operations of children's primary-process emotional systems. Understanding basic emotional systems becomes increasingly difficult with adults.

SUMMARY

Abundant evidence indicates that circuits in primitive parts of the brain generate fearful states—states that evolved long before our more sophisticated cognitive abilities. Although we have learned an enormous amount about how fear-conditioning, the learned linkage of fearfulness to

world events, is generated (see the next chapter), the study of the FEAR system itself has been comparatively neglected in the Anglo-American research tradition, but not in other laboratories in the world, especially those in Saõ Paulo, Brazil (e.g., Brandão et al., 2008). Thus, there is an enormous amount of detailed affective neuroscience work that needs to be done before we will have a complete picture of this, as well as of all the other primal emotional systems.

Still, many thoughtful observers down through the ages have acknowledged the existence of this primitive state of fearfulness. And that is why we chose the epigraph for this chapter from Jack London's *White Fang*. The young wolf had never "encountered anything of which to be afraid. Yet fear was in him. It had come down to him from a remote ancestry through a thousand lives. It was a heritage he had received directly . . . through all the generations of wolves that had gone before" (p. 52). This fictional portrayal contains more than a few grains of truth for human beings as well.

Once we scientifically understand this kind of "FEAR itself" more thoroughly, we will be able to reverse many intrinsic and learned vexations of the human spirit, from chronic anxiety disorders to PTSD. Because we share such ancestral emotions, animal brain research can finally help clarify the deep nature of our own anxieties and how we come to experience fear in our interactions with the world. The next chapter will delve into the neuroscience of the kinds of emotional memories that brains create, starting with simple subcortical learning, and proceeding to higher cortical participation. There are many ways to mold the FEAR system into the dynamically flexible terror that it can become. As we have seen, there are also many ways to tame it, from pharmacological influences on the primary-process affective energies of the system to the tertiary-process cognitive regulations that can be maximized through judicious prosocial and psychotherapeutic interventions. As President Roosevelt put it so poignantly, "The only thing we have to fear is fear itself." So when we finally scientifically understand the FEAR system, we will know, more exactly, what he was talking about.

CHAPTER 6
Beyond Instincts

Learning and the Affective Foundations of Memory

I'm truly sorry man's dominion
Has broken Nature's social union
An' justifies that ill opinion
Which makes thee startle
At me, thy poor, earth-born companion
An' fellow mortal!

Still thou art blest, compar'd wi' me!
the present only toucheth thee
but och! I backward cast my e'e
on prospects drear
An' forward, tho' I canna see
I guess an' fear.

—Robert Burns, "To a Mouse" (1785)

IN HIS EIGHT-VERSE POEM (the second and last verses shared above) the Scottish poet Robert Burns highlighted the continuity of fears between mice and men. While the mouse usually experiences FEAR in the present moment, in response to distinct environmental challenges, our capacity to look forward and backward in our mind's eye can create phantoms of the imagination (Figure 5.1). Through our autobiographical memories, we humans and perhaps some other animals have the capacity for subjective

time travel within the affectively laden texture of remembrances that are rich with personal meanings. As the Nobelist Eric Kandel, who studied the neurology of fear-type learning (classical-conditioning of pain) in sea snails stated, "For all of us explicit memory makes it possible to leap across space and time and conjure up events and emotional states that have vanished into the past, yet somehow continue to live on in our minds" (2007, p. 281). But memories are not always explicit. Some are implicit, cognitively unconscious but still affectively capable of influencing behavior.

Many emotional memories in humans surely arise without awareness of their causes, but that does not mean their accompanying affects are not experienced. Indeed, although the cognitive reasons for changing feelings may typically be unconscious (perhaps retrievable with psychoanalysis), the feelings themselves are not. Since affect is a form of phenomenal consciousness, experienced feelings should not be deemed to be unconscious, although their reasons may be cognitively impenetrable. That is just one reason why affective memories are of great psychiatric importance. We want to know why we feel the way we do, and often the sources are best identified with the help of mental health professionals. Because emotional affects are major psychological "powers" of our lives, psychiatrists can be confident, and we too, that much of our higher mental apparatus was crafted by the way our affective experiences interfaced with the many challenges and vicissitudes of the world (Davidson et al., 2003).

In this chapter we will focus on our growing understanding of how our emotional memories—our secondary-process emotions—are formed. We are surprised that this large area of research, mostly arising from the study of fear-conditioning, rarely considers how the primary-process FEAR circuits of the brain—the unconditioned emotional response systems of the brain that generate raw affects—are of critical importance in generating fearful memories. Most investigators simply treat such primary-process emotional integrative systems as mere "outputs" of learning processes. This is one way that behavioral scientists have avoided the conundrums that animal emotional experiences pose for our fuller understanding of mammalian, especially human, BrainMind functions.

In addressing the nature of emotional memories, we will focus largely on the abundant and well-cultivated tradition of research on FEAR-learning in animals. We use this capitalized term hesitantly here, because affective change is largely unaddressed by the contemporary scientists who are

working out the details of fear-learning and memories. However, because of the chasm in communication highlighted by Steve Maren (see below), we will also attempt to fill in a variety of major gaps in knowledge that have been ignored by those scientists who are more interested in learning than emotions. Thus when we introduce the primary-process emotional issues—the unconditioned responses of this system—we will use "FEAR" and when we describe the work of fear-conditioners, we will use the lower-case form.

The most common model is the classical-conditioning of fear in rats and mice, similar to the Pavlovian procedures that Eric Kandel used in sea snails. In the rodent models, tones and lights (conditioned cues) are promptly followed by electric shocks to the animals' feet. After a few pairings, animals exhibit intense fear of just the tones and lights. As noted, few who study animals in this way explicitly acknowledge that the animals feel pain and fear. Some say such subjective aspects of animal minds cannot be empirically studied (LeDoux, 1996). But we believe that this is wrong, because we know that direct arousal of FEAR, and other primal emotional systems, with brain stimulation can serve as punishments in various learning tasks (Panksepp, 1991). This is the gold standard for concluding that certain types of brain activities are, in fact, experienced by animals. Since the existing evidence indicates that emotional feelings arise from the unconditioned (instinctual) aversion-generating FEAR networks within the brain, we may be wise to also consider how the neurology of such affective states contributes to learning. There are abundant reasons to believe that the memories that evoke anxieties, aversive to both animals and humans, have downward access to the FEAR circuitry. This does not imply that animals cognitively dwell on the events that caused such feelings—certainly laboratory rats and mice have limited tertiary processing of FEAR when compared to humans. In contrast, as highlighted in the poem that led off this chapter, chronic traumatically fearful thoughts in humans may emerge from the intrapsychic dynamics of the BrainMind, which is not only dwelling on anxieties about the remembered past but also worries about anticipated futures, which often arise from sensitized FEAR circuits with primary-process affective minds of their own.

In the case of FEAR, the memories of traumatic events may prompt us to suffer from chronic anxiety and nervousness, commonly accompanied by obsessive ruminations, much of which may occur in the medial regions of our frontal lobes (Northoff et al., 2010). In contrast, memories centered on

happier emotions may promote sustained cheerfulness, which often leads to a flow of positive ideas, hopes, and aspirations. Memories of devoted and fun-loving parents can leave a lifelong positive stamp—an invaluable psychological resource for navigating the tempests of future adversities. Such secure early emotional bonds are a lasting affective gift for the rest of one's life (see Chapter 9).

Memory, of course, is a useful tool for anticipating and dealing with future events, using past successes as a compass for future actions. Because raw affects are ancestral memories that are also experienced by animals, we can understand their functions in similar ways. By anticipating survival issues, intrinsic affective states provide immediate guidance of behavior. These feelings are connected to world events through learning. If so, we must conclude that animals experience many aspects of their reactivated emotions during memory experiments. Thus, through the blending of our instinctual emotional abilities and associated memories of external life events, we begin to experience many neutral aspects of the world affectively. Considering the vast complexities of learning and memory, there are many ways for this to happen, some of which have yet to be empirically evaluated. Here we will focus not only on the most solid learning research findings in the area, but also on potential paths of affectively guided learning overlooked by investigators who rarely consider the emotional-affective nature of animals which arise from their unconditioned emotional response circuits.

We pause here, just after the chapter on instinctual FEAR, to reflect on the remarkable achievements of learning/memory researchers. The great neuroscientific breakthroughs, achieved by studying simpler creatures such as sea snails (the work of Kandel and others), are now being emulated in studies of rats (Davis et al., 2010; LeDoux, 2000; Fanselow & Poulos, 2005; Maren & Quirk, 2004). It is likely that many of the general principles currently being revealed will generalize to other forms of emotional learning, albeit with abundant variations of details. Regrettably, most who currently study fear-learning do not explicitly acknowledge that their animals are experiencing fear. Why are most such investigators so hesitant to consider that research animals have any fearful experiences or even a coherent primary-process FEAR system?

After the appearance of a fine review article on conditioned anxiety (Maren & Quirk, 2004), Panksepp electronically asked a young colleague

whom he admired, Steve Maren, at the University of Michigan, the following question: "Why do the fear-learning people never mention or acknowledge the existence of a FEAR system coursing between the central amygdala and periaqueductal gray (PAG)?" He kindly responded with a very clear answer:

> I think the field generally appreciates that aversive stimuli can recruit unconditional fear responses at the level of the PAG and amygdala (although the specific properties of the US [unconditioned stimulus] that recruits responses at each level may be different)—so in that sense the system is not ignored. Nonetheless, the fear-conditioning world is dominated by people (including myself) that are primarily interested in how memory works using fear-conditioning as a model system, as opposed to people that are primarily concerned with how emotion works (the camp I assume you would affiliate with). So in response to your question, I think there are two cultures, one built around memory and conditioning and the other around motivational and emotional systems, and these cultures approach the same problems somewhat differently. Of course, these approaches shouldn't operate independently and more integrative work appears to be emerging especially now that human neuroimaging experiments have been folded into the mix.

This clearly helps explain the disjunction in the field, where the emotions of the animals are given second-rate status. It also explains odd statements such as "Not so long ago it [i.e., the amygdala] was an obscure region of the brain that attracted relatively little scientific interest" (LeDoux, 2007, p. R868). In fact, the roots of modern emotionally focused brain research go back to this region of the brain, in the discovery of what came to be called the "Klüver-Bucy (1939) syndrome," highlighting the tameness of wild animals after temporal-pole lesions that included the amygdala, part of the hippocampus and surrounding temporal cortex (see Figure 1.1). Half a century ago, it had long been clear "that lesions restricted mainly to the amygdala will produce docility in wild animals. This result has been observed in monkeys, in domesticated cats, and even in the very wild lynx. Wildcats, for instance, too ferocious to be handled without nets and protective gloves can be safely petted following bilateral ablation of the appropriate part of the amygdala" (McCleary & Moore, 1965, p. 121). Further, famous studies had shown that "when monkeys are tame in this way and then put back with their normal cagemates, the social relationships within the group undergo a change. The experimental animals fall to a lower level in the social scale" (ibid., p. 121, describing the work of Rosvold et al., 1954). A major collected work on the role of the amygdala appeared 40 years ago (Eleftheriou, 1972), and soon thereafter it had

become clear that the central nucleus of the amygdala was critically important for the FEAR response itself. What has happened more recently is that with the refinement of neuroscience methods, the analysis has now focused at a much finer circuit level than could be achieved even 20 years ago. Recent work has surely yielded a more precise understanding of the details of fear-conditioning by those interested in learning, but at the cost of leaving true emotions, which are very widespread brain phenomenon, outside their equation. This is yielding a rather narrow view of what the brain does when emotionally aroused. And this chapter will be couched not only in the recognition of the exquisite data harvested on the neural mechanisms of fear-conditioning but also on the potential mistakes that are being made by that narrow, behavioristic, nonaffective view of fear-conditioning.

The facts that have been harvested by the fear-conditioners are impressive, but one key issue continues to be disregarded—that primary-process neuro-affective processes of the brain may be *critical* mediators for how fear-learning occurs. Although it is not our major area of research, we are willing to predict that the affective, unconditional FEAR substrates of the brain play a critical role in setting up the fear-conditioning processes in the amygdala. Indeed, this can be generalized to all of the various "basal-ganglia" brain regions (e.g., amygdala, nucleus accumbens, bed-nucleus of the stria terminalis, etc.) where most secondary-process emotional learning transpires. How this might happen will be discussed in some detail later. But first, let us consider the happier side of the story. Fearful memories can be erased or overridden by "therapeutic" maneuvers that cleverly use the consolidation process against itself. "Consolidation" is the name for the complex brain processes that transform fleeting experiences first into short-term memories, and with a few repetitions of the experiences, into long-term memories. However, when painful memories are retrieved, they can be "reprocessed" and then "reconsolidated" in ways that are not as troublesome.

MEMORY IS NO LONGER AS STABLE AS A MOUNTAIN

Not so long ago, memory investigators thought of lasting emotional memories as permanent entities in the brain. Once forged, they were

assumed to be immutable. One metaphor was that they were solid as mountains. That perspective is no longer tenable. Emotional memories remain forever malleable, subject to influence by future events—through a phenomenon called reconsolidation (Nader & Hardt, 2009). This knowledge is especially important for effective psychotherapy. If we can soften the sting of emotionally painful memories by retrieving them in different affective contexts—rotating them in the mind's eye in different ways, so to speak—then it becomes possible to therapeutically capitalize on the simple fact that positive affects can counteract negative affects. By understanding that old and painful memories are not as immutable as mountains, therapeutic change becomes possible without drugs (although some medications can speed such changes). As we will see, that has become a major theme in the emerging modern science of psychotherapy (see Chapter 12).

With better techniques, therapists should be able to more effectively guide clients away from the memories of painful life experiences toward positive frames of mind. The hurtful aspects of many troublesome memories can be reconsolidated with the penumbra of new positive perspectives that are not so tormenting. Indeed, perhaps the day will come when undesirable affective memories may be pharmacologically mellowed, quite specifically and more effectively than with any current medicines. Such future therapies might be done, for instance, by dampening anxiety promoting norepinephrine (NE) influences in the brain with so called beta blockers such as propranolol (antagonists for one type of NE receptor) that can reduce the consolidation of hurtful experiences (McGaugh & Roozendaal, 2009). This specific drug, as noted in the previous chapter, is often used to reduce the bodily arousal common in "performance anxiety" that could disrupt the ability of people to show their skills or knowledge optimally. It might also be effectively used in reconsolidation. At present, there is another drug, *d-cycloserine*, which gently promotes glutamate transmission that can be used to therapeutically reconsolidate haunting, aversive memories in more life-affirming ways, by directly promoting the reconsolidation processes during psychotherapy. This idea has already been patented (Amaral & Roesler, 2008).

In short, it is now widely accepted that memories can be therapeutically remodeled. In the future, they may even be erased (Schiller, et al., 2010). However, the vast amount of knowledge about how our brains remember

and retrieve past events now needs to be supplemented by a better understanding of how our emotional arousals (i.e., the unconditioned responses provoked by unconditioned stimuli) set up learning processes within the brain. So, let us sort through some of the conceptual issues in FEAR learning/memory research.

CAVEATS: PRIMARY-PROCESS EMOTIONAL CONTROL OF LEARNING AND MEMORY

All basic emotional systems promote vast amounts of learning and memory in the brain, and in this chapter we will describe some of the ways that this process happens—how learning and memory (secondary processes) expand and elaborate our innate primary-process emotional capacities. Although we will focus mostly on FEAR-learning, we suspect that much of this knowledge will also apply to other emotional systems. However, the details for other emotional networks, except for SEEKING, are not as well worked out as they are for FEAR. Because we are especially interested in the clinical relevance of this work, we will also dwell on the many ambiguities that remain to be clarified. To reiterate, we believe the unconditioned brain mechanisms by which we experience "FEAR itself" greatly influence how fears are learned (Panksepp et al., 2011). Few fear-conditioning researchers have explicitly considered this possibility (i.e., that better understanding of FEAR unconditioned responses [UCRs] is critical for really understanding how fearful learning occurs in the brain). For sensory affects, perhaps the brain's unconditioned stimulus (UCS) processes are more important, but in considering emotional learning, we need to remember that emotional feelings are integrally anchored to the emotional action systems of the brain (FEAR UCRs). Thus, the pain of a foot shock gets directly into the PAG, and there it helps generate the unconditioned FEAR responses of freezing and flight.

In other words, although the seminal research in this area typically focuses on traditional concepts of learning (e.g., using predictive cues in classical and instrumental conditioning to allow animals to anticipate events), we believe the data warrant considering primary-process emotional systems—the nature of the UCRs—more explicitly in such schemes. The reason this has not been done in the past is because neuroscientists envision UCRs as mere "outputs" of the brain, rather than integrated emotional

systems. Thus, they have devoted little effort to understanding the inbuilt affective urges of the brain that they must use to obtain conditioning. Instead, they seem satisfied to believe that learning can be sufficiently envisioned as simply the association of "ideas" (a classic view in learning) —namely, that you just need to understand how conditioned responses arise from the pairing of external conditioned stimuli (CSs like predictive lights and tones, and aversive UCSs such as foot shocks).

But as soon as one envisions UCRs in fear-conditioning as being integrated unconditioned emotional response systems, which engender instinctual FEAR behaviors along with their punishing-negative feelings, the overall picture changes drastically. From our evolutionary perspective, such basic brain mechanisms of affect must surely be "instrumental" in how emotional learning occurs. In other words, the unconditioned emotional responses to environmental events are the felt "rewards" and "punishments" within the brain. If so, FEAR itself may be of foremost importance in fear-conditioning. In contrast, traditional behaviorist learning views largely restrict discussions to affectively neutral "reinforcement" processes. By focusing on such imaginary mechanisms, one can blind themselves to the evident fact that rewards and punishments are experienced, and if so, then the neural representations of the "affects" contribute substantially to the strength of the "reinforced" behavioral changes (we will call this the "affective neuroscience model"). This is a radical departure from the more commonly accepted behaviorist view. The behaviorist approach excludes, on first principles (i.e., because subjective *experience* does not count), any explicit consideration of how the various positive and negative affective processes of the BrainMind contribute to learning.

Before we examine the neuroscientific details of how traditional fear-conditioning has been used to effectively study the neural mechanisms of learning and memory, let us try to clear up a few of the many other conceptual issues in the massive learning and memory research area. This will take some time but we will eventually return to the novel ideas advanced above about the nature of fear-conditioning, where affect counts, as well as traditional fear-conditioning, where it does not. To set the stage, let's first focus on three common misconceptions about *learning* and *memory*, and then we will proceed to an extended summary of the many types and processes of complex memories, before returning to the affective neuroscience model.

First, nonscientists often think of learning and memory as intentional processes. Of course, intentional learning occurs when humans study something they want to master, whether in a classroom or the wider theater of life. In academic settings, learning and memory obviously involve calculated effort. As a child, you had to consciously apply yourself through repeated effort in order to learn and remember the multiplication tables and other factual memories (all generically called *semantic* memories). Indeed, perhaps this is the only way the cortex can be forced to learn what are often boring materials. To the best of our knowledge, most of the learning in other animals occurs when there are strong feelings involved. Indeed, left to our own devices, we are just like animals. We learn best when our interests —our SEEKING—has been aroused. All other emotional arousals also promote their own forms of learning!

People and animals typically learn and remember most important things automatically. For example, our fearful rat had no choice about learning to fear the bell that our proverbial cat wore around its neck. Similarly most people remember where they were when President Kennedy was shot or when the atrocities of 9/11 changed the world. They made no effort to learn or remember these facts. They could not help doing so. Emotional "flashbulb" memories are automatically consolidated within our brain networks because of, we would suggest, the power of the neurochemistries accompanying affective arousals. Indeed, for humans, we must also consider that, during the psychological turmoil of strong emotional episodes, we may learn things that are largely constructed within our imaginations.

Second, we tend to think that learning and memory always involve cognitive functions that are experienced in conscious "awareness". We think that we consciously figure something out when we learn and this is what allows us to remember. This is rarely the case. For instance, people and animals acquire and retain physical skills, such as riding a bicycle, via *procedural* learning—a form of memory that simply involves practice rather than cognitive insights. Although procedural learning often involves some level of instruction, which does involve experienced cognitions, we do not regurgitate those instructions when we "remember" how to perform a skill. Active imagery can facilitate and refine performance, but it is the practiced execution of a procedural sequence that makes it part of our increasingly well-oiled motor-habit apparatus. We typically refine the

performance of new skills without thinking about them. In fact, thinking about what one is doing actually disrupts performance. People even develop emotional habits in this way, as procedurally learned motor sequences, which are exhibited in their tone of voice, gestures, postures, and overall affective persona.

Higher cognitive experiences also play no essential role in the success of brain-conditioning mechanisms that result in emotional learning and memory. Much of the learning we will discuss proceeds without any help from the neocortex. Emotional learning involves the acquisition of an emotional response to a previously neutral experience. Emotional memory is the retention of this response over time. The cues that provoke learned emotional changes may often be unperceived—they may be totally unconscious. However, we do not think this applies to the unconditioned stimuli that provoke the most striking forms of emotional learning—for instance, the pairing of a tone or a light with a foot shock. The pain of the foot shock and the resulting FEAR are surely experienced even by animals without a neocortex. These animals show all the indices of pain, intensified in fact. This is not the same as to say that learning always requires affective experiences. It does not, especially in strictly cognitive forms of "declarative learning" that often require rote repetition (e.g., 7 times 7 = 49). But it is usually involved in real-life memories ("episodic" memories). In any event, for the kind of emotional learning we will consider here (and there are many, many types), acquisition of new responses is automatic and involuntary—and all the essential circuitry is situated below the neocortex. In this context it is important to recall that the raw experiences of the various primary-process emotions are also subneocortically generated—they are aspects of the unconditioned responses of the brain.

Third, many people think that there is just one type of learning and memory. This is not true. There are many, many ways this complexity has been subconceptualized by scientists (yielding ideas not always independent of each other). For instance, the procedural memories, and semantic/declarative factual memories (from which more personal, affectively rich, episodic, and autobiographical memories are constructed) that we mentioned above. This is not the place to discuss these complexities. We would simply emphasize that the best neuroscience work has been done with the simplest types of emotional memories, namely factual memories that can be studied (at least from the outside) by using

Pavlovian classical-conditioning principles. A slightly more complex type of such learning, contextual fear-conditioning, is highlighted in Figure 6.1, where animals learn to fear areas where they encountered predator odor. The important lessons we have learned through such studies do not necessarily apply to many other types of learning of great personal and clinical significance.

In sum, although we often tend to define learning and memory in terms of conscious intent and higher-order cognitions, many aspects of learning and memory are neither conscious nor necessarily cognitive. These memories can occur long before the maturation of our ability to have episodic-autobiographical remembrances—before we can recollect the various events of our lives, many with profound affective meaning. This is what often makes early childhood traumas so difficult to treat. Although people feel intensely about many things that are happening in their adult lives, they often have no way of knowing the causes of their feelings that were consolidated at an early age, long before they had the capacity for long-term explicit autobiographical memories. These are the most difficult emotional memories to manage with the "talking cure." In the penultimate chapter of the book, Panksepp will discuss alternative therapies that might address such early emotional memories.

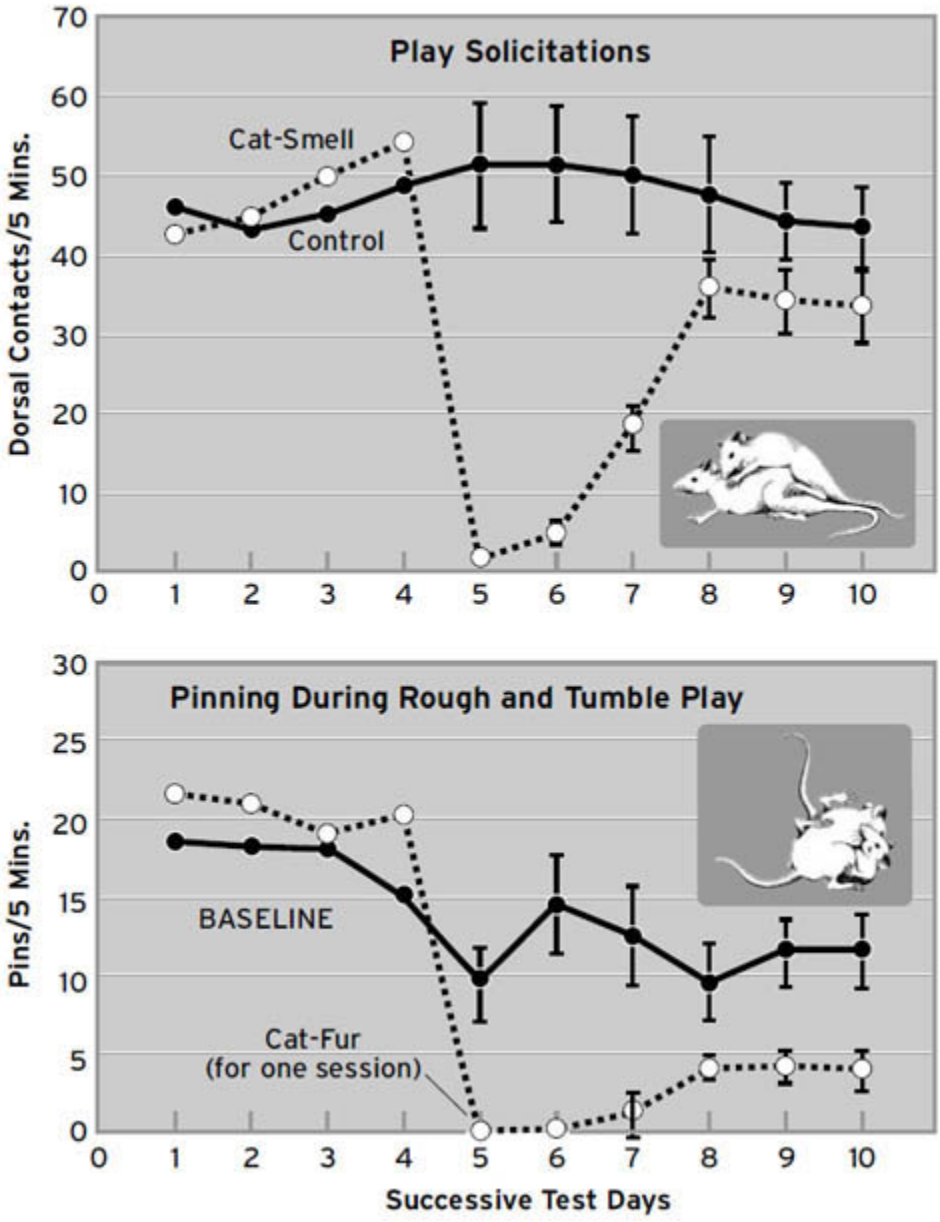

Figure 6.1. Following four baseline days of play, cat smell was introduced into the play chamber for a single test day (i.e., during a standard 5-minute observation session). Although the chamber was clean on all subsequent days, play solicitations (i.e, dorsal contacts) were markedly reduced for 3 days, while pinning was reduced for all 5 subsequent test days. The control group (solid lines) was not exposed to any cat fur. Data are means and ± SEMs (data according to Panksepp et al., 1994; adapted from Panksepp, 1998a, and republished with the permission of Oxford University Press).

There is a great deal of neuroscientific work left to be done before we can scientifically deal with such subtle issues. So far, most of the experimental work has been done with very simple, recently acquired emotional-behavior memories, as can be studied via classical-conditioning, rather than the seemingly free-floating affective residues from many past life experiences. Still, it cannot be emphasized enough that one great discovery of the past few years is that emotional reconsolidation of memories occurs each time something is remembered (Hardt et al., 2010). And astute new clinical interventions may be devised to soften the disturbing emotional impact of even forgotten memories. The field is rich with new ideas on how such memories can be reprocessed (e.g., see Fosha et al., 2009a and Chapter 12), but that literature is too broad and important to be adequately covered in the present chapter.

DIFFERENT TYPES OF LEARNING AND MEMORY

There is more to learning and memory than meets the eye, and investigators have carved the field into many distinct concepts. At the most coarse level, investigators distinguish between *explicit* (clearly cognitively experienced) and *implicit* (not cognitively, but commonly affectively, experienced) memories. We will occasionally use that categorization. Explicit memories have declarative, episodic, and autobiographical forms. The most common form of implicit memories is a procedural memory (e.g., learning a new motor skill). On top of this complexity, and at times underneath it all, we have the concepts of short-term, long-term, and working memories. Thus, modern cognitive scientists and neuroscientists have parsed the complexities of memory in various ways. We are left without any consensus that the carving has been done gracefully at the kinds of "natural joints" that would properly constitute the multidimensional complexities of learning. Thus, it would be premature to claim that the different types of learning and memory are completely independent of each other. Indeed, all these categories of learning and memory share many overlapping neurochemical processes within the brain. For instance, all rely on the neurotransmitters glutamate, GABA, acetylcholine, and norepinephrine to achieve their appointed functions in diverse regions of the brain.

The most meaningful memories for all of us are those highly embroidered, self-centered remembrances of our lives—the so-called episodic memories of important events, and our place in them, yielding abundant, highly personal autobiographical memories. Episodic memory essentially is a fully formed, personally meaningful remembrance that has integrated many aspects of an event, including information about specifically *what* happened, *where* and *when* it happened, and *who* were the main people involved. These distinctions were first emphasized by Endel Tulving (2001). He highlighted how episodic, especially intimately autobiographical, memories allow us to travel forward and backward in time through our experienced past—permitting us to imagine, and especially to anticipate and think about, future possibilities (Suddendorf & Corballis, 1997). In order to dwell and ruminate on the importance of such possibilities, one also has to utilize a limited-capacity, general-purpose "working memory" that is essential for explicit thinking processes. Here, past memories can be supposedly retrieved into a mental workspace that allows new perspectives to emerge.

Tulving suggested that other animals do not have such deeply thoughtful episodic memories, because they do not have an explicit sense of their own selves. However, modern research in several creatures, including scrub jays, suggests that these animals can use past information in current deliberations in relation to future goals (Clayton & Russell, 2009). For instance, birds that have cached food in specific locations, when being observed by another bird, will proceed to hide their food elsewhere when alone again. Do they thus have a sense of themselves? In Chapter 12, we will argue that all mammals do have a core SELF, perhaps deeply implicit, or minimally conscious on its own, but constituting a brain substrate that allows animals to have primary-process emotional feelings and ultimately explicit affective memories of past events (Northoff & Panksepp, 2008; Panksepp & Northoff, 2009). We will not focus extensively here on such important higher-order issues in other animals, though, because they are exceedingly difficult to study. It takes great experimental skill to obtain clues about the episodic capacities of other creatures. They do not have language to convey their past experiences and future aspirations. All information must be inferred from their behaviors. It is much easier to conclude that they have affective feelings from their emotional behaviors than to infer what else might be on their minds cognitively.

AN INTERLUDE: AN EXAMPLE OF THE FLOW OF MEMORIES

Let us pause briefly to discuss declarative memories, so-called because we can usually put such memories into words—we can "declare" them. These are the kinds of factual memories we can consciously recollect. We usually refer to this type of memory when we speak of "my memory" in everyday speech. Most of our memories of this type do not have strong emotional undertones. For example, if your Uncle Fred called to ask you to have lunch with him next Thursday, you would retain this information as a declarative memory. But you would not have particularly strong feelings about it. You might also remember the vibrant autumnal colors of a tree in your backyard, that 9 times 7 equals 63, that your car brakes need repair, and that you have lots of paperwork to do on Thursday. All these recollections would be declarative memories. However, some such memories can readily evoke strong feelings, namely those that were laid down solidly in the midst of emotional arousal.

It is widely accepted that many animals have declarative memories, although when studied in the laboratory they are reduced down to brass-tacks, namely the least complicated perceptual events. In this chapter, we too will eventually focus on classical-conditioning, the simplest type of declarative memory (namely learning that one thing follows another—usually a neutral event followed by an affectively arousing event). In the human animal, however, declarative memories can be far more complicated and elaborate, so let us begin with them. Consider an imaginary scenario: When your Uncle Fred called, you might have been deep in thought and the ringing of the phone might have startled you. You might subsequently remember how jarring the "bringgg!" of the telephone was. When Fred asked you to lunch, you did not simply remember the sounds that he uttered over the telephone. You had to understand and think about the meaning and implications of the things that he said. You had to consult your calendar. You also wondered why he seemed so keen to see you, leading you to think of various possible reasons. You worried that he might be in ill health. Such memories allow us to think through complex circumstances.

Thinking something through is a cognitive act that relies on keeping many pieces of past learning in a part of the mind that neuroscientists call "working memory," the neural machinery for which is very highly

concentrated in our massive dorsolateral frontal cortical regions (Goldman-Rakic, 1998). As you worked through the possible interpersonal intricacies, portions of your thinking processes might have been subsequently retained as new declarative memories, especially if they evoked strong emotional feelings in your MindBrain. You might remember the tense tone of Fred's voice, the apparent sense of urgency about having a personal meeting even though your schedule was packed. You would surely remember that you had clearly indicated that you could only meet on Thursday. After the conversation, you plan to fix in your memory the fact that you had eventually arranged to meet at a particular restaurant at 12:30. . . . But knowing how much you have to keep in mind and how limited your retrieval of stored information can be at times, you jot it down in your calendar, to aid your memory. All memories have short- and long-term components. Working memory operates with both of these components, as well as with episodic, autobiographical, and semantic contents. Because it uses such a complex array of memory systems, capable of being juggled in various permutations, the concept of working memory comes very close to the core meaning of "thinking."

As you think all this over, in a limited-capacity workspace, your working memory devises a plan of action: You will get up at 6:00 A.M. on Thursday and be in the office by 7:00. First, you will do your paperwork. Probably you will be done by 9:00 and then your colleagues will be at work and able to receive your calls. You have made an appointment to have your car serviced at a garage that is on the way to the restaurant that Fred suggested. While you wait, you will take time to unwind and read the *New York Times* over a cup of coffee. Then you will meet your uncle.

Items in working memory can be retained for longer periods of time in order for them to become encoded as declarative memories. When a declarative memory is created, it is then available for retrieval (for future use) by working memory. This means that when you are trying to think something through, you will have access to thoughts that you had in the past. As you think about Uncle Fred's lunch invitation, you remember that he is a retired neuroscientist with a very active imagination. And because of his interest in consciousness, you suspect he may want to share some strange new theory he has, perhaps to elaborate on his wild new idea of how the growth of dopamine systems in the brain guided human mental and cultural evolution. In any event, his urgent phone call stirred up many

emotional and cognitive possibilities in the recesses of your overly creative imagination (which is little more than memory in action). But you won't know what's really up until you meet him on that Thursday.

Our only point for now is that your consideration of all kinds of fantastic possibilities arises from your fluid reasoning capacities, which requires a lot of neocortical power and is what the neuroscientists call working memory. Working memory achieves its complexity from our vastly expanded frontal lobes, especially the more recently expanded lateral extensions (i.e., the dorsolateral frontal cortex), which evolved later than the more medial emotionally self-centered higher brain regions. We would only parenthetically note that such a capacity to juggle smaller units of memory can lead to novel ideas, many of which may turn out to be delusional. Considering that such memory abilities, especially in medial self-referential regions of the brain, are energized by ancient dopamine-SEEKING brain networks, we can see how memory formation, as it serves personal needs, can become skewed. For instance, patterns of SEEKING arousal, in conjunction with the spontaneous formation of memories about presumed causal relationships among events, can lead to various self-centered delusional behaviors in both mice and men. The transformation of correlated events into causal convictions, as lower brain reaches of the SEEKING system (Chapter 3) combine with associated cognitive events, allows us to envision how many of the grand breakthrough ideas of our species were created, as well as the many idiosyncratic psychotic delusions of individual humans.

In addition, affective feelings often guide our selection of autobiographical memories for retrieval and discussion. To follow up on our previous example, you know that your uncle has several serious medical problems. Suppose that Fred is on edge and solemn when you meet him at the restaurant. You ask if anything was wrong. He says that there is a serious matter to discuss. He has just turned 65, signed up for Medicare, and for the first time, is contemplating his mortality and his legacy. He starts by saying he has been keeping a secret from you for a long time. He proceeds to tell his story with a tinge of shyness and shame, blending into guilt. Suddenly, you notice every emotional nuance of his face and body. Years ago, when your father, his brother, was stationed overseas, Fred and your mother had engaged in an ill-fated love affair, during which you were conceived. Fred too was married at the time and when his brother, your

supposed father, came home, they all decided that it would be best for you to be raised as if you were the child of your mother's marriage. But now that his brother has been dead for a few years, Fred does not want to die without your knowing the truth. He is your father and the man whom you had always called "Dad" was your uncle. All of a sudden your world, both cognitively and emotionally, has been turned upside-down.

At that moment, you consolidate a "flashbulb" memory, because your emotions are so profoundly stirred. After the initial shock, you are deeply moved by this conversation (by a more surprising piece of information than you could have ever imagined on your own). And even if Fred had only mentioned it on that one occasion, you would never forget that he is your biological father. Such episodic memories would require a massive reorientation of how you understood your life and who you are. Memories as intense as these only fade if you develop the mental deterioration that is characteristic of Alzheimer's disease or fronto-temporal dementia (Pick's disease). Clearly, intense emotional arousal is a big player in these kinds of lasting declarative-episodic memories that have the potential to remake us as they are integrated into our vast autobiographical storehouses of personal memories. The higher mind functions that accompany these memories, widely distributed in the brain and important for psychiatric disorders, can now be partly visualized with brain-imaging technologies such as functional magnetic resonance imaging (fMRI) (Naghavi & Nyberg, 2005; Ragland et al., 2007).

THE ACTUAL MECHANISMS OF MEMORIES

Most of learning-memory research has proceeded without much concern about emotional issues. The central hypothesis since the middle of the last century was one proposed in 1949 by the Canadian psychologist Donald O. Hebb (1904–1985). His core idea is captured by the celebrated catchphrase "neurons that fire together wire together." In other words, when two neurons in a network fire in a cascade, an enduring synaptic bond is created between them (Hebb, 1949).

All neural pathways, whether stable or fleeting, are created as concatenations of neurons that secrete chemical neurotransmitters across the tiny synapses that separate them from neighboring neurons. The presynaptic neurons secrete neurotransmitters that bind with receptors on

the surface membrane of the postsynaptic neurons. Decades of research have shown that, for most memories, the cardinal transmitter (but not the only relevant one) is glutamate, arousing primarily NMDA-type receptors (there are two other major types). Most neuroscientists envision that, during the creation of a memory, neurons secrete glutamate across synaptic clefts onto receptors on various postsynaptic neurons, and through a complex chain of such intensified firings, a phenomenon known as long-term potentiation (LTP) emerges (Bliss & Lomo, 1973). LTP is mediated by cascades of intracellular molecular events (too complex to discuss here), which increase the arousability of the neural pathways that constitute such memories. Every time the same pathways are aroused by related events, they become more sensitized, and thereby become ever more arousable.

Much about the neurophysiology of long-term memory formation has been revealed by *in vitro* approaches with slabs of hippocampal tissue (Tronson & Tayor, 2007). Still, even as these details are deciphered, there remains a chasm of ignorance between the fine molecular mechanisms of LTP and the nature of the real-life memories that we have been discussing. To understand our lasting personal memories, we will need to have a much clearer understanding of how basic emotional and motivational systems, the major evolutionary tools for living inherited by organisms, participate in learning. In other words, the fine molecular details need to be complemented by large-scale neuronal network approaches—in the lexicon of chaos theory, nonlinear dynamic network views—to understanding the real psychology of learning in the BrainMind. As noted in Chapter 3, we could also envision simplified *in vitro* models that study anticipatory neural changes in the SEEKING system. That would be a major step above LTP in the mechanistic analysis of memory formation.

Our memories are forged with the essential help of complex networks that represent organismic needs and emotions, as opposed to simple stimulus-response neuronal chains. In this modern view, learning may reflect the way various stimulus-response networks get embedded in the much larger-scale networks that represent the primary biological and psychological concerns of organisms. Such global images of MindBrain functions are, we believe, essential ingredients for understanding the kinds of learning and memory that are most meaningful for us, such as our capacity to anticipate important events in the context of the autobiographical memories that are so intimately connected to our feelings.

The decaying of memories is just as important as the formation of memories. We have very little solid knowledge about why certain memories are retained while others are forgotten. Most neuroscientists currently think that forgetting is an active brain process for eliminating unused information, perhaps as a way of learning in reverse. In addition to the discovery of chemicals that strengthen memories, scientists have found other chemicals that can actually erase them. For instance, rapid chemical erasure of specific long-term memories in the neocortex has recently been achieved by a molecule called zeta inhibitory peptide (ZIP), which helps disperse glutamate receptors from synapses that originally consolidated the memories (Shema et al., 2007).

In this context it is important to recall once more, like a mantra, the recent discovery that memory storage is an ongoing dynamic process. Memories are not only constantly subject to the dynamic process of consolidation but they are also affected by "reconsolidation" (Tronson & Tayor, 2007; Schiller, et al., 2010). This means that when humans and other animals are using their memories, and the memories thereby revert to an active processing mode, they can be remodeled and then reconsolidated in forms that are different from the original memories. Such reconstituted memories typically include information about new emotional contexts that were not present when the original memory was consolidated. Thus, old memories become temporarily labile when retrieved in new contexts, and they are re-processed accordingly. Even though Freud did not know anything about such brain mechanisms, it seems that he was already well aware of the fact that memory processes operate in this way, and he invented the word Nachträglichkeit to describe the kind of mental process that is characterized by psychic temporality and construction (Eickhoff, 2006; Faimberg, 2007). This basically means that memories can be reconstructed from not only the past to the future, but from an imagined future to the past.

We think the most emotionally troublesome memories can be effectively changed in this way during the course of especially skilled psychotherapy. This may be one reason why certain forms of psychotherapy are more effective than others. From recent evidence, contrasting many studies using various psychotherapeutic approaches, it seems that the psychodynamic-psychoanalytic approaches that revolve so centrally around memory processes will often yield the longest-lasting benefits (Shedler, 2010). This

may be due to the fuller use of affective re-processing of the past than the less ambitious therapies that focus just on current cognitive interpretations —issues that obviously should not be neglected.

"WORKING MEMORY" IS ESSENTIAL FOR OUR ABILITY TO THINK

Initial formulations about working memory were purely psychological constructs, and like most psychological constructs, the concept of working memory did not inform us very clearly about the underlying brain functions. Psychologists initially focused on the fact that we can remember only a limited number of items at any given time (seven items plus or minus two). But much evidence indicates that working memory is more broadly a higher-order cognitive function. For example, working memory capacity is proportional to IQ (Conway et al., 2003). The substrates of working memory are scattered widely across cognitive areas of the brain, but as already indicated, they are apparently concentrated heavily in the dorsolateral frontal lobes of the neocortex. These neural substrates help generate an extensive array of cognitive functions, ranging from language recognition to visual/spatial information processing, to attention and overall cognitive coordination and higher reasoning processes (Baddeley and Hitch, 1974). The world of memory is full of complexities that bedevil the simple ways that scientists are forced to conceptualize the cognitive networks of our minds.

For example, working memory encompasses such a wealth of cognitive activities that it could more accurately be termed *working learning*. It encompasses brain functions that juggle and process information that is derived from the external senses with knowledge arising from memory stores, not to mention the emotional contexts in which all this happens and that, in the process, can change the memory stores themselves. The fact that this kind of learning can be completely internal, often under the rule of associated emotional arousals, is of great importance for psychotherapy as well as for the everyday misunderstandings that people have in interpreting the same events.

At present, working memory is poorly understood in any well-resolved neuroscientific terms. The neocortex is the brain's major cognitive substrate. Its interconnections are vast and complicated, and it uses many

brain regions concurrently; after a lifetime of learning, it is like the conductor of one extensive orchestra. However, it is important to emphasize that cortical processing is under the control of a host of subcortical state-control processes, such as those that control forebrain levels of acetylcholine, dopamine, norepinephrine, orexin, and serotonin, working alongside the ever-pervasive glutamate and GABA neurons in every cognitive act. The cognitive cortex would be hopelessly deficient if it were not for these subcortical, global regulatory systems. Indeed, there is evidence that dopamine, a great facilitator of enthusiasm-filled ideas (both rational and delusional), has greater purview in the human brain than in most other mammalian brains. Anatomically, dopamine networks extend far back into the perceptual cortex of human brains, farther than they do in rats and most other animals, where these networks are confined to the frontal regions. Indeed, the amplification of dopaminergic processes, including predatory SEEKING urges, in human evolution (both cultural and biological), may explain the intellectual complexities and delusional tragedies of the human mind just as much as the massive expansion of information-processing tissues within our cortical thinking cap does (Previc, 2009). Thus, interaction of the diverse primary-process emotional systems with the higher neocortical-cognitive regions, which surely generate higher-order psychological consequences (many of them culturally molded), will never be fully understood through the study of overly simple animal models. Conversely, those higher complexities cannot be understood without clear visions of the ancient layers of the mind that we still share with all the other mammals—a foundation without which the higher mind would collapse. Animal models are always needed to work out the details of any and all basic neuropsychological mechanisms, but not the tertiary-process mental abilities in which humans excel.

Indeed, perhaps because of the inadequacy of animal models for understanding human cognitions, we may never understand the human mind at a fine neural level. For example, we do not even yet understand the neuroscience that supports the shuffling and consolidation of simple percepts into complex concepts. Psychologists have discovered that the number of usable items in working memory does not vary by much but that the complexity of each item does. We do not understand how primary-process emotional arousals link up to such tertiary cognitive-thinking processes, and the detailed neural work that is necessary to find out is

ethically impossible in humans. But as we will see, we do have a solid science of how emotional arousals link up to secondary processes, which are the simpler forms of learning such as classical conditioning and especially fear-conditioning.

HIGH AND LOW ROADS OF SENSORY-EMOTIONAL CONDITIONING

Now let us return to the simplest model of fear-learning, favored by behavioral neuroscientists. Traditional fear-conditioning works like clockwork. Tone and shock, tone and shock, just a few times, and the animal will behave fearfully in response to just the tone. The speed and precision of learning explains the appeal of such unappealingly stressful methodologies to those interested in understanding the brain basis of learning and memory. Before examining this research, let us briefly review how sensory information is processed. Practically all senses have to go through the thalamus before they ever get to the neocortex; the one exception is smell. The two conditioned stimuli that are most commonly used in fear-conditioning are sight and sound, each of which gets handled by distinct nuclei in the thalamus. However, the painfulness of a foot shock is already felt far below the thalamus in the PAG.

Indeed, all sensory information is first processed subcortically, and at some point, most of this subcortically processed information ends up at the thalamus, which serves not only as the main way station that sends external sensory information up to the cortex, where it is transformed into refined perceptions, but also as a sorting, mixing, and reprocessing station. However, the affective components of incoming sensory information often diverge into hypothalamic areas and into reticular fields of the thalamus that do not project to the cortex. And this may be important for fear-conditioning. A foot shock is not like an ordinary somatosensory cognitive-type stimulus like the feel of a hat on your head; the pain and fear that are induced are highly affective (painful first, and fearful next). It may be a big mistake to assume that the aversive unconditioned stimulus aspects of a foot shock need to go to the cortex (via the projection nuclei of the thalamus) in order to be transformed into pain and fright. But that is what some fear-conditioners seem to assume (LeDoux, 2007, Figure 4).

The thalamus receives as much "return" information from the cortex (always indirectly via the basal ganglia) as it initially receives directly from the senses. Different regional neuronal cell groups (nuclei) of the thalamus process the different kinds of sensory information. For example the lateral geniculate nucleus (LGN) processes visual (light-wave) stimuli while the medial geniculate nucleus (MGN) processes auditory (sound-wave) information, and so on. It is noteworthy that both types of nuclei are presumably fairly recent evolutionary additions to the thalamic level of sensory processing, since both are situated at the far lateral edges of the thalamus. Senses like those of taste, touch, pain, and kinesthesis are more centrally situated in more ancient regions of the thalamus. The most ancient of the external senses, smell—a primitive form of taste (tasting the air!)—does not even need to go through the thalamus to get to certain ancient regions of the cortex (the pyriform cortex), even though a great deal of olfactory information does end up in one of the most ancient parts of the thalamus, the dorsomedial nucleus. This nucleus is also very important for emotional processing, especially for social emotions related to attachments, including separation distress (see Chapter 9). In any event, current work on the classical conditioning of fear is largely restricted to auditory and visual stimuli that are processed by the newcomers, the LGN and MGN.

When the LGN receives visual information from the world, it sends it in two directions: The LGN sends information up to the sensory (auditory) cortex where animals have high-level consciously detailed experiences of seeing. However, the LGN also sends information downward into the amygdala, where the highest reaches of the primary-process FEAR and RAGE systems are situated. These have become known as the "high" and "low" roads to conditioning.

Experimental studies with rats indicate that subcortical visual processing of fear-predictive cues can directly arouse the lateral amygdala (e.g., Doron & LeDoux, 1999; Shi & Davis, 2001). Similar subcortical pathways have also been observed in human subjects (Campeau & Davis, 1995; Linke et al., 1999). Probably other forms of low-road sensory processing, regarding sound, touch, taste, and smell, can also arouse various emotional responses, although this is apt to vary from species to species. Overall, however, an animal does not have to have a vivid cortical experience of seeing, hearing, smelling, tasting, or touching in order to have unconditioned (instinctual) emotional responses or simple learned (classically conditioned) responses

to certain stimuli. The low-road processing arouses conditioned emotional responses by sending subcortically processed sensory information to the thalamus, which in turn sends the information down to the headwater of the FEAR system in the amygdala, namely the central nucleus. Almost by definition, when this happens, an animal has the experience of a *raw emotional affect*. Again, empirically, the existence of affect is demonstrated by the fact that mere electrical stimulation of the FEAR system can serve as a punishment in learning.

The cortical route to fear-conditioning—the high road (the LGN to auditory cortex and then back down to amygdala)—has been praised for being "clean" because it provides a high level of stimulus resolution. For example, the auditory cortex can distinguish between a gunshot and a loud blast of rock music. However, the high road is comparatively slow to process information. The low road can process information far more rapidly. The low-road "shortcut"—directly from the LGN to the lateral amygdala—has been touted as being "fast" but "dirty," because it takes less time for processing (an estimated twelve one-thousandths of a second) and does not provide animals with any fine-grained perceptual distinctions (LeDoux, 1996). The high road is half as fast. Too much should not be made of this; it probably reflects little more than the fact that it is a much longer road with more synapses. In any event, it is assumed the low-road processing might not be able to distinguish two startling sounds—such as a gunshot from a blast of loud music—but such stimulus discrimination issues have not been adequately studied. What we can be sure of, though, is that the low-road (brainstem to LGN directly to amygdala) can condition more rapidly, at least in rats (Figure 6.2).

It is important to be clear that not all low-road processing of emotions is conducted via the amygdala. For instance, sudden loud sounds that produce a startle reflex do so strictly at the level of the brain stem (this is twice as quick as the "low road" to the amygdala), and it is well established that anxiety—sustained fearfulness—sensitizes this startle pathway. For instance, visual cues that predict shock make the startle reflex more intense. Thus, fearfulness sensitizes primitive protective reflexes. We also think that arousal of the FEAR circuitry sensitizes the conditioning mechanisms largely, we propose, by upward influences from the PAG. In a sense, the FEAR system is the conductor of fear-learning. This is not a popular or even widely discussed view, because what we call the FEAR network is

typically treated as if it were a psychologically vacuous, mere behavioral and autonomic "output" system for conditioning (Davis, 1992; LeDoux, 1996).

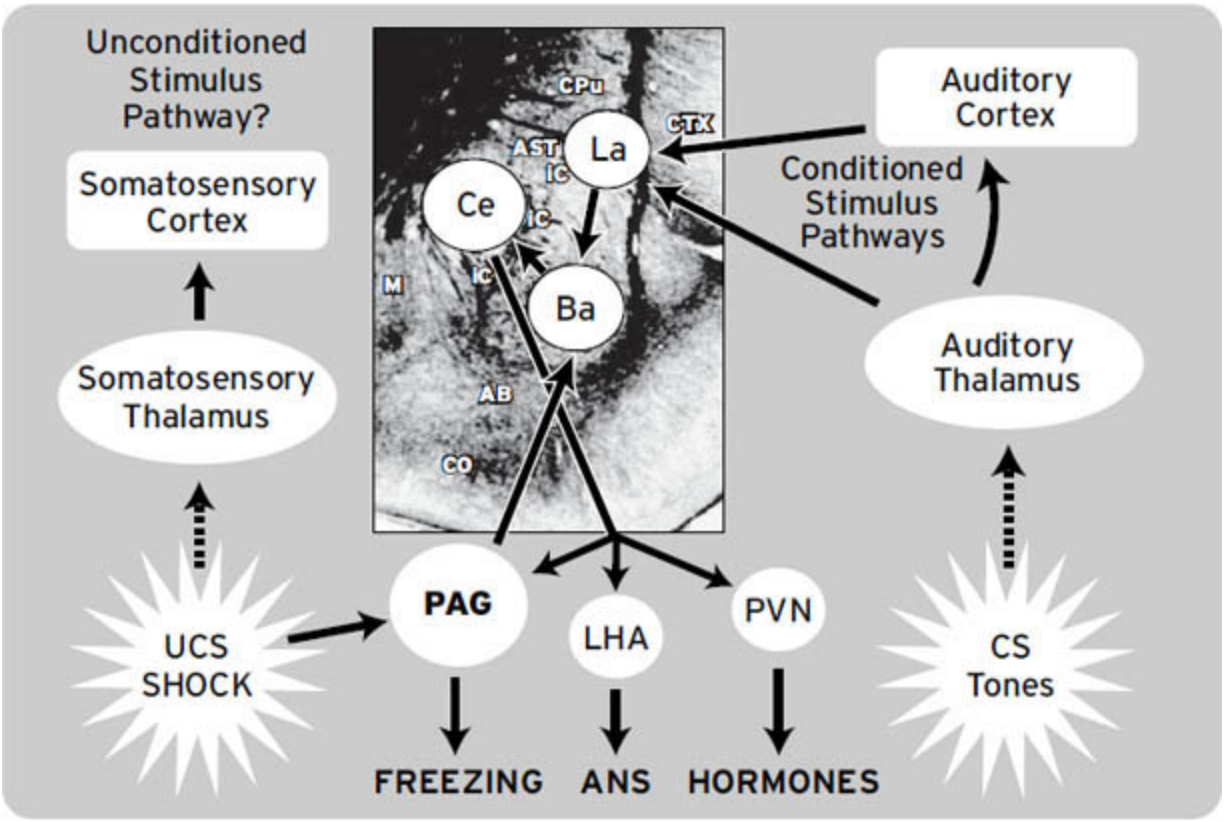

Figure 6.2. A schematic summary of classical conditioning of the FEAR response. Typically, in such work a tone-conditioned stimulus (CS) is followed by a foot shock (the UCS). The auditory stimulus ascends via Cranial Nerve VIII to the cochlear nucleus in the brain stem, which projects to the midbrain auditory processing way station of the inferior colliculus (not shown), which projects to the medial geniculate nucleus (MGN) of the auditory thalamus, and then projected to the neocortex (right side). The MGN has pathways down into the amygdala first the lateral nucleus (La) and then further down to the basal nucleus (Ba). These normally do not access the FEAR system, which starts in the central nucleus (Ce). However, the shock (UCS) does have a similar pathway upward in the brain, but it also diverges into the FEAR system of the periaqueductal gray (PAG), which directly activates the UCR—the unconditioned instinctual fear responses. With the conjunction of the CS getting into the amygdala, it is

proposed here that the upward FEAR influence of the PAG is instrumental in leading to the "opening of the amygdaloid gate," whereby the CS gains access to the FEAR system after learning has occurred in a few trials. This is the alternative affective neuroscience interpretation. The traditional view is that the UCS reaches the thalamus, just like the CS, and there the conjunction of the sound and the touch in the La is the critical link for conditioning. These two views remain to be directly empirically contrasted (adapted from, with substantial modifications, from LeDoux, 2007).

This very reliable fear-conditioning model, using shocks to the feet, is currently being studied intensively by dozens of labs. One only hopes that some labs will eventually shift to milder fearful "punishments" than the foot shock, such as air puffs to the back of the neck that promote 22-kHz ultrasonic complaints (Brudzynski & Holland, 2005), a stressor that generates a much milder form of anxiety than the outright fearfulness of an impending foot shock, which suppresses these same calls (see Soltysik & Jelen, 2005). An even less obviously aversive "punishment" for rats is to expose them to the smell of a cat; rats are very fearful of such stimuli, which we can barely smell, and they associate those experiences with the environmental contexts in which they occurred (Figure 6.1). Such milder models may be much more relevant to understanding human anxieties. Indeed, considering that positive emotions can be conditioned just as rapidly as the FEAR system can provoke anxiety, namely as with conditioned tickling responses that generate happy 50-kHz calls (Panksepp & Burgdorf, 1999), one can only hope that investigators who are simply interested in the mechanisms of conditioned learning will shift to model systems that reflect positive emotional "reward" learning rather than intense negative, affective "punishment" conditioning. The kind of appetitive conditioning that Jim Olds conducted at the end of his life (see Chapter 3) is an excellent model of rapid conditioning that involves no aversive stimuli.

In any event, high- and low-road FEAR-learning usually occur simultaneously and complement each other; in general, the higher regions of the brain regulate lower regions and the lower regions arouse and sensitize higher ones. So these forms of learning are coordinated in presently unknown ways. In this context, it is important to remember that the neocortex, through its many downward inhibitory influences, can quell

emotional arousal. This is, of course, a very adaptive response. Suppose that a speeding car backfired just as it was passing you. This sound would be initially processed via the lowest auditory "road" in your brain, making you startle, which might then lead to a FEARful arousal. You might freeze in your tracks, with your eyes wide open and your breathing shallow. A moment later, various neocortical cognitions would inform you that it was only a blast from a poorly tuned car. Your neocortex would then regulate (inhibit) the arousal of your FEAR system and you would rapidly calm down.

As we have noted, currently many investigators envision affective experience to be a subset of cognitive activities, which is reasonable from the perspective that they surely interact strongly in the intact brain (e.g., Pessoa, 2008). However, from an evolutionary perspective, it is wise to see these activities as distinct levels of control: At the primary-process level (and even with the addition of simple learning), there is no reason to believe that animals with very little cortex have thoughts about their experienced emotional arousals. Still, we cannot exclude that possibility, especially since rodents that learn to be fearful very rapidly do require the participation of the higher medial frontal cortex to unlearn their fears. It is now generally agreed that this process, called "extinction" (the cessation of responding when rewards or punishments are terminated) is not simply forgetting but is an active learning process. And this type of unlearning requires more brainpower—namely, more neocortical participation—than the initial learning itself (Myers & Davis, 2007; Sierra-Mercado et al., 2011).

As with every emotional system, there is more to be learned than is yet understood. One great mystery is why the punishment that is produced by direct stimulation of the FEAR system does not link up readily to learning a specific activity (such as pressing levers in response to predictive CSs in order to avoid an aversive event) in the way that they do to a foot shock (see Panksepp, Sacks et al., 1991, for a full discussion). In contrast, animals do easily pick up a conditioned place avoidance in response to FEARful brain stimulation (Panksepp, 1998a, p. 214). We suspect that this is simply due to the fact that it is hard for animals to learn how to be afraid of FEAR itself without the affective companionship of pain.

But even with electric shock mediated FEAR-learning, it is not yet completely clear whether all predictive stimuli (hearing, smell, touch, etc.)

can yield the type of light- or sound-signaled fear-conditioning that is commonly studied by the fear-conditioner or how many sensory systems have the fast, low-road access to the FEAR system. Vision may not. For example, in rats, sound waves that are processed along the low road will travel to the thalamic MGN and condition fear better than visual stimuli, which go to the adjacent LGN. However, if one surgically coaxes the visual system to terminate in the MGN early in life, these animals condition much more effectively to a visual stimulus (Newton et al., 2004). Apparently in the rat it is not so much a matter of which sensory system enters the thalamus, but rather how effectively the thalamic projection nuclei send information to the FEAR system. This suggests that different sensory systems in different species may be differently "prepared" to mediate rapid fear-conditioning.

However, it seems unlikely that the neural mechanisms of conditioning, whereby the incoming sensory information from the MGN to lateral amygdala develops new access routes (learned linkages) to the central nucleus of the amygdala (the headwater of the FEAR system), are different in different species. Likewise, it is unlikely that the principles of operation of the underlying FEAR system, descending from the central nucleus of the amygdala to the PAG (Chapter 5), are much different in different species. Once you have good solutions for evolutionary memories (e.g., the unconditional FEAR response system) as well as solid mechanisms for emotional memories (fear-conditioning), why discard them? On the other hand, one would, of course, expect a rabbit to have a relatively larger and more responsive FEAR system than a lion, and that higher brain mechanisms in different species would deal with emotional situations in different ways.

THE EXPERIMENTS OF JOSEPH LEDOUX

Much of the most informative fear-conditioning work was done in the laboratory of Joseph LeDoux. He was also at the forefront of working out the actual molecular details of conditioning in the amygdala, emulating comparable earlier work by Eric Kandel in sea snails. LeDoux wanted to fathom the neural changes that mediate successful conditioning in subregions of the amygdala. The conditioning work of his group, and others, was meticulous and promoted many additional advances (Davis et

al., 2010; Ehrlich et al., 2009). But it also regrettably led to the mistaken idea that the amygdala is literally the "headquarters"—the Grand Central Station—for emotion generation in general (LeDoux, 1996). It is not. There are many brain regions of equal or greater importance for the primary processing of various emotions. And if any area of the brain deserves the distinction of being called the Grand Central Station of emotion, then clearly it is the PAG rather than the amygdala. The PAG is involved in *every* primary-process emotion, in absolutely critical ways. In contrast, the amygdala participates mostly in FEAR, RAGE, and LUST, and it contributes much more in the service of stimulus-stimulus learning (CS-UCS) than the actual orchestration of the unconditioned (instinctual) fear responses themselves, which are more globally essential for that type of learning. Here we will present only some of the general principles revealed by LeDoux's seminal research on fear-conditioning, which has been amply supplemented by other prominent investigators like Michael Davis (Davis et al., 2010), Mike Fanselow (Fanselow & Poulos, 2005), and Steve Maren (2005).

It has long been clear that, prior to conditioning, modest sounds and lights do not have intrinsic access to the FEAR system (they are not fear UCSs like a cat smell). This is why rats are not initially afraid of the tones and lights typically used as conditioned stimuli in classical-conditioning experiments. However, information about the most commonly used unconditioned stimulus—the painful electric shock that is used to simulate a predator's bite—always has access to the FEAR system. The exact pathways through which pain directly impacts the FEAR system have not been clearly delineated. This is probably because pain can enter the FEAR system at many levels—all the way down in the PAG (thereby activating the whole FEAR system at very low levels of the brain) as well as by higher inputs from the various thalamic reticular nuclei that mediate pain transmission. There are also many other inputs and facilitators, for example, the pathways of nonspecific brain modulators, which promote cognitive linkages, such as acetylcholine and norepinephrine. When information about the shock reaches the FEAR system, it can coordinate with associated neutral information coming in from the low road of the thalamic MGN to the lateral regions of the amygdala. When any stimulus has proven to be fearfully significant—has evoked negative affect consistently, as an electric shock always does—then nerve cells in the basal and lateral amygdala

(BLA) respond by developing more robust functional connections to the central nucleus of the amygdala. Now, neutral stimuli can activate the "royal road" to FEARful feelings.

The central nucleus of the amygdala along with many lower brain structures in the hypothalamus and midbrain constitute the FEAR system (see Chapter 5). When the central nucleus, at the pinnacle of the primary-process FEAR system, comes to be aroused by conditioned FEAR stimuli, typically the whole FEAR system has been aroused, and the rat exhibits the whole gamut of fearful reactions—freezing, elevations in blood pressure, pooping, and a host of other autonomic responses. These diverse fear responses all have slightly different pathways further down in the brain stem, but they typically all work together in the intact animal. The animals also display a distinct negative affect—they seem to feel uptight in a very trembly, scared sort of way. It is important to remember that the pain from a foot shock sets up the conditions in the nervous system whereby closely associated stimuli—predictive cues—can come to control those anticipatory-conditioned emotional behaviors that are essentially identical to the unconditional (instinctual) FEAR responses. In other words, the arousal of the FEAR system (and hence fearful affect, which is a neural state) within the central amygdala may be critical for learning to occur.

In any event it is among those new functional connections between the lateral and central amygdala where associative cues mediate fear-conditioning, and hence this is where behavioral neuroscientists interested in fear-learning have devoted most of their attention, without clearly acknowledging that the affective UCR of FEAR arousal was critical for conditioning to occur! Whether any similar conditioning occurs in lower brain regions such as the PAG remains unclear, but if it does, we would anticipate that it would be a very broad network change, as is entailed in "sensitization"—the prolonged change in emotion-network responsivity induced by repeated emotional arousals.

Since the above mentioned new UCR-FEAR dimension for conditioning to occur has been neglected, let us reiterate the traditional view of conditioning. When a naive rat is initially exposed to an unconditioned predictive cue (e.g., the tone), its FEAR is not aroused. How then does the conditioning process, arising from the quick succession of neutral stimuli and shocks, give the previously neutral cues new access to the FEAR system? LeDoux surgically damaged either the auditory cortex (the high

road) or the pathway leading more directly from the thalamus to the lateral amygdala (the low road), and he found that rats with high-road damage became conditioned quite rapidly, which meant that predictive information could proceed effectively along the low road and arouse the rat's FEAR system. On the other hand, rats with a damaged low road (i.e., MGN lesions) became conditioned but the process came about very slowly.

MGN-lesioned rats gradually became fearful in response to the shock-associated tones. This same slow emotional conditioning via the cortex had already been observed a long time ago, with tones that predicted food, whereas conditioning of neurons in subcortical regions proceeded more rapidly (Olds et al., 1972). The advantage of having rapid conditioning is obvious, but it is not at all obvious why the subcortical systems would condition more rapidly than the cortical ones.

From our vantage, it is clear that the more ancient brain systems should have priority in learning simple adaptive responses. In other words, lower regions of the brain may condition more rapidly because they achieved that competence much earlier in evolutionary time, and hence they have some priority in the overall learning process. Perhaps it also makes sense if one recognizes that neocortical areas are unable to initiate any emotional responses on their own unless they have been trained to do so. Only with persistent training do certain higher brain regions come to instigate emotions. In other words, the neocortex only slowly develops the ability to relay explicit cognitive information to the amygdala in order to instigate learned emotionality. This may be one reason that strict cognitive approaches to psychotherapy may not be as effective as those that concurrently use skilled primary-process affective maneuvers (see Chapter 12).

Clearly, the subcortical sensory low roads directed toward the emotional systems hold some of the keys to effective, rapid conditioning. But we think it is actually the instinctual FEAR system—the shock induced UCR—that provides a critical key for fear-conditioning to happen. This is not the way most traditional learning theorists see it, however. If our perspective were generally accepted, this shift in understanding would have profound implications not only on the kinds of experiments conducted, but also on the clinical treatment of emotional disorders (see Chapter 12). In any event, such low-road conditioning probably proceeds without conscious "awareness"—without any cognitive understanding—but it surely is replete

with intense affective experiences, namely affective phenomenal consciousness.

This has important clinical implications. The idea that feelings only come to be experienced—become conscious—if conveyed through the cognitive-conceptual "libraries" in the neocortex via some kind of "read-out" (see Chapter 2), is a belief, not a fact. In contrast, the ability of the FEAR system to generate an awful experience is a fact, not a belief. In any event, LeDoux's group observed that when a rat is conditioned to be afraid, cells in the BLA fire more frequently as they develop connections to the central nucleus of the FEAR system. For a summary of this learning circuitry, see Figure 6.2, which we have modified from LeDoux's work to highlight the way that the FEAR system may sensitize the amygdala's learning mechanisms from below (for present purposes, we conflate the separate neural processings within the BLA areas into a single BLA component).

The BLA cells utilize glutamate, and they act on postsynaptic glutamate sensitive NMDA receptors (there are several types of glutamate receptors)—presumably converting "silent synapses" to active ones (Kerchner & Nicoll, 2008). That linkage—increased permeability of the initially closed synaptic gates, mediated partly by local inhibitory neural loops (Ehrlich et al., 2009)—then proceeds to transfer conditioned information emanating from the BLA area to the central nucleus of the FEAR system. This fear-conditioning mechanism is similar to learning mechanisms used by other emotional systems, which are being studied in very comparable ways, but about which we typically know somewhat less (for instance, learned appetitive-SEEKING behavior in the nucleus accumbens, which typically incorporates the "rewarding" power of the ascending dopamine systems into its vision of conditioning). So far, it appears to be a general principle that each of these interfaces between cognitions and primary-process emotional systems uses increased glutamatergic transmission as the mechanism to generate learned responses.

Still, it seems self-evident that for emotional learning to occur, people and animals must become emotionally aroused by an unconditioned stimulus. This suggests that the unconditioned response needs to be seen as an active part of the conditioning process—helping explain how linkages are made to the predictive conditional stimuli. If so, it may be the unconditioned emotional response of the nervous system—in the present case, the arousal of the FEAR system—that may be especially critical for

learning. In our estimation, the way in which the FEAR system mediates fear-learning remains a much under-discussed aspect of how emotional learning in the brain works. This neglect of the UCRs in learning may reflect that motor systems have typically been envisioned as mere "output" circuits, without the recognition that the complex primary-process emotional action systems are actually within-brain integrative action circuits, with psychological-affective dimensions.

Let us make a specific hypothesis, and this may be more than general readers may wish to follow: First it should be noted that glutamate receptors come in two broad categories—AMPA and NMDA varieties. Most of the focus so far has been on the role of the NMDA receptors in mediating the conditioned responses. We would suggest that the FEAR response itself may establish the necessary ingredients (perhaps the sprouting of AMPA-sensitized glutamate receptors in the neurons of the FEAR system in the central nucleus of the amygdala) that may be necessary for the many "silent" NMDA glutamate synaptic inputs (Kerchner & Nicoll, 2008) of conditioned stimuli arriving through the BLA complex to penetrate, as learning proceeds, the headwater of the FEAR system in the central nucleus. This is an eminently testable hypothesis that deserves more experimental attention (but see Rumpel et al., 2005). If something along these lines is eventually confirmed, it would again highlight how the more ancient primary-process emotional networks of ancient regions of the brain have primacy in how higher brain functions work. That is a most "sensible" way for evolution to operate.

Currently, work on the simplest fear-conditioning models is so popular that we anticipate the above hypothesis will be tested soon, by someone (perhaps before this book is published!). We anticipate it will be done by those who take a great interest in the subfunctions of the central nucleus, such as the very recent studies on the different functional neuronal populations of the central amygdala, with the lateral part being required for conditioning, while the actual conditioned responses are driven by neurons in the medial subdivision (Ciocchi et al., 2010) with an abundance of unique neurochemical controls (Haubensak et al., 2010). Perhaps the most interesting, from an immediate intervention perspective, is the discovery that subsets of neurons are controlled by oxytocin and vasopressin, with oxytocin generally reducing fearfulness while vasopressin elevates it. Because female brains contain more oxytocin neurons than males, and male

brains contain more vasopressin neurons (Panksepp, 1998a), this seems to suggest that there are female-male differences in fearfulness at the very headwater of the FEAR system (Huber et al., 2005; Viviani & Stoop, 2008). Indeed, oxytocin enhances the power of traditional benzodiazepine antianxiety agents such as diazepam (Viviani et al., 2010). This suggests that intranasal oxytocin may eventually have some role in psychotherapeutic interventions that are related to anxiety as well as other affective disorders such as post-partum depression.

In summary, let us focus on some of the key conceptual issues (as outlined by Maren & Quirk, 2004). The plasticity seen in the BLA is truly associative, because simple sensitization—repetition of the shock—does not elevate neuronal firing in this nucleus. Learning only occurs when there are associated predictive stimuli (e.g., tones) that are paired with the unconditioned responses of the brain. The learned plasticity in the BLA depends not at all on the cortical processing of sound. Conditioning proceeds completely without the auditory neocortex. It also proceeds without any prior learned changes in the MGN that might send conditioned sound information down into the FEAR system. As far as we know, the conditioning occurs first in the amygdala itself. However, the eventual plasticity that occurs in the thalamus (the MGN eventually exhibits conditioned responses also) does seem to depend on the conditioning that had already occurred in the amygdala. The BLA seems to "instruct" the thalamic MGN area from which it receives fearful information. We suspect the same can be said for the FEAR system itself: In some currently unknown way, the evolutionary memory of the FEAR system instructs the BLA-central amygdala linkage to condition.

Finally, let us briefly consider whether conditioned fearful behaviors are absolutely essential consequences when neurons in the BLA have been conditioned? Surprisingly, that is not the case. In well trained animals, the neural conditioning in the BLA can be dissociated from fearful behaviors. In other words, one can measure conditioned neural responses within the BLA without the animal exhibiting any fear. This can be achieved by putting NMDA-receptor blockers into synapses where BLA information can be blocked from getting into the central nucleus (the uppermost structure of the instinctual FEAR system). Thus, the conditioned neuronal responses are still evident in the BLA, but animals remain behaviorally (and hence we assume affectively) relaxed during the presentation of previously

fearful stimuli (Maren & Quirk, 2004). Our interpretation is that for animals to experience learned fearfulness, the conditioned information must first get into the FEAR system itself. And there are many higher processes that gain access to such primary processes (i.e., the "royal roads" to emotions—the primary-process affective systems). For instance, in addition to responding to discrete conditioned stimuli like shock-predicting tones, FEAR-conditioned animals also respond to a number of complex *contextual* stimuli about the general environments in which they were hurt or simply scared without being hurt (e.g., see Figure 6.1).

THE CONTEXTUAL CONDITIONING OF FEAR

We are not just afraid of fear-inducing things in the world, but we also fear places that are dangerous. Thus, in real life, and in most experimental conditioning situations, there are several concurrent ways for fear-associated stimuli to get into the FEAR system. Another road to conditioned fear is via the hippocampus, which processes contextual stimuli, such as all the other cues of a scary place besides the discrete auditory tone. For example, LeDoux's rats became afraid of the chambers in which they were tested, including perhaps the steel bars used to deliver the foot shock, and the smell of sawdust on the floor. If the cage had been next to a window with bright curtains, the rats might begin to freeze at the sight of all curtains. They might also feel afraid when they heard the lab assistant's footsteps as she approached to remove animals from their home cages to undergo the experimental paces. From our own work, we know that rats will commonly exhibit 22-kHz alarm calls when a researcher is approaching who might take the animal to experimental situations that they do not like.

Although the hippocampus is not essential for the creation of many emotional memories, it is now clear that hippocampal involvement is necessary before contextual stimuli are sent to the "royal road" of FEAR that begins in the central amygdala. This is because the hippocampus, in addition to playing a crucial role in the creation of declarative memories, also computes spatial orientations, and it provides information about the context in which the conditioning has taken place. This contextual information is also transmitted directly from the hippocampus to the lateral amygdala to arouse the FEAR system (Ehrlich et al., 2009). To reiterate,

when an animal is conditioned to a very discrete cue as discussed earlier—a specific conditioned stimulus—the learning occurs by a different MGN-to-amygdala pathway.

Conditioning broadly, in response to contextual stimuli, obviously increases the adaptive value of learning. However, this occurs via slightly different pathways in the amygdala, namely more basal nuclei than the more dorsal ones that receive input from the MGN. However, the neurobiological principles of learning—the molecular changes in neuronal machinery that generate cue and contextual conditioning—remain very similar (as they do for all other types of basic emotional learning in the other primary-process systems). In other words, increased efficacy of glutamate transmission (acting on NMDA receptors) is as critically important for contextual fear-conditioning as it is for cue conditioning.

THE AFFECTIVE STRESS OF FEAR CIRCUITRY: DO RATS FEEL FEAR?

LeDoux's conditioning research focuses on the way that learned emotional behaviors are acquired rapidly along the low road—this is how implicit emotional learning happens. However, he and most other fear-conditioners do not acknowledge that there is also an evolutionary "royal road" to the experience of raw FEARfulness, a very aversive BrainMind state. As even FDR understood, the only thing we have to fear is FEAR itself. Much as we admire the scientific finesse of these conditioning experiments, we part company with LeDoux and many of the others who conduct this kind of work when it comes to understanding what emotional feelings really are. This is because they studiously ignore the feelings of their animals, and they often claim that the existence or nonexistence of the animals' feelings is a nonscientific issue (although there are some signs of changing sentiments on these momentous issues). In any event, as we noted in Chapter 2, LeDoux has specifically endorsed the read-out theory—to the effect that affects are created by neocortical working-memory functions, uniquely expanded in human brains. In other words, he sees affects as a higher-order cognitive construct (perhaps only elaborated in humans), and thereby he envisions the striking FEAR responses of his animals to be purely physiological effects with no experiential consequences (LeDoux, 1996, p. 302).

Modern brain imaging of fearfulness in humans has yielded many interesting facts about the amygdala, but one fact stands out: When investigators have imaged fears that are still rather remote from those that are immediately dangerous, the amygdala tends to light up. But when an experimental "predator", so to speak, is right on your heels, ready to bite (namely finger shock), the lowest parts of the FEAR system, the PAG of the midbrain, will light up (Mobbs et al., 2009). This brain region is the epicenter for FEARful feelings and behaviors (Brandão et al., 2008; Panksepp, 1998a). And when we analyze the punishing properties of electrical stimulation here in animals, we get the strongest aversive responses imaginable at the lowest levels of brain stimulation, and humans experience the most fearful states of mind imaginable (Graeff, 2004).

Such issues of affective experience should haunt fear-conditioners much more than they apparently do. We believe that in this day and age, it would be wise for us to conceptualize the issue of raw affective experience as being quite distinct from the question of whether animals are "self-aware" of what is happening to them with enormous worries about the future, which would indeed require working memory. The evidence strongly indicates that there are primary-process emotional networks in the brain that help generate phenomenal affective experiences in all mammals, and perhaps in many other vertebrates and invertebrates. These are neuronal systems for some of the most important "unconditioned responses" of the MindBrain that neuroscientists who are interested in understanding learning must routinely use in order for animals to learn as readily as they do. We believe the neural systems that generate such experiences—the various rewards and punishments of the BrainMind—are of critical importance for generating learned affective memories that psychiatrists and psychotherapists must deal with in their daily work. This knowledge about primary-process emotions is also allowing us, for the first time, to understand how emotions influence higher mental processes.

In closing this summary of fear-conditioning, let us be clear: Only neuroscientific study can truly clarify what is going on in the brain and how mental experiences are created. The mind is instantiated by complex brain processes that operate in living bodies, usually in complex worlds. Still, while the behavioral analyses proceed without much scientific controversy, the mental analysis currently seems to be little more than a source of momentous controversy. We think this is only because behavioral

techniques work so well, and most animal investigators feel that they do not have to consider mental constructs, because they may be figments of our imagination. What affective neuroscience has sought to do through the identification of key brain systems that help create primal emotional feelings, through the rewarding and punishing properties of various brain emotional systems, is to provide an evolutionary strategy for understanding the foundations of our emotional mind. The genetically ingrained emotional systems of the brain reflect *ancestral memories*—adaptive affective functions of such universal importance for survival that they were built into the brain, rather than having to be learned afresh by each generation of individuals. These genetically ingrained memories (instincts) serve as a solid platform for further developments in the emergence of both learning and higher-order reflective consciousness.

A HISTORICAL EXAMPLE: IMPLICIT EMOTIONAL LEARNING AND MEMORY

Let us share a story about the supposedly unconscious nature of fear; this story is well known and oft-recited by neuroscientists who assume that the FEAR-learning described above proceeds without fearful feelings. The phenomenon of "implicit emotional memory" was famously demonstrated in 1911 when a French physician named Edouard Claparede worked with a female patient whose hippocampus on both sides of the brain (areas needed to translate short-term memories into long-term ones) had been damaged. As one would now expect, the brain of Claparede's patient was incapable of creating any lasting declarative-episodic memories. Thus she forgot everything as soon as it passed out of her working memory. Every time she saw Claparede, he was obliged to introduce himself to her as if they had never met.

It was the custom to shake hands upon meeting, and because the patient could retain procedural habits she was able to participate in this social ritual. One day, Claparede concealed a pin in his palm, puncturing her finger when they shook hands. The pain startled her but the wound was superficial and soon healed. Of course she forgot all about the incident. Yet when Claparede met his patient again, she refused to shake his hand. She could not explain why she was so disinclined, and she made the sort of excuse that amnesiacs commonly do to cover up their inability to recall

events. (She said, “Does a lady not have the right to withhold her hand from a gentleman?”) Claparede’s patient could not have consciously remembered that he had inflicted injury on her at their last meeting. Her hippocampal damage was to both sides of the brain, so she was incapable of generating a declarative memory of the event.

Nevertheless she had learned something from the pinprick. Previously she had not been afraid to shake Claparede’s hand. Shaking hands had been a neutral, possibly even a positive, experience. However, after receiving the pinprick, the patient acquired—learned to have—a negative emotional response to a previously neutral stimulus, namely the doctor’s outstretched hand and perhaps to the doctor himself, even though she might not have been able to say why. The acquisition of a new affective response to a neutral stimulus constitutes emotional learning. And the retention of that learned response is an emotional memory. This example also nicely highlights that affective experiences are very different in the brain than declarative cognitive ones. Bilateral damage to the hippocampus eliminates the latter but not the former. This example also highlights how affects can be completely independent of cognitions. We expect that had Claparede, or any of the modern scientists who have done such work, carefully asked their patients how they felt after such shenanigans, they would have said that they were a bit more scared of the investigator, without knowing why (something that might only be retrieved with psychoanalytic interviews by clinicians who understand that affect can exist independently of related learning and cognitions).

AFFECTIVE FORCES GUIDE MEMORY FORMATION

The power of emotions to determine how we behave, as well as what we perceive, think about, and remember, is a remarkable quality. Emotions make us actively reach out and engage with the world, both with our bodies and our minds. For this reason we prefer to envision emotional systems as “attractor landscapes” (in the lingo of nonlinear dynamic systems theory) that help us to make particular connections with our environments both in thought and in deed. Thus, we envision primary-process emotional systems to be in the “catbird seat”—having the upper hand—when it comes to how learning controls the formation of memories in our brains. This is by no

means a traditional viewpoint in the BrainMind sciences, largely because neuroscientists generally neglect the feelings of laboratory animals. Many neuroscientists have yet to understand the affective nature of the many "unconditioned" processes of the brain (instances of UCS, e.g., painful shocks, and UCRs, e.g., very scary FEAR arousal) that they use to provoke "conditioned fear responses" (e.g., freezing and flight) that can be used so effectively to study memories in animals. But the neutral conditioned stimuli that they pair with their various instances of a UCS may only work so well because the associated UCRs (instinctual emotional responses of the nervous system) are also "dripping" with the neurochemistries of affect.

Indeed, there is an intrinsic "memory" process that transpires completely as a result of repeated emotional arousals accompanying primary-process UCRs—intrinsic affective memories that can wreak internal havoc with the way we think. As noted earlier, and as we will see over and over as we proceed, each of the emotional systems can become stronger (sensitized) and weaker (desensitized) through repeated use or prolonged disuse. Such chronic changes in our internal affective mindscapes allow us to understand how repeated early emotional experiences (traumas) can be formative for the long-term affective qualities of adult minds, qualities we often call temperament or personality traits.

The remarkable thing about modern neuroscience is that most investigators feel that cognitions, much more than our affects, guide the directions of our mental lives. That is partly the case because our neocortex is so vastly capable of developing knowledge structures, where we seem to live most of the mental moments of our lives. However, when we look at them closely, many such views are just beliefs that teeter between being rational and delusional. The illusion of the fully "rational animal" (for a fine analysis, see Fogelin, 2003) may be largely due to the fact that we are the only species that has a language that can construct and convey complex ideas and delusions. Many believe that we could not be fully conscious without language, but much hinges on our definition of "fully". But several philosophers and neuroscientists, from David Hume to Antonio Damasio, feel that our emotions energize and guide our cognitive processes. We are among them. Language is the most recent of lasting human brain developments, and it is constructed and guided as much by culture as biology.

The neural mechanisms that typically elaborate language allow us to communicate cognitively with others (i.e., they are acquired tools that are adept at linear processing of information). Such mechanisms are typically concentrated in the left cerebral hemisphere, which is generally less emotional than the right hemisphere. In contrast, the more holistically and more emotionally astute right hemisphere sustains and elaborates the affective aspects of life with little explicit cognitive consciousness, perhaps because it views life affectively and holistically. For instance, it adds prosody—the emotional melody or affective harshness—to our voice.

Of course, it would be foolish to deny the importance of cognitive activities in mental life, but a great deal of one's rationale for viewing the world a certain way arises from one's feelings. Just think of the convictions that we have—our strange, strongly held beliefs. The apparent coherence of many cognitive viewpoints would soon degrade without the affective and attentional "energies" emanating from the emotional networks concentrated below the neocortex.

For this reason, it is also important to realize that higher brain regions can be trained to regulate emotional arousals, and this is one of the great achievements of healthy mind maturation (Goleman, 2006). Consider once more the wisdom of Aristotle, in the epigraph to Chapter 4. Although cognitions and emotions remain conceptually blended in higher regions of the brain, mental health arises when this interpenetration is substantially—gently and with equanimity—regulated from both below and above. This is why cognitive-behavioral and mindfulness therapies remain mainstays of the psychotherapeutic landscape. One goal of psychotherapy, with its ability to reframe emotional trouble zones, is to help refine such skills. Another is to understand the emotional nature of our mental lives. Much of psychotherapy is one human being helping another to understand the furies of our minds, and to be more at peace with what Mother Nature and Father Nurture (or the lack thereof) has wrought in our individual lives.

GENETIC MEMORIES: BEYOND TRADITIONAL MEMORIES TO BRAIN NETWORK SENSITIZATION AND EPIGENETIC MOLDING OF THE MINDBRAIN

Only a few decades ago, scientists believed that each human being was born with about 100,000 genes. We now know that each person is born with only

about 22,000 genes. This is surely sufficient to create the seven basic emotional systems with which we are all endowed. Such ancestral BrainMind memories also include various attentional and motivational mechanisms, which we will not focus on, that are controlled by many, many brain chemistries. However, it is important to recognize that there are not nearly enough genes to account for the variety and subtlety of our MindBrain functions, but there are quite enough to get infant animals and people well on their way to acquiring the endless features of adult minds, most of which are created by learning. In adults, the array of personal characteristics, not to mention individual patterns of thought and specialized areas of knowledge, can only have emerged with our massive capacity for learning and memory described above. These characteristics are anchored to stable sets of emotional skills and temperaments that we discuss in this book.

However, even these emotional traits and personality dimensions are solidified by our experiences in the world. There are many strengths and weaknesses that we inherit directly from our parents, but much also emerges from the genetic changes, after birth, from how we are reared—namely, the *epigenetic* moldings of brain networks that result in various patterns of sensitization and desensitization in the primary-process emotional and motivational networks of the brain. These long-term environmentally molded genetic memories have only recently moved to the forefront of developmental thinking (for a fine recent overview of such effects before birth, see Paul (2010).

Epigenesis describes the ways that experiences can change gene expression patterns to allow for the creation of many individual traits. Epigenesis is not a mutation, which is a physical change in the genes with which we are born. Epigenesis is an experience-dependent change that occurs to genes, typically after we are born. One of the epigenetic ways in which genes can change is through the variation in the degree of gene expression. All cells of the body have the same genes, but in each cell only some of the genes are active or "expressed." When previously dormant genes become active, we say that gene expression has occurred. Gene expression results in the production of new proteins. When the strength of gene expression is environmentally either decreased or increased, we call it epigenesis.

Chemically, epigenesis arises from changes in chromatin, the supportive substance that surrounds genes. The chemical processes of acetylation and methylation can change the three-dimensional structure of chromatin, and this can give transcription factors access to genes (see Szyf et al., 2008, whose work on the epigenetic effects of maternal CARE on infants' brains we will discuss in Chapter 8). When transcription factors have access to dormant genes, the rate of gene expression can be ramped up or down to produce proteins at new levels. Another way for Mother Nature to nurture different life trajectories is through the generation of small regulatory gene-controlling snippets called "micro-RNAs," which can also help control how the genetic orchestra plays at different points in life. The changing amounts of protein produced as a function of our experiences and of unfolding gene expression often play crucial roles in the creation of the new neural pathways. Some of these pathways help encode new skills, knowledge, and personality traits.

When any of the seven subcortical emotional systems is aroused, they will in turn arouse parts of the neocortex. This sometimes involves epigenesis and the creation of new neural pathways within those most plastic regions of the brain. The epigenetic developments and specializations of the neocortex may be fundamentally dependent on how subcortical attentional, sensory, emotional, and motivational functions help to weave new patterns of connections. At the subcortical levels, other epigenetic processes may help mold the maturation of emotional systems, yielding life-long emotional strengths and weaknesses that determine the affective personality of animals and humans for a lifetime.

As we have noted, below the neocortex most people—most mammals—are remarkably similar in emotional kinds, notwithstanding species-typical differences in the vigor of each emotional network, and the amount of neocortex they can influence. Many of the detailed epigenetic refinements of our characters take place in the neocortex. However, epigenesis also helps to explain how our core (subcortical) emotional temperaments are refined developmentally—how we become the kinds of individuals we are because of the social and physical environments in which we find ourselves. Epigenesis, along with learning/memory, helps us understand why we are able to have such complex and variable personalities, skills, and funds of knowledge, even though each of us is born with only ~22,000 genes. In addition to the traditional mechanisms of learning and memory,

the differential intensities and patterns of gene expressions in different regions of the MindBrain allow each individual's limited gene pool to become diversified by experience.

SUMMARY

In this chapter we have tried to convey a sense of what it means to learn and remember. For the most part, learning and memory are automatic and involuntary responses (mediated by unconscious mechanisms of the brain), but in almost all cases learning and memory, in their most lasting forms, are commonly tethered to emotional arousal. Our everyday working memory commonly tends to focus on things that are emotionally meaningful. More often than not, episodic and autobiographical memories concern emotionally meaningful aspects of our lives. Eventually researchers may be able to reveal a molecular and chemical sequence that starts with emotional arousal and ends with the creation of lasting, highly personalized affective episodic memories that influence how we are temperamentally as unique individuals. By saying this, we are not suggesting that there are not some kinds of learning and memory that occur without affective arousal, but we do not know of any great examples of that kind, excepting rote memory (the stuff we hated in school, which progressed especially slowly, no doubt, because of boredom) and LTP-type procedural memories.

Clearly, emotional arousal is a necessary condition for the creation of FEAR-learning memories—for example, freezing responses to previously neutral stimuli—because animals can only be conditioned if the training procedure evokes affective arousal. Emotions, especially SEEKING enthusiasms, also surely motivate animals to learn procedural skills. Generally speaking, these skills require repetitive practice, but we need to be motivated in order to practice. However, procedural memories are the only exceptions to the rule that affective arousal is very important for the retrieval of memories, because they appear to persist even when people and animals are emotionally calm. Mothers can efficiently go about the business of nurturing without feeling especially moved. Although the retrieval of procedural memories may not require emotional arousal, it plays a clear role in affect regulation as well as in the regulation of the affects of others. When children are polite, this makes other people like them. This is a positive experience for all children. And when a mother is able to nurture

effectively, even when she is not in the mood, this regulates the affects of her children.

We do not understand the cellular and molecular details of the cognitive mind, but the neuroscience of classical conditioning (as discussed above) has revealed a series of important facts about the interconnections between the emotional and cognitive substrates of the brain. Numerous experiments have confirmed the effect of emotion on the way we learn and the way we think, partly through the way our working memory operates (Davidson et al., 2003; Lewis et al., 2008). This research indicates that emotional arousal largely determines the kinds of things that are naturally juggled in working memory—the kinds of items that we consider when we are trying to make sense of our lives. How this occurs is not at all clear, but there are simple possibilities. The FEAR system, for example, may arouse working memory because many associated neural pathways (such as those fueled by the neurotransmitters acetylcholine, glutamate, and norepinephrine, which control the intensity of attention) are aroused by the FEAR system and are projected into relevant areas of the neocortex that mediate working memory (LeDoux, 2002).

Emotional systems other than FEAR can also direct one's thoughts and attention in similar ways, but to very different concerns. A new mother's CARE system will render her highly attuned to any sign of distress in her baby. While relaxed, and dwelling on the sweetness of nurturing, a mother might muse about adorable outfits to buy or ponder over endearing photographs. Anyone in the grip of an aroused LUST system will find that his or her thoughts and perceptions center on sexual situations and amazing permutations of possibilities. When RAGE is aroused, we are apt to ruminate on vengeful thoughts and all too easily find cognitive excuses (rationalizations) for our flare-ups. When our PANIC/GRIEF system is aroused, we search for a friendly face and think with longing feelings about happy reunions. Our PLAY system can help us become cognitive jesters, with clever remarks to delight those who enjoy the play of the higher mind. And the SEEKING system, which is aroused to some degree each waking moment of a normal life, provides us with a great capacity to learn about the world when the other more discrete emotional systems are quiescent.

Considering these relations between affective experiences and learning, it will be very important for psychotherapists to consider troublesome memories in deeper neuroscientific contexts, especially because

reprocessing and reconsolidation may open up new and better opportunities to work with the troubling affective aspects of certain long-term memories (Panksepp, 2009a). Further, considering that affectively loaded memories are spontaneously remembered more readily than nonemotional ones, it is possible that people who dwell endlessly on their vexing problems may only make matters worse. This highlights the importance of others, who are able to listen and respond sensitively and positively, to help reframe the memory in more beneficial affective perspectives. Indeed, one can now imagine totally new ways to soften the disruptive impact of traumatic memories in people's mental lives (see Chapter 12).

The therapeutic implications of all the above knowledge are vast. These facts seem to highlight general principles about emotional learning and perhaps how they relate to associated psychopathologies (e.g., generalized anxiety disorders). Additional layers of emotional learning are even more broadly represented in higher regions of the brain, potentially in highly idiosyncratic ways, which may be what makes them so hard to manage therapeutically. But ultimately the practical value of all this fine work on fear-conditioning is that it gives us a solid understanding of how pathological emotional distress may arise in the MindBrain through specific life experiences. Understanding such mechanisms opens up new avenues for how fearful memories can be extinguished, reconditioned and recontextualized. A great deal of work is proceeding on those issues, and in general most investigators now realize that "extinction"—the loss of a learned response through a lack of "reinforcement"—is also an active learning process. And this undoing of the influence of past aversive memories and reconsolidation (through new learning) can be facilitated by drugs, such as d-cycloserine, which are learning facilitators (for thorough discussions, see Davis et al., 2006; Myers & Davis, 2007). Whether such drugs work by promoting reconsolidation—emotional memories being modified through new affective contexts and re-learning—rather than by erasing old learning, is not yet certain.

This information may be useful and enlightening for psychotherapists because it explains how emotional learning transpires in human lives. All animals, including humans, are massively conditioned in the course of life, yielding various secondary- and tertiary-process emotional phenomena. When conditioning circuitry becomes memory circuitry, especially autobiographical circuitry, the complexities of episodic tertiary-process

emotional memories and thoughts emerge, melding with the continual flow and endless complexities of culture. Reciprocally, when the cortical aspects of the mind begin to trigger intense and often idiosyncratic emotional responses, one had better understand the dynamics of individual lives. This is why the therapeutic conversation is so meaningful. When people talk about their concerns, they become emotionally aroused and interpersonally engaged, and this in turn arouses further aspects of declarative and episodic memories, providing the "fuel" for new working memories. Thoughts and emotions snowball in a cascade of ideas and feelings that characterize the therapeutic conversation. If well used, this conversation can be the basis for lasting affective change—a profound BrainMind plasticity (Doidge, 2007)—that we do not quite yet understand at a fine neuroscientific level. But remarkable progress has been made.

CHAPTER 7

LUSTful Passions of the Mind

From Reproductive Urges to Romantic Love

Had we but world enough, and time,
This coyness, lady, were no crime.
We would sit down and think which way
To walk, and pass our long love's day; . . .

A hundred years should go to praise
Thine eyes, and on thy forehead gaze;
Two hundred to adore each breast,
But thirty thousand to the rest;
An age at least to every part,
And the last age should show your heart.

—"To His Coy Mistress," written in 1651–1652 and published in 1681 by Andrew Marvell

HOW EROTIC FEELINGS, FROM PRIMAL lust to tender love, are created within the human brain remains one of the more important yet least understood scientific problems in the mind sciences. There are abundant theories but little consensus. In contrast, we know a great deal about the sexual circuitry of the brain in rodents. With such animal models, where the details of some of the most intriguing questions about primal LUST can be worked out, we have some hope of illuminating the general principles that underlie human

lustful love. The implications of the animal findings for understanding human sexuality (Figure 7.1) are enormous.

The LUST system lies at the very fulcrum of our attempts to understand basic mammalian physical drives (sexual affects) on the one hand and social emotions on the other, conjoined twins as they are. The primal urge of LUST makes social life exquisitely intriguing because sexuality is a primary motive in the lives of all mammals, indeed all creatures that have any possibility of illuminating the human condition. But sexual feelings do not anticipate our own survival concerns, but rather the survival of our familial genes into future generations. Erotic gratifications play no crucial role in the survival of the individual having the experiences; they only serve the survival of the species . . . or so the story goes. It is likely, however, that a satisfying sex life promotes a competent immune system and longevity, just as physical exercise does. Although sexual gratification may not be an immediate aid to survival, well-bonded, sexually satisfied people often live longer than those without the security of happy relationships, whether their pairings be man with woman, man with man, or woman with woman. The blessings of fulfilling loving relationships, with sexual satisfaction as a solid component, seem to be a tonic for body and soul, allowing many humans to live much longer *beyond* their reproductive years than is the norm for other species. But much of this also comes at a cost: Women and men are typically of different minds when it comes to issues about what is important and satisfying when living together. Books are continually being written about such issues, ranging from *Psychic War in Men and Women* in the 1970s (Lewis, 1976) to advice in *Men Are From Mars and Women From Venus* at the end of the century (Gray, 1992) to *What Could He Be Thinking? How a Man's Mind Really Works* in this past decade (Gurian, 2004), highlighting what women should know about the relatively diminutive affective lives of many men as compared to many emotionally well-endowed women. Yes, male brains and female minds have some amusingly painful distinguishing characteristics. Thankfully, our coverage here will focus mainly on rats, but we will always keep an eye toward the human condition.

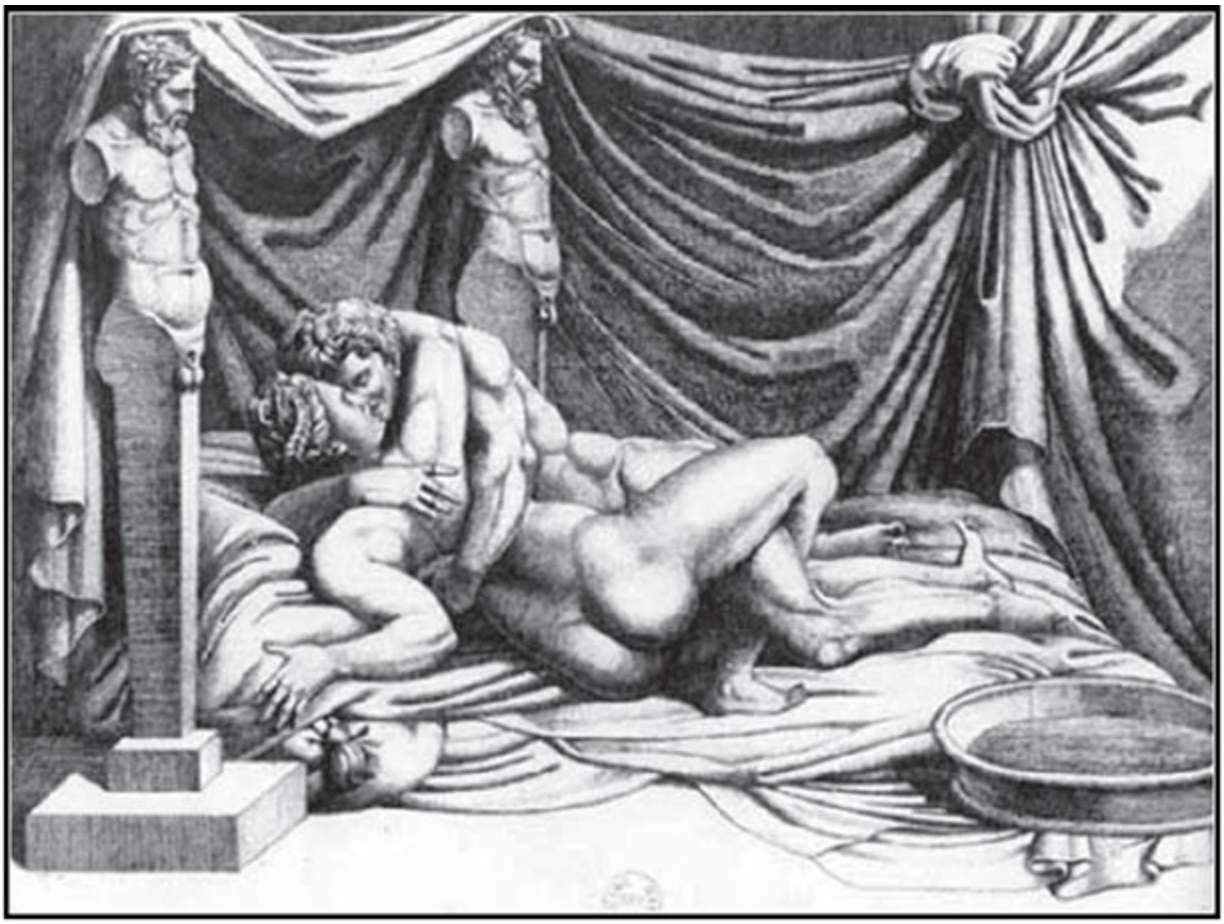

Figure 7.1. An erotic engraving by Marcantonio Raimondi (1524) from original paintings by Giuliano Romano, Raphael's talented 25-year-old pupil. This print is supposed to be the only surviving image from one of the earliest collections of erotic art—the "I MODI" series. In 1524, Pietro Aretino wrote sonnets to accompany the drawings of 16 sexual positions by Romano, which is considered to be one of history's most notorious works of erotic art: Aretino's "Lusty Sonnets." This work was compiled into a printed edition with Raimondi's splendid engravings. All except a very few copies of this book were promptly burned by the Church. No surviving complete copy is known to exist (from http://www.artarchiv.net/doku/museum/Aretino.htm).

So what does it mean to be a sexual man or woman? What is natural (or normative) male and female sexuality? Surely the language and the labels we use to refer to the varieties of sexual identity today have expanded along with our cultural awareness of the "natural" variety that exists in humans.

But there is little agreement about words and labels when we actually know much less about human sexuality than that of rats. This uncertainty, at times, may be a source of discomfort and disagreement, as the culture wars regarding the substance of gender and sexual identities continues to be waged. What does it mean, for instance, to have intersexual identities where supposedly normative male and female psychological identities and behavioral tendencies intermingle with bodily appearances and at times barely coexist? What does it really mean to be transgendered, transsexual, homosexual, or bisexual? How is one's "gender role" or "identity" created through the intermixtures of biology, culture, and personal choice?

These concepts are just a sampling of semantics in a confusing, cacophonic grouping of debates about our sexual heritage. Such debates are part biological and part politicized culture—this creates a noisy landscape in which more credence frequently is given to poorly understood language than to the biological facts that undergird our sexuality. In this chapter, we endeavor to use the terminology of sexual and gender identity in relatively straightforward psychobiological and behavioral ways. Admittedly, our usage will not necessarily fit in all respects with usage in current cultural discourse (as if such a thing were even possible). But as we attempt to stick as closely as possible to discussions of primary-process evidence, as worked out in animal models, it perhaps must be the case that our usage of sex and gender terminology will not meet everyone's expectations. We ask that the reader excuse any apparent shortcomings in this respect and join us in this chapter's approach to the remarkable vista on sexuality that has been opened through the explorations of a cross-species affective neuroscience.

At the biological level, some clarity surrounding the substance of sex and gender identities arises because sexual bodily appearance and brain sexual organization may not match up well with one's assumed sociocultural identity. At the biological level, diversity can range from overt psychobehavioral masculinity in genetic females with the typical XX sex chromosomes to the ambiguities of genetically typical XY males who have exquisitely "typical" female body qualities, but minds that feel thoroughly masculine. In fact, people can be situated in a whole range of "mismatching" bodies and minds. When we add to this our higher psychological self-identities, one can be sure that things become infinitely complex, with conceptual rip-tides and cultural cross-currents at many levels, ranging from what we call the primary to the tertiary levels of

MindBrain organization. We will not even touch the culturally and societally influenced "gender role" identity issues that add so many tertiary-process levels of complexity to the primary-process neurobiological and hormonal complexities that will be our focus. Human cultural issues cannot be modeled well in other animals. Nevertheless, many biological features of sexual urges and gender identities have been illuminated by studying our fellow mammals. We think that the implications of this shared biological heritage for the ways we humans and other mammals feel erotically, at primary-process levels, are profound.

This chapter will examine the anatomical and chemical differences between male and female brains. We will also explore some modern neuroscientific research that affirms why LUST circuitries should be deemed a primary-process emotional system, one that is somewhat different for females and for males. Like all other emotional systems, LUST networks link up with various homeostatic and sensory affective mechanisms. For instance, starvation superbly reduces sexual urges, as do fear and most of the negative emotional feelings, even though mild pain can increase sexual arousal at times and not just in sadomasochistic humans (Caggiula & Eibergen, 1969). But mostly, we will delve into some fascinating research about the ways that sexual brains and sexual bodies exhibit relatively independent embryonic development. This independent development goes a long way toward explaining extremes of homosexual/transgender phenomena, including probably milder gradations of psychologically feminine characteristics in genetic (XY) males and masculine mentality in genetic (XX) women. We will also dwell on BrainMind mechanisms that promote intersex desires, those that seem to violate simplistic notions of male and female identity. Along the way, we will turn our attention to some related clinical considerations. Although our knowledge of the specifics of the brain anatomies and chemistries underlying diverse transgender phenomena in humans is limited, we are becoming quite well informed about these issues in animal models. There is burgeoning acceptance, in the scientific community at least, that the biological facts discovered in other animals can clarify related human conditions. As part of this discussion we will also examine some emerging mythology about oxytocin as "*the* love hormone"—which typically has a few ounces of truth as well as, all too often, pounds of exaggeration. The concept of "confidence" may help explain more of the effects of oxytocin

than the concept of "love." We will conclude by considering the gulf that exists between neuroscientific and classical psychoanalytic theories of psychosexual development.

THE BRAIN'S SEXUAL CIRCUITS: WHAT IS THE NATURE OF THE SEXUALLY AFFECTIVE MIND?

Some people still believe that male and female brains are alike and that the sexual preferences of men and women are entirely learned. Modern neuroscience has provided ample evidence to dispel these notions. While it is quite true that each sex has both male and female brain networks, these circuits are not usually of equal strength. So, just as the bodies of males and females are different in some important respects, their brains also are distinct in many ways, with an enormous variety of psychologically nuanced differences (Hoyenga & Hoyenga, 1993). A schematic summary of the underlying sexual circuitry of rats, probably similar across most mammals, is summarized in Figure 7.2.

In male mammals, the epicenter for primary sexual urges is found in the medial regions of the anterior hypothalamus, although the precise brain locations and terminology involved vary a bit from one species to another—in rats, it is the sexually dimorphic preoptic area (POA) and in humans it is the interstitial nuclei of the anterior hypothalamus (INAH) that are surely evolutionarily related. As first clarified by Phoenix et al. (1959) in guinea pigs, and soon extended to rhesus monkeys, the organization of brain sexual circuitry begins during fetal life, ultimately leading young boys and girls to differ in many of their tertiary-process interests. This is substantially controlled by testosterone, which is secreted in infants before and soon after birth. Then in adolescent females, the maturation of ovarian estrogen and progesterone steroid production heralds puberty. And in adolescent males, intense sexual awakening occurs when the testicles begin to produce abundant testosterone. These defining female and male hormones bind with various steroid receptors in many subcortical sexual regions of the brain, especially the anterior portions of the hypothalamus. Pleasurable affects are created by this binding, at least for testosterone. Thus, male rats clearly like to have testosterone injected into the POA of the brain. They will work for it and will revisit places where their sexual circuits were "oiled" with injections of testosterone (King et al., 1999).

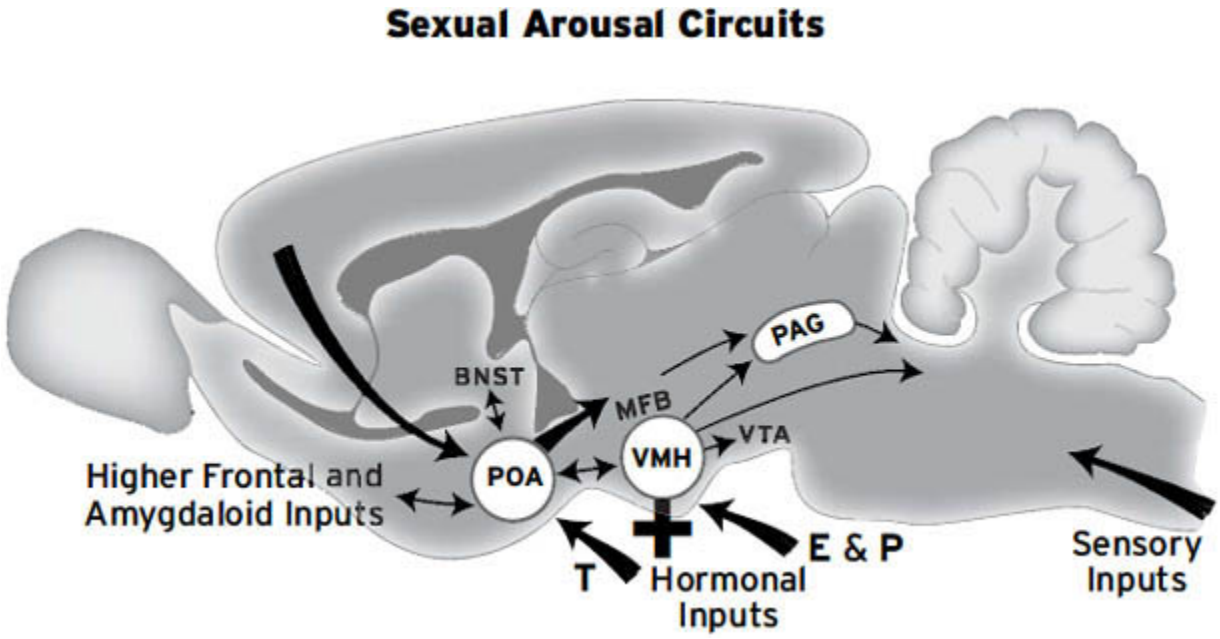

Figure 7.2. Lateral view of the rat brain summarizing major subcortical networks that provide differential control over male and female sexual behaviors. Males contain a larger POA, and this area is essential for male sexual competence. The VMH (ventromedial hypothalamus) is clearly more influential in female sexual responsivity. These systems operate, in part, by sensitizing various sensory input channels that promote copulatory reflexes. The extent to which these circuits control the affective components of sexual behavior remains uncertain (adapted from Panksepp, 1998a; republished with the permission of Oxford University Press).

Such circulating steroids enter the brain easily. A vital principle of neurochemistry comes into play here and should be kept in mind—hormones and other fluid-borne neurochemicals are only half the equation in completing the messages conveyed by these chemicals throughout the nervous system. Most neurochemicals in solution, moving through intra- and intercellular spaces, will be able to carry out their characteristic effects only to the extent that there are corresponding receptor molecules present in the cell membranes, and even receptors within neurons, within relevant areas of the nervous system. Testosterone exerts a greater effect on the male brain because males have larger neuronal fields rich with testosterone receptors in key areas of the anterior hypothalamus—especially the POA. It is almost as if boys had a bigger set of sex glands right within their brains,

corresponding to the more obvious external gonads. Female sexuality, as we will see, has more complex underpinnings, which are not as well understood.

The idea of neuro-symbolic gonads in the male brain is not too far from reality. As has been especially well studied in animal models, damage to the testicles *or* to these key anterior hypothalamic regions produces similar effects. For instance, lesions to the POA dramatically weaken male sexual urges and abilities, especially if the damage occurred before adolescence in sexually naive animals. If young animals lose their testicles before they reach sexual maturity, they will never develop strong sexual urges, unless testosterone is introduced into the appropriate regions of their brains. However, more general social urges are not impaired in such animals, indicating that social needs are not completely tethered to sexual needs. Also, once adult male rats have developed sexual habits, the same damage to the POA does not disrupt male sexuality as severely as it does among inexperienced animals. Presumably, sexual motivation declines more slowly because the animals have developed higher-order sexual habits that have taken on a life of their own (i.e., the motivation was sustained at the secondary- and tertiary-process levels). Through life experiences, sexual motivation has been transferred, but only in part, to other regions of the brain. After POA damage, sexually experienced rats still continue to work for access to receptive females, but their sexual follow-through is rather sluggish. These lessons probably apply to humans too, but the relevant scientific data (particularly relevant brain data) run thin. Sexually active men who lose their testicles tend to sustain an erotic life longer than castrated animals, but a gradual decline in motivation is inevitable. In other animals, the decline is generally much faster (Meston & Frohlich, 2000), presumably because they have fewer higher-brain mechanisms to sustain motivation.

Why is testosterone so powerful in engaging male libido? A variety of neuropeptides that are activated by testosterone surely play a role. The best studied is vasopressin, which in animal models promotes sexual ardor, courtship, territorial marking, intermale aggression, and possibly sexual jealousy (Goodson & Bass, 2001; Hart & Legerstee, 2010). Whether the rewarding effects of testosterone in the brain are due to the facilitation of vasopressin circuits is not known. In any event, males have twice as much vasopressin as females do. Testosterone also activates a gaseous transmitter

in the brain, nitric oxide (NO), which promotes heightened sexual eagerness as well as male-typical heightened aggressiveness—a well-established testosterone dependent form of "offensiveness" (Nelson et al., 2006). The current crop of male sexual performance-enhancing drugs, such as Viagra, can improve erectile function more consistently than any aphrodisiacs of the past because they elevate production of NO in both brains and penises, perhaps in clitorises too. Once again, we see how body and mind work together, under the sway of similar chemicals.

Testosterone and Male Aggression

In addition to facilitating male sexual responses, testosterone also plays a crucial role in generating the male's aggressive urges for social dominance. It has now been established that male sexuality and the assertive urge for dominance interact to a substantial extent in the subcortical areas of the brain, and it is also clear that this interaction is largely rooted in high levels of testosterone. In other words, testosterone fuels circuits that promote male sexuality as well as the many brain mechanisms that promote the urge for social dominance. As we noted in Chapter 5, testosterone can also sensitize the RAGE circuits of the brain. Females, whether humans or rats, react in similar ways when given testosterone—they become more assertive and self-confident, along with a heightened suspiciousness of others (van Honk et al., 2004). Presumably females are generally more trusting (or socially confident), because of their elevated oxytocin levels, while the testosterone influence on males, who are more liable to indulge in aggression and more subtle dominance displays, also promotes a greater suspiciousness of others' motives. In mouse models of sexuality, certain smell inputs into the female brain tend to inhibit the circuitry in their brains that might otherwise promote typical male behaviors (Kimchi et al. 2007).

However, although testosterone is essential to both male sexuality and the intensified aggression commonly exhibited by males, it is unlikely that the urge for social dominance is simply identical with the male sexual urge. There are many reasons to believe that sexuality and aggression are separable, albeit interactive, systems in the brain, especially in subcortical regions such as the medial amygdala. Some neurons in the amygdala respond only to sexuality, others to aggression. However, there are also many neurons that respond to both sexual and aggressive situations. In a

similar vein, research in humans indicates that temporal lobe areas (where aggression circuitry is concentrated) are more active in males, while the anterior cingulate cortical areas (where nurturance and social pain circuitries are concentrated) are more active in females (Gur et al., 1995).

When male sexual desire has no satisfactory outlet, it can yield disquieting feelings of tension, some of which take organisms onto the field of competition and various aggressive encounters. Statistically, males tend to physically abuse females more than the other way around. This is partly because testosterone invigorates aggressive urges for social dominance in the mind. Indeed, the crescendo of peripheral sexual hormones in adolescence can promote enormous sexual frustrations if primitive lusty urges are not satisfied, as they so often are not in civilized societies. Surely such internal forces lead to sexual aggression and to other socially inappropriate advances.

We have learned a great deal about our sexual passions by studying other animals. For instance, some of these gender-related temperamental differences are due to the way that sex hormones influence brain chemistries: Estrogen "fertilizes" oxytocin neural systems in the female brain, while testosterone increases the power of vasopressin in the brains of males. As we will discuss below, oxytocin exerts a calming effect on the brain, and this appears to facilitate the formation of positive social bonds in both males and females. Vasopressin, on the other hand, tends to induce competitiveness in males, but it can also increase sexual bonding and defensiveness (jealousy?) in them, while in females it typically reduces sexual eagerness. As we will see in subsequent chapters, these sexually distinct neuropeptides control many nonsexual social behaviors in mammals (Goodson & Bass, 2001). The avian peptide, *vasotocin*, combines both oxytocinergic and vasopressin functions, which, along with endogenous opioids, strongly control social motivation in birds (Panksepp, 1982), especially birdsong, a long-established sexual-territorial response (Riters, 2011).

Much of the evidence about male aggression and these peptides is obtained from studies with birds, in particular from research on the effects of avian vasotocin, which differs chemically from both oxytocin and vasopressin by only a single amino acid. Vasotocin apparently calms emotional distress and promotes peacefulness. Indeed, we have found that vasotocin can reduce male dominance behaviors without modifying their

apparent dominance feelings. As we noted in chapter one, a rather peculiar finding we observed in male pairs of quail illustrates this point (Riters & Panksepp, 1997). Male quails, when first introduced to each other, will peck each other's heads rather vigorously in apparent attempts to resolve dominance relationships. But when vasotocin was given to one of the males, directly administered into the ventricles of the brain, the bird showed little reciprocal aggression, taking each nasty peck in apparent stride. Remarkably, when these formerly peaceful, "aggression-accepting" males were then given a control solution, they promptly started to peck their presumptive "masters," turning the tables.

This outcome is really rather strange! Under normal circumstances the animal that submits to the pecking is the submissive one. So we assumed that vasotocin induced submissive tendencies—and we expected these tendencies to endure. The vasotocin together with the earlier pecking, however, failed to make the quails truly submissive. Why then did the quail that had been treated with vasotocin submit to being so viciously pecked? Perhaps the vasotocin engendered a sense of confidence and peacefulness that served as a kind of affective "protection," preventing the previous social assaults from consolidating into sustained submissive attitudes. Once we are in the intellectual position, through such basic cross-species emotional neuroscience research, to consider the affective changes that neurochemistries can create, many novel ways of looking at behavioral control begin to emerge.

In many animals, the vigor of male sexuality and the imperative of male assertiveness (i.e., social dominance behavior) converge in the complex rituals of competing for mating rights. For example, in many herd animals the dominant male is the only one that has the prerogative to mate with the females. Also, in many animals males have telltale signs of reproductive fitness that females focus on when choosing a mate. Prominent examples include the luxuriant tail plumage in peacocks, face coloring in mandrills, and chest-patch coloring in Gelada baboons (almost symbolizing that one's heart lies open to female attention). Female receptivity is signaled by many factors, such as an especially attractive smell or by eye-catching color, such as the blushed swellings on chimpanzee behinds. Indeed some human male-female differences, especially among individuals with schizophrenic tendencies, highlight issues of psychiatric importance (Goldstein, 2006),

which may give new meaning to understanding split personalities. Such psychic aspects are bound to be unique in human beings.

We humans see ourselves as being sophisticated in our sexual preferences. But males are easily attracted to young and beautiful women. Female beauty is certainly related to a variety of facial and bodily characteristics, with the most notorious one from evolutionary psychology being the mathematics of the "hourglass" figure, with a waist to hip ratio of 0.73 that hits "the spot" for many males (Singh & Randall, 2007). Where is that spot? Perhaps not in the POA, but in the higher-order emotional perceptions of the visual system. There is also evidence for pheromonal smell signals in both men and women (Savic et al., 2009), but we will not dwell on those still controversial intricacies here. Human females, like many other female animals, are often attracted to dominant males—among humans, these are usually those men who are wealthy, powerful (not just muscular, even though that may help), or otherwise accomplished. Romance novels are populated with just such men, and the heroes are not always the most benign of characters. (One need only consider the compelling but emotionally brutal Heathcliff in Emily Brontë's *Wuthering Heights*.) Romance novels are, of course, escapist literature designed to entertain rather than enlighten. But these books consistently feature dominant heroes and are a perennial draw for many women, suggesting that they strike a primal chord in the female sexual-romantic imagination. Women likely are attracted to dominant men because of their visible signs of selective advantage—both in terms of genetic legacy to be passed on to individual offspring, and in terms of ability to provide for a family of several offspring.

Social dominance interactions are not as strong or clear-cut in females as males because female brains are not as intensely governed by the effect of circulating testosterone on brain circuits. But we will see that human female sexual urges are also substantially influenced by this "male hormone," and perhaps women would be as aggressive as men if they had as much testosterone. Indeed, there is one striking example that supports the testosterone rule in social dominance. The peculiar development of secondary sex characteristics and adult social behaviors in female hyenas demonstrates the link between testosterone and socially dominant aggression. Unlike most other mammals, female hyenas have unusually high levels of circulating testosterone, and their external genitalia

remarkably resemble that of males. One cannot easily tell the sexes apart because the female's enlarged clitoris is the same size as a male's penis, and the clitoris is quite capable of erection. Females appear to exhibit their genitalia as a way to sexually entice males and also as part of social dominance displays.

Indeed, females play powerful roles in hyena societies and are consistently dominant over males. This anomalous development in female hyenas highlights the causal pathway from testosterone to dominant aggression. High levels of this hormone are key to the social dominance of female hyenas. In human females too, perhaps surprisingly, it has been observed that a single dose of testosterone increases aggressiveness and other male-typical psychological characteristics (Bos et al., 2010; van Honk et al., 2004), with abundant brain imaging to back up such steroid effects on the brain (van Honk & Pruessner, 2010).

Female LUST Circuits of the Brain

The female circuits for LUST have been worked out in some detail in a variety of laboratory animals. These circuits differ markedly from those found in males. Female urges for sexual receptivity originate in the ventromedial hypothalamus (VMH), a different part of the hypothalamus than male urges, which are critically dependent on the POA—the preoptic area of the anterior hypothalamus. Damage to the VMH region of the brain severely impairs female sexual receptivity, while such a wound has comparatively little effect on the sexuality of male rats, as long as they have not become too aggressive.

As we mentioned above, most female mammals do not produce much testosterone (although some is manufactured in their adrenal glands). Female sexual readiness is controlled primarily by estrogen and progesterone in most mammals. But as we will see, adrenal testosterone contributes much to female receptivity. Female sexual arousal in most species is governed by regular estrus cycles, which in turn are controlled by the strict timing of the release and interplay of estrogen and progesterone. The estrus cycle begins when the hypothalamus secretes the hormones that lead to ovarian readiness via the pituitary gland, a small appendage suspended at the very base of the hypothalamus. The pituitary gland participates in the estrus cycle by secreting gonadotropin-releasing hormone

(GnRH, also known as luteinizing hormone-releasing hormone, or LH-RH). This in turn induces the ovaries to secrete estrogen as ovarian "eggs" are ripening, followed by a pulse of progesterone when the mature eggs spill into the fallopian tubes, ready for fertilization.

Estrogen and progesterone also prepare the female to be emotionally receptive to and trusting of the advances of suitors by promoting the manufacture of oxytocin, a key chemical that mediates female sexual readiness. Estrogen turns on dormant oxytocin genes in hypothalamic neurons, leading to elevated brain oxytocin production. Estrogen and progesterone also promote the dramatic expansion of oxytocin-receptor fields in the VMH, almost like flowers bursting into bloom on a spring meadow. The combination of increased oxytocin production and receptor proliferation results in arousal signals that initiate and support the *lordosis* reflex via circuits in the spinal cord. This reflex causes a sexually receptive body posture that consists of an upward arching of the lower back, with resulting exposure of the genitalia (Pfaff, 1999). Although the lordosis reflex is organized within the lower spinal cord where there is also an abundance of oxytocin receptors, it is powerfully promoted by descending neural pathways that originate in the VMH. So when females, of many mammalian species, are sexually aroused, they assume a posture that facilitates copulation. To the best of our knowledge, in human brains this readiness is more largely reflected in the qualities of mental life, but that may only be because body postures have not been adequately studied.

In most mammals, sexual receptivity is often evident simply by the enticing smell of the female's body. Male rats can rapidly "sniff out" a single receptive female from a hundred unreceptive ones. As already noted, however, the details of female sexual attractiveness vary considerably from one species of mammal to another. Some species, for instance, do not have estrus cycles. Such species are reflex ovulators, which means that the sexual act itself triggers eggs to be released from the ovaries. Human females do have psychological estrus cycles but on the surface women are largely *concealed* ovulators, lacking the florid signs of sexual receptivity exhibited by many other species. For women, receptivity is much more a state of mind, reflected, in part, by "monthly" fluctuations in erotic arousability. A certain potential for receptivity is always there, though, responsive to various social variables, albeit not as intensely as in males.

A woman's erotic state of mind is influenced by cyclical changes in brain chemistry. At the peak of fertility, when both estrogen and progesterone are high, a woman's thoughts turn more easily to erotic fantasies than when these hormone levels are low. This is, in part, due to direct effects on brain circuits, although there are also multiple indirect effects as on the fluctuating activities of brain oxytocin systems. As sociologists are ready to emphasize, there are limits to which the biological stories culled from other animals apply to humans, especially women, who exhibit much more choice, discernment, and subtlety in sexual matters (Udry, 2000). Still, many of the underlying neurochemical principles generalize well across mammalian species (Meston & Frohlich, 2000), but there are always some unique aspects in each species, including the high sensitivity of human female sexuality to testosterone.

In humans, adrenal testosterone adds a lustful ingredient to women's sexuality that is not as important for sexual enthusiasm in females of most other species. It almost looks as if there is a fragment of male sexual urgency that has been added to human females' sexual equation. As a result of this component of women's sexuality, supplemental testosterone is proving to be an effective way to restore sexual eagerness in women who have reached menopause (Al-Azzawi et al., 2010). Many older women need no such supplementation, perhaps because their erotic-caring attitudes developed across a lifetime and operate largely at learned secondary- and mentally sustained tertiary-process levels. Of course, for satisfying sexuality, for both males and females, the most important ingredient is the affective quality of mind. As we mentioned in Chapter 1, oxytocin may enhance CAREing feelings, but there are surely many other chemistries besides oxytocin that promote the internally experienced and externally expressed ardor of sexuality. Oxytocin, however, has been especially well studied in mammals (Uhl-Bronner et al., 2005), along with the ancestral vasotocin in birds (Panzica et al., 2001).

It is interesting to note that oxytocin, a key chemical of female sexuality, can also arouse males. The administration of oxytocin to male rodent brains, and perhaps the secretion of oxytocin within the male human brains, will produce erections. Furthermore, oxytocin is secreted in large amounts during ejaculation. Vasopressin, on the other hand, which strongly promotes sexual eagerness (perhaps even pushiness) in males, has the opposite effect on females. If vasopressin is injected into the brain of a female rat, her

sexual receptivity is drastically inhibited. Still, both male and female rats enjoy sex. Males rapidly develop preferences for places where they have engaged in sex. Although female rats also display such place preferences, they typically do so only if allowed to self-pace sexual activities (Pfaus et al., 2003). Even female rats do not like having sex imposed on them. Still, if a human investigator artificially stimulates a female rat's clitoris (with a cotton swab on the tip of a vibrator) she becomes more sexually solicitous toward male rats, is more likely to become pregnant, and also shows preferences for places where she received such stimulation (Cibrian-Llanderal et al., 2010). The rewarding properties of this clitoral stimulation appear to derive from the activation of the rat's POA (Parada et al., 2010).

Despite their many interesting differences, the sexes also share so much, even at the primary-process level. We mentioned above that the brains of each sex contain residual sexual circuits typical of the opposite sex. So vasopressin circuits are found in the brains of females in smaller abundance, and oxytocin circuits exist in male brains but in smaller abundance. How might these circuits function? We speculate that vasopressin systems in the female brain may help to energize some of the more aggressive aspects of maternal behavior (e.g., protecting the young from harm); conversely, oxytocin systems may sustain some of the gentler aspects of male behavior (e.g., the tendency of fathers to be nonaggressive and supportive toward their offspring). In any event, despite the many differences among species in the way they manage sexuality, it appears at the primary-process level as if all mammals share remarkably similar LUST circuits (Pfaus et al., 2003).

LUST AND THE SEEKING SYSTEM

It is important to recognize that, as with the pursuit of every other sort of reward, the SEEKING system is recruited in the task of finding sexual companions. This means that, in addition to the sexual chemicals mentioned above, sexual desire and eagerness are promoted by dopamine-fueled SEEKING. In human society, the dopamine-driven search for companionship is facilitated in myriad ways: matchmaking friends, singles bars, dating agencies, and the Internet, to name a few. Indeed, whether one is seeking intellectual or carnal knowledge via the Internet, it is the SEEKING system that drives the action. As we emphasized in Chapter 3,

this dopamine-fueled engagement in affect-filled (euphoric) actions plays a part in the search for all environmental delights, including sex. There is some evidence that this system may be somewhat more vigorous in males than in females, but this observation could be largely situation-specific—dictated more by specific rewards, survival duties, and the ecological constraints in which animals find themselves. For instance, when mother rats gather offspring that have dispersed from the nest, it is oxytocin stoking the SEEKING system that initiates much of the work. Males are not especially eager to undertake such tasks, so as far as caring is concerned, the SEEKING system appears to be more responsive in females. We know that the SEEKING system is sensitized—becomes hyper-responsive—to a variety of life challenges, including stress, hunger, and drugs of abuse. When it is sensitized, animals are more eager to pursue all kinds of rewards—food, sexual contacts, and drug-induced thrills (Nocjar & Panksepp, 2002). Thus, even though the SEEKING system seems slightly more active in males than females in many situations, the reverse is the case in others.

GENDER WITHIN THE BRAINMIND: PRIMARY-PROCESS GENDER MENTALITY

Although there are many neurochemical differences between men and women, oxytocin and vasopressin underlie particularly important affective distinctions. It is clear these hormones have broad effects as social sexual peptides because they interact with higher mental processes. This means that these peptides promote psychological traits that are differently weighted in each sex. Oxytocin encourages female-typical nurturing attitudes, captured in the phrase "to tend and befriend" (Taylor et al., 2000). Vasopressin shifts animals toward male-typical attitudes that might be captured in the phrase "pushy and competitive." As a rule of thumb, adult female rats have twice as much oxytocin activity to influence their brains, minds, and behaviors, while adult males have twice as much vasopressin (whether this translates to humans, is not known). There are a host of other BrainMind differences between the sexes. The greatest intrinsic differences exist at the affective levels, however, with comparatively few definitive differences at cognitive levels. Most cognitive differences—and many have been documented—probably arise as much from nurture as nature.

There are other emotional differences between the sexes. As noted earlier, males generally have stronger SEEKING and RAGE urges, and perhaps in some species stronger PLAYful tendencies. Female mammals typically have stronger CARE and separation-distress (PANIC/GRIEF) responsivity (explaining their higher tendency to invest in infant nurturance). While females also seem to be more likely to exhibit FEARfulness, the hormonal changes of motherhood distinctly increase confidence. This maternal confidence boost may derive in part from the psychological effects of elevated brain levels of oxytocin (Panksepp, 2009c).

THE REAL OXYTOCIN STORY

As noted at the end of the introductory chapter, oxytocin has grabbed the popular imagination as being the "love hormone," which is a glossy oversimplification of how the brain operates. It is true that oxytocin plays a prominent role in sexuality and other positive emotions. It strengthens the vigor of orgasms, and during childbirth it intensifies the capacity of a mother to complete the heroic task of pushing a baby into the world. It also helps quell the pain that accompanies emotional and physical distress. Above all, it promotes female confidence in the face of the difficult task of raising children. But oxytocin does not act alone as the "love chemical." It operates with the assistance of many other brain chemistries and environmental inputs.

Animal studies indicate that oxytocin acting alone does not produce a robust positive affective state. One study did produce a modestly successful conditioned place preference (CPP) (Liberzon et al., 1997). However, in Panksepp's lab, a CPP was only obtained when oxytocin was administered to animals in social environments where they could engage in friendly interactions (unpublished data from the 1990s). In other words, their oxytocin-induced place preference was tied to the availability of positive social experiences. Thus, it is possible that oxytocin alone does not directly produce intense positive affective experiences, but only from concurrent social interactions; thus, it may enhance the effects of other brain chemicals that more directly promote positive social feelings, such as endogenous opioids (opiate-like chemicals made by the brain) that are also released when people and animals engage in friendly social interactions (see Chapter

9). Endogenous opioids are major "comfort and joy" chemicals and one readily obtains CPP when opiates are administered, with no need to add social interactions to the mix (Tzschentke, 2007).

As we noted above, oxytocin can increase the brain's sensitivity to opioids (Kovács et al., 1998). When animals engage in friendly interactions, endogenous opioids are released in their brains (Keverne et al., 1989; Panksepp & Bishop, 1981), and perhaps oxytocin intensifies the pleasure aroused by those opioids. We will understand much more about this phenomenon as more work is conducted to determine how effectively various socially facilitated CPPs can be reduced by opiate receptor antagonists such as naltrexone (see Panksepp et al., 1997).

In this context, it should be emphasized that whenever we talk about these social neuropeptides we are talking about central (brain) effects. Although oxytocin and vasopressin are also peripheral (blood) hormones that respond vigorously to stress, there is little evidence that the release of these hormones from the posterior pituitary gets them back into the socio-sexual circuits of the brain. Indeed, from a social neuroscience perspective, peripheral hormone levels can yield paradoxical effects. For instance, if one monitors levels of tension in human couples who are distressed, blood levels of oxytocin, but not vasopressin, are positively related to levels of stress in females but not in males; to the contrary, in males the levels of vasopressin, but not oxytocin, are highly correlated with the social stress (Taylor et al., 2010). Should we assume that both oxytocin and vasopressin cause stress in the brain? No. Peripheral measures of these hormones provide no clear information about their central effects. Indeed, perhaps oxytocin is released into the circulation by stressful events in order to counteract negative effects on the body.

OXYTOCIN AND OTHER RELEVANT ANIMAL RESEARCH

Whether or not brain oxytocin directly induces positive affects, a variety of experiments demonstrate that oxytocin plays a crucial role in positive social interactions. For example, oxytocin inhibits cries of distress when young animals are separated from their mothers, indicating that oxytocin plays a pivotal role in providing emotional comfort when animals are alone (see Chapter 9). This effect does not require opioid involvement. Also, both

centrally and peripherally administered oxytocin diminish the tendency of male rats to kill their young. Following sexual intercourse, oxytocin is secreted. As we saw with the quail example, such molecules can evoke peaceful tendencies. In rats it seems that such effects reach a peak after about 3 weeks—this being the gestation period. It is known that at this time, when their own pups would be born, father rats lose their urge to kill baby rats. Thus, their sexual behavior seems to have registered in their brains in such a way that the father rats are highly unlikely to kill their own offspring.

Abundant research findings show that extra oxytocin placed in animal brains can promote maternal moods in female rats, even virgin ones (see the next chapter on CARE). The same can be achieved by injecting virgin female rats with blood from a nursing mother; whether this outcome is due to oxytocin is not known. In any event, oxytocin facilitates the strong social bond ("pure love"?) that emerges between mother and child. This effect has been most thoroughly studied by Keith Kendrick and colleagues in sheep mothers (Kendrick et al., 1992).

Oxytocin activity and the specific distribution of oxytocin receptors in the brain also facilitate adult pair-bonding in certain species of voles, especially the well-studied prairie vole, which has a tendency to form family groups with stable sexually-mediated pair-bonds between adult males and females (Carter et al., 1995). "Stability" here does not mean "exclusivity"; practically all pair-bonded animals show some "extra-pair copulations," which is a scientific way to say they fool around, if they can, while not developing lasting bonds (commitments) with the temporary mates. Research indicates that the prairie voles pair-bond, and tend to be especially gregarious, because of the particular distribution of oxytocin receptors in their brains. Another species of vole, the montane vole, on the other hand, lives a solitary life, like a hermit, and its oxytocin receptors are distributed in rather different, albeit nearby areas of the brain (Insel & Shapiro, 1992).

But we still do not quite know exactly what feeling oxytocin produces—is it to feel more loving, more confident, perhaps even courageous while being less aggressive? Or does it simply help one to be more relaxed and laid-back? Perhaps all are true. We will return to these difficult issues in the next chapter.

But birds might also provide clues. Studies with birds indicate that vasotocin (as well as oxytocin) can facilitate spontaneous wing-flapping, which may indicate confidence, especially because the effect is increased markedly in the presence of other birds (Panksepp, 1992). Indeed, in unpublished work about 20 years ago, Panksepp evaluated this idea directly. Groups of eight infant chicks were given brain ventricular infusions of oxytocin, or placebo, and their confidence was measured by observing how far they dispersed in a large well-lit novel room where they could freely explore after being kept restrained under a bucket for one minute. When the bucket was lifted, the oxytocin-treated chicks explored more widely around the room than placebo-treated birds, suggesting that the oxytocin-treated chicks felt more secure—which amounts to increased confidence.

What most animal studies do not consider adequately is how these diverse findings could be explained in terms of primary affective changes. Instead, too much animal research still focuses just on behavioral changes and bypasses affective considerations. One reason we should seek to identify the primary affective processes in such experiments is because oxytocinergic systems exist exclusively within the subcortical reaches of mammalian brains—the parts of the brain that generate emotional affects. If we get the primary-process interpretations correct, then we will be in a better position to understand the higher-order emotional concepts, commonly used in human oxytocin research, that we often use to understand human emotions.

Human attachments, both sexual and otherwise, are subtle and complex, with enormous layers of cognitive complexities. However, if we accept that the fundamentals of positive attachments are largely mediated by primary-process, subcortical oxytocin circuits, then we can obtain a better understanding of those higher, secondary- and tertiary-process psychological aspects of human attachment. For instance, one recent human study found that intranasal oxytocin promoted an attitude of *schadenfreude*—a German term that may be translated loosely as “gloating” (Shamay-Tsoory et al., 2009). How puzzling for a “love” molecule. However, perhaps this is merely a tertiary-process response, reflecting some kind of primary-process confidence generated by the oxytocin?

Once we assimilate the primary-process lessons of oxytocinergic effects on the MindBrain, we should have better ideas for the utilization of oxytocin in psychiatry. It will be of special interest to determine how well

oxytocin and (safe) opiates can counteract depression, especially the type of depression that sometimes occurs soon after childbirth. Parenthetically, it is already known that "safe" opioids, such as buprenorphine, can rapidly counteract depressive symptoms in many people who have not been helped by other medications (Bodkin et al., 1995).

Human Psychological Effects of Oxytocin and How They Relate to Mammalian Primary-Emotional Processes

Animal studies indicate that oxytocin reduces separation anxiety and that it promotes confidence and positive social interactions. Does it produce similar effects in human beings? There is every indication that it does. There has been an increasing amount of human work with oxytocin, mainly because it can be administered via the nose (intranasally) without any undesirable side effects. Many fascinating findings about the effects of intranasal oxytocin have been reported in the past few years. Oxytocin facilitates the retrieval of positive social memories and feelings of trust, while decreasing anxiety and stress during social interactions (Ishak et al., 2010). Recently Ditzen and colleagues (2009) gave intranasal oxytocin to cohabiting couples to see how it would modulate their affectively positive and negative social interactions while they were discussing various topics that were likely to arouse conflict. Following intranasal oxytocin (40 IU), the ratio of positive interactions went up (as measured by eye contact, interest, emotional self-disclosure, validation, caring, nonverbal positive behavior), compared to negative interactions (criticism, contempt, defense, domination, belligerence, stonewalling, nonverbal negative behavior, interruption). This contrast was slightly more pronounced in males, who probably tend to be more feisty and pushy in arguments than females. Further, the stress levels of both partners declined as measured by plasma cortisol, but it decreased more so among females than males. In short, oxytocin soothed tensions in situations where conflict might otherwise have prevailed.

Although we do not know exactly why oxytocin has this pacifying effect, we can speculate on the basis of our knowledge that oxytocin reduces separation distress in young animals and that it promotes confidence. Perhaps when feelings of insecurity about separation are alleviated, people will simply have more friendly social interactions; they will not be

defensive or irritable. It is also plausible that when humans become more "confident" they are able to trust. Indeed we might even consider that "confidence" is the optimal affective term to describe pro-trust behaviors. This allows people to engage more readily with others. Recent work with fathers indicates that they get more intensely involved in playing with their children following a few snorts of oxytocin (Naber et al., 2010). In our own work, we find that oxytocin can improve the ability of depressed people to read other people's emotions, just by observing their eyes, and their brains showed increased arousal as they were doing such tasks (Pincus et al., 2010).

Overall, we suspect that oxytocin produces primary-process psychological changes in the minds of animals that are similar to those also produced in human brains. Clearly, it would be wise for human and animal investigators to try to find common concepts that will allow us to discuss the primary-process psychological changes in all species. This has not happened yet. Right now it seems that oxytocin may deepen positive social engagements in both animals and humans, especially when the environment supports positive social interactions. This can improve the quality of social life and the flow of sexual feelings as well.

To this end, more subtle phenomenological studies of expert observers of their own minds are needed. And diverse individuals should be studied carefully in many situations with both standard scales and autobiographical descriptions, before we really have a satisfactory grasp on how oxytocin influences human moods. In short we need more neuropsychoanalytic research, where phenomenology is put first (Benedek & Rubenstein, 1942; Panksepp, 1999; Kaplan-Solms & Solms, 2000) as long advocated by visionaries such as Francisco Varela (1999).

CONCEPTUAL ISSUES: EMOTIONAL LUST AND THE BODILY AFFECTS

Is LUST truly an *emotional* affect? In earlier chapters we discussed the tripartite distinction between (i) bodily-homeostatic affects, such as hunger and thirst, (ii) sensory affects such as the pleasure of taste or a good massage, and (iii) intrinsic brain-based emotional feelings. We noted that homeostatic, sensory and emotional feelings arise from different brain networks and that emotional affects, unlike homeostatic and sensory affects,

are always attended by emotional behaviors—by action urges and explicit tendencies in other animals that do not regulate their emotions the way we do. It is easy to see that FEAR, for example, is an emotional affect because it produces undeniably robust emotional feelings and it also generates actions like freezing and running away (with low and high activations of the FEAR system). But LUST is not such a clear-cut case, because both homeostatic and sensory considerations play a larger role in sexual arousal. One might wonder if LUST is a homeostatic or sensory affect rather than an emotional affect, or if it might be a combination of all three. A homeostatic explanation seems plausible because bodily/homeostatic hormone release plays an important role in determining an animal's readiness for sex. Lust is driven directly and vigorously by what is happening with sex hormones secreted from testes, ovaries, and to a lesser extent by adrenal glands, all of which are under the control of the "master gland," the pituitary, at the base of the brain. Could one not argue that LUST generates homeostatic affects, more comparable to hunger and thirst, than the "real emotions" that emanate from brain networks such as FEAR and RAGE?

Similarly, sensory experiences, especially touch and smell, play essential roles in sexual preference and arousal, a consideration that might prompt us to think that lust is a sensory affect. In some species, sexual eagerness is triggered by olfactory pheromones, without which their LUST systems simply would not work properly. In all mammals, sexuality is promoted by provocative skin contact prior to direct sexual stimulation. Often, the prelude to satisfying sexuality consists of abundant playful courting activities, along with somatosensory stimulation that typically culminates in genital stimulation. In many species, especially humans, there are abundant verbal stimulations, hugs, kisses, gentle touching, and visual delights. Thus, the LUST system, just like all the other social emotional systems—CARE, PANIC/GRIEF, and PLAY—is strongly regulated by the feelings aroused by bodily feelings. LUST is probably one of the most sensorially and homeostatically well-connected emotional systems, more so than RAGE and FEAR.

Given such close ties to homeostatic and sensory considerations, why would we still wish to consider LUST to be a basic *emotion*? This just goes to show how nature does not respect our man-made categories. Still, categories allow us to see patterns and relationships in complex sets of data that are worthy of our attention. And LUST sits most comfortably in the

"emotion" category. We maintain that LUST is an emotional-affective process because action readiness is so evident in the whole-body courting and copulatory activities of sexually active animals—and because emotional action readiness is an essential feature of the overall affective state of LUST. Furthermore, one of the reasons why peripheral (homeostatic) hormone release causes sexual readiness is because it regulates various brain chemicals that in turn regulate the brain's LUSTy actions. Thus, sex hormones don't simply have circumscribed receptors, like thirst and hunger seem to, but their overall impact lies in the ways that they influence the extensive LUSTful circuits concentrated in subcortical regions of animal and human brains.

Similar global claims can be made for sensory aspects as well. Olfaction has long figured as a prime signal for sexual readiness and eagerness, but other senses contribute to the overall picture. For instance, female rats, and females of many other species, go into receptive lordosis postures when male rats rapidly palpate their rumps. This touch-triggered "lordosis reflex" is a sure sign of female sexual readiness—this sustained female body posture, with back curved, rump up, and tail deflected to the side, facilitates male mounting and entry. Although many details about ways that sensory experiences arouse LUST remain to be clarified (for instance, the input from the clitoris to higher parts of the brain), such questions are getting increasing attention (Pfaff, 1999; Pfaus et al., 2003). Also in many avian species, primates, and certainly humans, the visual aspects of sensory arousal are well recognized. Still, the way erotic images so easily gain access to sexual arousal systems, especially in males, is a mystery. Is this one prime example where cortical visual specializations gain direct access to primordial subcortical emotional systems? Is this merely the result of learning processes, or is it intrinsic to primate brains? We just don't know. But we do know that without subcortical arousals, sexual feelings become meager. There are abundant subcortical brain sites that generate both sexual arousals and brain stimulation reward (Caggiula, 1970; MacLean & Ploog, 1962), highlighting the widespread locations of sexual pleasure.

However, even as we have come to understand that sexual readiness is generated by subcortical networks, there are other dilemmas to ponder. These brain sites are located well within the trajectory of the extended SEEKING system, which generates a great diversity of appetitive behaviors (see Chapter 3). Lesions along the trajectory of the lateral hypothalamic

SEEKING system disrupt sexuality and, indeed, all appetitive behaviors. So why don't we *just* envision lustful behaviors like courting and copulation simply as part of the SEEKING system? Is there anything unique about sexual courting as opposed to other typical SEEKING behaviors, like exploring, foraging, and stalking?

Clearly, the intrinsic LUST networks devoted specifically for sexual readiness are intimately intertwined with SEEKING urges. But male copulatory behavior is distinct from simple SEEKING behaviors, with dedicated circuits to facilitate mounting, intromission, and ejaculation. We also know that there are various distinct as well as shared chemistries that facilitate female and male lustiness, among the best-studied being oxytocin and vasopressin, respectively. Because orgasms in humans are the most intensely pleasurable aspect of sexual activity, it is especially noteworthy that in both human males and females it is all of these ancient trans-hypothalamic emotional systems, from the ventral midbrain to the reptilian basal ganglia, that light up during brain imaging of human orgasms (Georgiadis et al., 2006; Holstege et al., 2003). Although gender differences are apparent in many higher brain regions during the appetitive touch and petting phases of human sexual activity (Georgiadis et al., 2010), orgasms yield very similar pictures in men and women—generally with decreased higher-brain activity and profound subcortical arousals in the same brain regions that mediate sexual behaviors—except perhaps for the fact that the male orgasm is a bit more "primitive" in having more arousal in the periaqueductal gray (PAG) of the midbrain (Georgiadis et al., 2009).

But there is another dilemma in envisioning the arousal of LUST, as opposed to climactic orgasms, as being a simple reward-inducing process. Sadomasochism—the infliction of pain to promote sexual arousal—has long been known to be a feature of human sexuality. It has now been discovered that even rats get sexually aroused by mild stressors, such as modest pain (Caggiula & Eibergen, 1969), and that this is largely due to stress arousing the lateral-hypothalamic reward-SEEKING system (Everitt, 1990). However, here again we are seeing just another way that animals cope with certain stressors that promote generalized SEEKING urges. Mild foot shocks can activate brain dopamine release. And mild stressors, such as pressure on a rat's tail, are sufficient to amplify a large variety of motivational urges (Antelman et al., 1975). Thus, such effects may be based on the same brain SEEKING mechanisms that Valenstein and colleagues

(1970) implied when they first described the apparent motivational plasticity within "the brain reward system"—all of which was easily explained by the fact that SEEKING arousal can participate in a variety of positive and negative emotional and motivational arousals.

It is difficult to resolve all ambiguities in trying to generate neat categories of affective feelings, especially those entailed in mammalian sexuality, because we are so often dealing with cultural concepts generated by humans (tertiary-process, thought-related aspects of mind) rather than brain functions created by evolution. The primal-emotional systems are ancestral treasures, the primary-process "tools" that allow us to be the vibrant creatures that we are, which is the main focus of this book. Still, raw LUST reflects a brain-body state that lies at the confluence of what we have chosen to call sensory affects, homeostatic affects, and distinct brain emotional affects. Also, as emphasized at the beginning of this chapter, it is at this low primary-process level that we have to find some of the biological rules that lead to gender-identity *problems*, or perhaps we should say *issues*—transsexuality, homosexuality, and bisexuality. Now that we have discussed selected aspects of subcortical LUST circuits, we return to those dicey *identity* issues where, perhaps to the surprise of some, the most solid knowledge is arising from animal brain research.

GENDER DIFFERENCES WITHIN THE BRAINMIND

Male and female brain systems are somewhat different in terms of their sexual urges, satisfactions, and many other psychological traits. As we noted, the various brain systems that generate these traits are rather distinct in males and females. LUST urges, as well as other differences between the sexes, do not simply reflect peripheral bodily needs but also, *decisively*, the organization of the BrainMind. One of the most interesting aspects of mammalian sexuality is that physical and psychological expressions of gender have distinct but overlapping controls, which means that the gender of the body and the gender of the mind develop somewhat independently. Furthermore the relatively independent development of the mind and body starts to take place in utero—in the developing mammalian embryo—long before organisms can have any thoughts about sexual matters.

Biologically, we define females as those born with an XX chromosome endowment and males as those who are chromosomally XY. Female-type brain circuitry is certainly apt to be stronger in the majority of biological females and male-type brain circuitry is stronger in biological males. However, in this section we will consider how it happens that these two aspects of sexuality do not always match in ways that we might expect—when the mind/body genders/sexes are mixed, yielding a female brain in a male body or a male brain in a female body.

When we combine these complex biological phenomena with the equally complex personal, social, and cultural phenomenon that is individual identity—a phenomenon that includes sexual orientation and gender identification—we can be sure things will become infinitely more complex, with cross-currents at many levels, from biology to culture and from primary to tertiary levels of MindBrain organization. As we mentioned earlier, we will not attempt to discuss issues of societal expectations and gender roles because these are truly tertiary-process creations that cannot be understood through animal models. Conversely, the primary-process biological complexities can be much better studied in other animals than in humans. We think this biological research has profound implications for understanding our erotic feelings.

Before we begin, it is important to clearly define the labels we will use. Here, *homosexuality* implies the desire to have erotic, sexual relationships, in a variety of forms, with members of one's own externally apparent biological sex. It does not necessarily mean that a person with a man-typical body feels like a woman or that a woman with a woman-typical body feels like a man inside their minds. Homosexuality may or may not (in some or all instances) have a biological basis at the primary-process MindBrain level. We simply have no easy way of knowing. We cannot peer into the sexual differentiation of the human brain as readily as we can in animal models, where underlying biological issues can be illuminated in great detail.

In contrast, the term *transgender* is an umbrella term, typically used where mental feelings about who one is in terms of maleness or femaleness clearly fail to match up with one's physical appearances and/or chromosomal biology. When this "gender identity" disparity is so strong that individuals contemplate changing their body form surgically to match their mental, emotional and psychological feelings about who they are, the

term *transsexual* is more commonly used. The slowly accumulating data, more often than not, suggest that transgendered individuals do, in fact, have brains that are erotically differently organized from most other people who share their bodily sex (Gooren, 2006). It is an ongoing cultural tragedy that such individuals so frequently have to struggle against societal biases in order to live satisfying lives.

Animal research has demonstrated that at birth the latent imprints of gender identity within the BrainMind are invisible, while the sex of the body is usually unambiguous. Yet the sex of the brain is decisive in determining one's sexual identity. A female brain in a male body will result in a person who feels like a woman in the body of a male, and a male brain in a female body produces a person who feels like a man in the body of a woman. This is a basic scientific definition of the state of being transgendered. Clearly, the biology of the brain has profound implications for one's sexual identity and destiny. We can be confident that the little boys who keep insisting they have girls inside who want to come out, and little girls who feel their minds are more like those of boys, are often reflecting a biological wisdom that parents should listen to with courage and sensitivity, rather than with denial.

HOW BIOLOGY BECOMES DESTINY WITHIN THE PRIMARY-PROCESS AFFECTIVE FUNCTIONS OF THE MIND

The close study of fetal maturation in several nonhuman mammals has clarified that the sexual development of the brain and body proceed along different pathways. These studies highlight general *principles* of gender development in all mammals, not simply differences in one particular species. This knowledge is now known to be highly relevant for homologous processes in our own species, at least in the few places where the difficult conceptual translation from our knowledge about transgender factors in animal brains has been attempted in the human species (Zhou et al., 1995). In short, the sex hormones that determine the sex-specific organization of the brain during prenatal development are different from those that help specify the appearance of the genital apparatus of males and females.

If those fetal brain chemistries unfold atypically, a developing organism's braingender identity can be shifted at the primary-process level. This can

occur for any of a large number of reasons, including extreme psychological stress that mothers may experience during pregnancy, as well as hormonal medical treatments, and even exposure to congeners from the environment (e.g., there are many chemicals in our polluted environments that act like sex hormones, helping to explain how some amphibians are becoming reproductively compromised). When these stressors prevail during crucial stages of pregnancy, an infant can quite literally be born with a male-typical brain in a female-type body or a female-typical brain in a male body, as well as abundant gradations between the "extremes." Such diversity can be used to support a very liberal sort of argument about bisexuality: If gradations in the sex of the brain and body are common, then the extremes, including strict heterosexuality, strict homosexuality, or extremes of gender identity would seem to be more likely the exception than the rule. We suspect the data support that the majority of individuals fall into categories where brain and bodily sexuality match up, but there is currently no clear empirical way to make such determinations.

In any event, the fact that the brains of fetuses have gender identities that may not match their bodily sexual organs indicates that gender identity is not simply learned during maturation. At the same time, obviously all self-identity issues are partly molded by learning and culture. But in their raw form, male- and female-type brains contain important constitutional differences. And they need not match visually evident maleness and femaleness of the body.

When a child is born with the body of one sex and the brain of another, social pressures will only make matters worse, because the child's innate gender identity cannot be altered by persuasion. It is regrettable that some countries, which consider themselves civilized, do not yet offer transgendered people equal rights and opportunities for happiness in the world. Hopefully, education will eventually change the hearts of those who would discriminate against nature (which is what Spinoza considered God to be). These neurobiological facts are concordant with social practices that many American Indian tribes traditionally followed: At times, nature ordains that a female sexual identity should flower within the brain of a biological male, and a masculine temperament should flourish within a biological female (Zhou et al., 1995). The wisdom of some of our ancestors readily accepted the psychosexual variety that Nature bestowed on vertebrates—a continuum of maleness and femaleness—that many in our

culture have learned to scorn. But such variations also abound in other animals, and the underlying principles are very similar (e.g., Bagemihl, 1999; Gavrilets & Rice, 2006).

MALE AND FEMALE MINDS: FETAL DEVELOPMENT OF GENDER WITHIN THE BRAIN

Developmental processes during the second trimester of gestation seem crucial in establishing the sexual identity of the human brain (Murray et al., 2000). This is not the same in all species. In rats, the third trimester (in fact maximally around the nineteenth day of pregnancy) is the pivotal time when the sex of the brain is established. These prenatal gender imprints have important consequences for vast patterns of brain circuitry and neurochemicals that control sexual urges, maternal urges, and other social tendencies such as aggression, not to mention a series of more controversial higher cognitive strengths and weaknesses, such as empathy and jealousy. This is why it is important to distinguish the concepts of biological "sex" and psychobiological "gender" when we discuss the imprints that gonadal hormones leave on the brain during early development.

In boys, most of these differences, but not all, arise from the single major thing the Y chromosome does for developing males. It promotes (via the secretion of testes determining factor) the growth of testicles with the capacity to secrete testosterone during gestation. In fetuses with Y chromosomes, testosterone is secreted before birth (with a stupendous spike in the second trimester of human gestation; in rats, this occurs in the third trimester). This testosterone sets in motion the masculinization of the brain and body (Berta et al., 1990). Things would not be so complex if both body and BrainMind organization were controlled by the same biochemistries, but, in fact, they each take a different path. The brain is masculinized when testosterone is converted to *estrogen* by an enzyme called *aromatase* (enzymes are chemicals that can instigate and facilitate chemical reactions, but they are not present in the final chemical product). The body, by contrast, is masculinized when testosterone is converted to *dihydrostestosterone* (DHT) by the enzyme *5-alpha-reductase* (Breedlove, 1992).

The early surge of estrogen (which, in the popular view, is associated with female sexual functions), then, is critical for masculinizing the fetal

brain. If human mothers pregnant with female offspring were injected with massive amounts of estrogen during this critical point in gestation (as has been done to rat mothers in many studies) the physically female offspring are likely to be born with male-typical attitudes. Similarly, if the boy fetus's own testosterone could not be converted to estrogen, he would be born with a feminized brain. Thus, the testosterone-to-estrogen cascade within an embryo's body determines the maleness or femaleness of its brain. Many of these facts have been well worked out in laboratory animals, including how certain environmental stressors can modify these processes. But there is only indirect, albeit rapidly increasing, evidence that these lessons also apply to humans.

The fetal male body develops along a different chemical route. In fetal development, all mammalian embryo bodies are initially female in appearance. The body of the embryo is masculinized by testosterone being converted to DHT. If it were not for the spike of fetal testosterone in the bodies of boys, all humans would appear physically female.

Thus, our genetic and gestational experiences contain the potential to form the sexual identity of our minds and bodies. Nevertheless, the individual sexual characters of our minds are not evident at birth. All these psychobiological tunes have to be played out by living in the world. Family and culture will eventually provide many teachers, but the earliest are the sex chromosomes that construct the mental and physical paths to maleness and femaleness.

Gender "Identity" Lessons From Rats

As noted above, when fetal male rats develop normally, there is a spurt of testosterone on the nineteenth day of gestation. Aromatase converts much of that testosterone to estrogen and this promotes masculine brain development. Concurrently, 5-alpha-reductase converts the testosterone to DHT, and this produces the male body. However, prenatal maternal stress has been linked to the development of a female brain in a genetically male fetus. If a mother rat experiences stress during that critical period, the testosterone release can come on too early, before the aromatase enzymes are ready to convert it to estrogen. As a result the male-LUST circuitry in the brain does not develop normally. However, much of this early testosterone release does get converted to DHT, which yields a male-typical

body. For humans, we simply do not know the parameters for such transformations, so one could imagine that normative human development progresses much more along a broader continuum much of the time.

In any event, the male rats born to highly stressed mothers have normal male bodies, but they display less masculine behaviors and more feminine behaviors when they mature sexually at puberty. This indicates that their brains were not fully masculinized. In a normal litter of rats (which typically ranges from just a few to a dozen pups), 80% of the male rats will be "*studs,"* meaning that they will display solely male sexual urges in adulthood, while 20% will be "*duds,"* meaning that they will be relatively asexual. When mother rats are stressed, resulting in disruptions in the manufacture of estrogen during gestation, only 20% of the males will be studs, with about 20% being duds, while the remaining 60% or so will display bisexual and homosexual behavioral tendencies (Ward, 1992). Again, we cannot be sure how this applies to humans: It is easy to imagine that humans are likely to have much more variability than is represented in the fairly straightforward outcomes in rat models, due to much longer gestation, larger brains, and development progressing against a background of chemical gradients that have much larger windows of variability. Such cautions notwithstanding, differences in details among species are bound to be vast. It is clear that the above general principles apply across mammalian species.

Embryonic chemistry probably does not account for all homosexual or even transgender tendencies, but it certainly accounts for some, and perhaps most, transsexual tendencies. But the evidence at a population level is by no means large. For instance, it has been documented that there was a greater level of homosexuality in German boys born during the hardest years of World War II, when expectant mothers endured a great deal of stress. But is that because of the above-mentioned variables? We don't know. But it is reasonable to assume that these mothers experienced excessive stress during the critical second trimester of pregnancy, resulting in potentially more than normal numbers of babies with male body types but female brain tendencies. But much of this is inference.

Let us now consider the opposite scenario, when estrogen is abundant but DHT is in short supply during the second trimester of fetal life. This situation, brought about by a genetic anomaly, has been vividly described in a small group of people living in the Dominican Republic. The males in this

group are genetically deficient in 5-alpha-reductase, the enzyme that helps to facilitate the development of external male body characteristics (scrotum and penis). Because they are not deficient in aromatase, the men's brains will develop along typical masculine lines. However, because of the lack of DHT, the bodies of these boys have a female appearance at birth, with no testicles (which remain undescended in the abdominal cavity) and a rudimentary penis that can be mistaken for an enlarged clitoris. Because these biological boys look like girls, and there is no way to see into the organization of their MindBrain, they are raised as girls.

Still, their Y-chromosomal endowment becomes active at puberty and produces testosterone, resulting in an increase of body hair, a deepening of the voice, an enlargement of the penis, and finally the descent of the testes. Because of this dramatic transformation at puberty, these lads are called *guevedoces*—literally "*penis at 12*" (Marks, 2004). Male-typical sexual urges also begin to emerge. So the boys' pubescent erotic desires come to be directed toward females, even though they were reared as girls throughout their childhoods. This probably indicates that the male brain is instinctively prepared to respond to certain features of human femaleness, such as facial and bodily characteristics, voice intonations, and so on.

It is remarkable, the ease with which *guevedoces* boys raised as girls can assume male typical roles. This is partly because the phenomenon is well known and societal supports are already in place. This has not been true in comparable situations in our own society. The problems have been highlighted by a famous case in which "the system" tried to force a male brain to assume a female identity: A biological boy was surgically converted to a female soon after birth, because of a botched circumcision operation (Diamond, 2004; Money, 1995). Little Johnny became Joan and was brought up with all the family and cultural expectations that "he" was a "she"; but things did not work out as well for Johnny/Joan as they do for the typical *guevedoces*. Johnny's physicians believed that gender identity was culturally determined, and they insisted that a concerted effort to rear him as a girl would succeed in Johnny believing that he was a girl. However, Johnny always continued to believe that he was a boy, and when he came of age, he insisted that his body be restored to correspond to his biologically predisposed male mentality. There are other cases like this without any botched surgery involved; people simply insist that the sexual characteristics of their bodies and the gender characteristics of their minds

do not match up. Among the many other possible reasons for such "mix-ups" is a condition called *androgen insensitivity syndrome*; in its most extreme form, such males have female genitalia along with undescended testicles.

A genetically female fetus is also vulnerable to sexual incongruities. If a genetically female fetus is exposed to too much estrogen during the sensitive periods of development, the brain will assume male-like characteristics while leaving the body feminine. These females will preferentially exhibit male-typical behaviors at maturity (Gorski, 1988). Indeed, in the 1940s and 1950s tomboyishness in girls was inadvertently promoted by injections of *diethylstilbestrol* (DES), an estrogen-producing hormone once used to prevent miscarriages, especially if mothers were exposed to the hormone during the second trimester of pregnancy (Ehrhardt et al., 1985). These sexual anomalies usually do not happen in response to the mother's own substantial estrogen levels, because the chromosomal XX endowment informs female fetuses to manufacture proteins (e.g., *alpha-fetoprotein*) that thwart cross-gender chemical influences early in development. However, when excessive estrogens are injected, they can swamp this safeguard system, which can no longer "mop up" all that "male-brain juice." Again, stress, in addition to environmental factors, can result in the creation of a male brain in a female body.

However, again we should point out that this very likely occurs along a broad gradient of variability in normative human development, with absolutely no way to determine if any single individual has deviated from the typical outcome.

THE POLITICS OF SEX AND CONCEPTUAL CONFUSIONS: WE CANNOT EVER SEE THE PRIMARY-PROCESS LEVEL CLEARLY IN ADULT HUMANS

In a sense, we have been skating on some thin ice throughout this chapter. People have strong feelings about sexuality. Homosexuals and transgendered people have a hard time of it in many cultures. They are often denied equal rights under the law. In many countries, they are not allowed to marry with others who seem externally to be of the same sex. They are discriminated against—all at the tertiary-process levels of human life, those levels that are generally beyond our focus of concern in this

book. However, these tertiary-process cultural phenomena are our great concern as human beings who respect human differences at all levels. And as scientists we must insist that those who do not comprehend or respect such human difference step forward from the dark cultural shadows of ignorance, fear, and hatred into the sunlight of scientific reality. Our cultural life is riddled with human stories where ignorance has promoted suffering. Thus, perhaps we who have been privileged to have a scientific education should seek to shine some light into the prevailing shadows that continue to surround this topic in many corners of modern culture.

Consider "Billy" Tipton, whose life story was depicted in *"Suits Me": The Double Life of Billy Tipton* (Middlebrook, 1998). Born as Dorothy Lucille Tipton, in 1914, she became an accomplished jazz musician in her teens. When she decided on a musical career, she dressed as a man, apparently in order to be better accepted within professional circles. When not performing, she retained her identity as a woman. She spent several years in a lesbian relationship and at some point in her life she started to dress as a man all the time. Several other relationships with women followed. She managed to pass as a man by binding her breasts. During sexual activity she preferred not to be touched and probably employed a prosthetic penis. Although she never married any of her lovers, one of Billy's partners confided that "he" was "the most fantastic love of my life." Billy and another partner adopted three boys, all of whom regarded "him" as their father and were astounded to discover "his" true sexual identity following "his" death.

We know nothing of the psychological state of Billy's mother during the second trimester of pregnancy. As a fetus, might Billy have been exposed to a surfeit of estrogen that programmed a male-type brain within a female body? Our knowledge of brain gender differentiation tempts us to accept the circumstantial evidence that Billy's brain was indeed masculinized even though s/he had a woman's body. However, perhaps Billy could not tolerate living in an era where a bright woman was not able to express all her fine artistic passions and skills with the same opportunities afforded any man with a comparable heart and mind. We simply do not know. These are questions that can never be answered in retrospect. And this is the problem that we are left with through much of this book. The kind of foundational knowledge we need to really understand the elements of mind in

mammalian evolution simply cannot ever be understood by studying the infinite complexities of human behavior.

The fascinating details of early development inform us of a profound fact of nature: There are four extreme possible sexual outcomes in fetal development (with, of course, more modest permutations in between). A fetus can be a typical female, with a female body and mind, or it can be a typical male with a male body and mind. Babies with female bodies and male emotional minds, or male bodies with female emotional minds are certainly rarer but are sufficiently well understood to consider them as normal modes of sexual development. During puberty, the early imprint of maleness and femaleness comes to life under the sway of massive secretions of sex hormones from the testes and ovaries. This *activational period* of sexual maturation carries forward the preconscious brain imprints of fetal development. Although the cultural impact of an intensely lived childhood also plays its role in sexual development, puberty activates the fetal legacy. And like an ancient "impish orchestra," it begins to play insistent biological tunes down in the deep LUSTful recesses of the brain. To understand this, and to accept it as destiny, is to have both a full measure of wisdom and tolerance. Not to consider such variations "normal" is to insist that cultural norms are more important than timeless biological variability.

CAVEAT—VARIETY IS THE SPICE OF LIFE

Although all mammals share quite similar primary-process brain mechanisms for male and female sexuality, they vary enormously in how they express these urges within the details of their ecological environments and social communities. Reproductive strategies differ even amongst closely related species (Carter et al., 1995). Gibbons, for example, mate for life with a single partner, but gorillas prefer a harem-type family structure, as do many other primates such as putty-nosed monkeys (*Ceropithecus nictitans*) where social groups usually consist of one male and up to nine females with their offspring. Orangutans tend to be social isolates, with the sexes coming together mostly for copulatory purposes, while chimpanzees are quite social and promiscuous, sharing partners rather indiscriminately. Thus, even our closest evolutionary cousins, the great apes, provide no clear insight into our intrinsic sexual nature. Perhaps because of our rich

imagination and ability to create a diversity of cultures, we are likely to see all variants of such reproductive strategies in humans.

We remain largely ignorant about the neuroscientific causes and correlates that underlie these variations of mammalian sexuality in human brains and we are equally uninformed about the brain activities that support most of the varieties of normative human sexual behavior. For example, there are many individual differences in human sexual activity and arousal, which may or may not reflect differences in sexual chemistry. Sustained copulation leading to a single intense orgasm in human males tends to lead to prolonged satisfaction, resulting in a period of sexual inactivity (a *refractory* period), probably due to the depletion of brain chemicals. Males of other mammalian species, certainly laboratory rodents, require multiple orgasms before they are fully satisfied, perhaps because their sexual pleasure chemicals are depleted more progressively in the brain. However, human females are also more capable of multiple successive orgasms than males, again perhaps because their affective chemistries are not depleted as rapidly. What might be the meaning of this? Perhaps we humans evolved from promiscuous species where female reproduction was facilitated by multiple sexual partners, as appears to be common in chimpanzees. In other words, perhaps the capacity for multiple orgasms in women might reflect an ancestral capacity to be sexually aroused repeatedly by several partners. Of course this is a "just-so" evolutionary story, as are many sexy ideas in evolutionary psychology, for most such hypotheses can't be tested by using rigorous research strategies.

We simply have no way of determining how much our own brains were masculinized (defeminized) or feminized during gestation. There is considerable data that the asymmetries in the length of our index and ring fingers (officially called the 2D:4D ratio, with D standing for digit) may serve to estimate such issues but that is far from definite. In any event, it is possible that the degree to which one's index finger (2D) is shorter than one's ring finger (4D) may reflect one's degree of masculinization while still a fetus. There is abundant data for sex and gender differences in this finger-length measure, first described in the nineteenth century. To explore these fascinating findings, just Google "ring and index finger length" or "2D:4D ratio" to ponder the many dimensions of this fascinating biomarker. Normative differences in this ratio are more prevalent in males than females, as is the degree of masculinization as a function of gender identity.

This measure has often been reported to be feminized in male homosexuals, and masculinized in female homosexuals, but we are far from having confidence that this can be taken as a selective measure of brain or body masculinization, or both, or neither. Validation of the meaning of the measure is hard to come by. The measure also varies as a function of geography-nationality, personality, and many other variables beside sexuality (Manning, 2002). Further, despite its apparent straightforwardness, there are abundant measurement problems, with considerable variability among experts (Voracek et al., 2007). Still, if it could be shown to be a valid biomarker of what happened hormonally in utero, that would be remarkable.

In general, we should remain wary of premature closure of issues based on theoretical perspectives that are hard to test in neurobiological terms. This has been especially poignant for classic theories of psychosexual development such as those originally formulated by Sigmund Freud. As a didactic exercise, we conclude this chapter by deconstructing Freud's once influential perspectives on our sexual nature, views that now need to be seen as creative theoretical speculation as opposed to scientifically based expert opinion.

AFTERTHOUGHT: PSYCHOANALYTIC REFLECTIONS ABOUT SEXUAL DEVELOPMENT

In an earlier chapter, we noted that neuroscience provides an emotional taxonomy that is far more complete and scientific than the shorter lists of fundamental affects offered by psychoanalytic and many other psychiatric theories. This fact is especially stark when one considers that Freud proposed that there were only two drives: libido and destructiveness. With such a limited scope, Freud sought to understand the seemingly nonsexual aspects of infancy and childhood in terms of what he called "component instincts"—libidinal precursors to adult genital sexuality. Freud (1905b/1968) proposed four component instincts: oral, anal, phallic, and genital. (He spoke of the phallic as opposed to the genital phase because he famously—or infamously—believed that the penis was the focus of attention for both sexes at this phase of childhood—about 3–5 years old). But is "penis envy" really any more influential in female psychological development than "breast admiration" in young lads? We doubt it.

The theory of the component instincts was based on three considerations. The first was the observation of infants and children. Freud observed that oral, anal, and phallic activities (the latter including masturbation) provide pleasure in childhood. Since he believed in the existence of only two drives, these pleasures were deemed to be libidinal in nature. Thus when infants took obvious pleasure in suckling or in nonnutritive mouthing, Freud believed that these were infantile libidinal pleasures. He noted that toddlers take pleasure in defecation; hence, his view of the anal drive as a libidinal pleasure. Phallic sexuality arrived at about 3–4 years, when the little boy discovered his penis and the little girl discovered her clitoris and this was clearly sexual behavior. The Oedipus complex overlapped with the genital phase, which occurred when children developed a sexual/romantic attachment to the parent of the opposite sex. His psychoanalytic investigations with children and adults convinced him that the Oedipal complex was a normal phase in sexual development.

The second reason why Freud believed in the existence of the component instincts is because he thought that they became incorporated into adult sexuality. Kissing, for example, is an oral activity in which adults participate during sex. There is also genital fondling similar to phallic masturbation in childhood. The third reason why Freud proposed the component instincts is because he thought that they dominated in cases of perversion. Freud lived in Victorian-era Viennese society, when perversions were seen to encompass many sexual practices that we consider to be normal today. For example, oral sex was considered perverse as were all forms of homosexuality. Freud thought that when libidinal development was arrested, immature forms of the component instincts dominated the sexual life of the adult in ways that constituted perversion. Thus, he concluded that perverts were sexually "fixated" at early phases of libidinal development (Freud, 1905b/1968).

Freud's view of component instincts is only plausible if one accepts that there are only two drives. However, modern neuroscience has demonstrated that pleasure can also be obtained from nonsexual emotional systems like the positive arm of the PANIC/GRIEF system (social bonding) or from PLAY or CARE or SEEKING. Therefore, it is not plausible to say that all infantile pleasures are fundamentally libidinal. For instance, we are on very shaky ground when we try to make a case for the libidinal nature of orality. Suckling and nonnutritive mouthing may not be expressions of libidinal

pleasure—at least not entirely. Rather they may express the nonsexual pleasure that an infant feels when it is close to its mother (the positive arm of the PANIC/GRIEF system). Alternatively, these activities may reflect the pleasurable homeostatic affects associated with feeding. One can also imagine that before feeding, an infant's SEEKING system is pleasurably aroused and that mouthing is part of this pleasurable anticipation. Then again, mouthing might sometimes be a form of PLAY—one in which the infant can participate despite its relative motor limitations.

We also have reasons to challenge Freud's timetable for the emergence of phallic sexuality at the age of 3 or 4 years. Subsequent research has shown that vigorous rocking might induce orgasm even in infants (Kinsey et al., 1948; Martinson, 1994; Yates, 1978), while others have observed that infants engage in persistent genital touching as early as 6 months for boys and 10 months for girls (Galenson & Roiphe, 1974). Of course, infants cannot tell researchers if they have experienced orgasm or not and one cannot be sure that less focused genital touching is sexually stimulating. After all, infants play with their ears in a similar way (Levine, 1951). There is also the consideration that some degree of genital play during the first 18 months of life is a positive sign in that it correlates with good mothering and a general sense of contentment. Neglected infants do not touch themselves (Spitz & Wolf, 1946). Therefore, if contented, infants touch themselves in order to experience transiently pleasant sexual sensations; this may add to the sense of contentment, without deeming it to be a focused sexual activity that is more typical of mature LUST.

Modern neuroscience tells us a great deal about the principles governing adult sexuality and it also tells us a lot about the embryonic development of the sexual brain, but it tells us little about sexuality prior to puberty—about childhood sexual development. So we cannot draw firm conclusions from observations about apparently libidinal behavior in infancy and childhood. We do not even know if Freud was right in proposing that infantile development centers around oral, anal, and phallic vicissitudes. However, even if orality, anality, and genitality are central issues in childhood, there is no reason to believe that they are all purely libidinal in nature. Knowing that the mammalian emotional taxonomy consists of at least seven different primes and given that four of these (the positive arm of PANIC/GRIEF, PLAY, CARE, SEEKING) generate positive affects that are nonsexual, it is

unlikely that all infantile (or adult) pleasures are fundamentally libidinal in the way that Freud suggested.

Thus, modern neuroscience gives us food for thought when it comes to reconsidering classical theories of psychosexual development. However, it does not inform us about the true nature of psychosexual development in childhood or about any aspect of the culturally driven tertiary-process level of BrainMind emergence. This leaves psychotherapists in the unsatisfactory position of having many unanswered questions—especially about sexual development. For example, does homosexuality entail an activation of brain sites and chemicals typical of the opposite sex? Do homosexual girls have denser cell populations in the anterior hypothalamus, and are their brains more replete with testosterone and vasopressin? Are homosexual boys in a commensurate position, with greater sensitivity in the ventromedial hypothalamus and greater oxytocin activity? We have no answers to these questions in humans. We do in several other species of animals, especially laboratory rats and mice. But, of course, humans are not rats or walruses or monkeys. So how strongly should we believe in the general principles that are emerging from the incorrectly so-called 'lower species'?

With regard to sexual pathology in childhood, we are equally ignorant. Does sexual overstimulation in childhood result in the premature activation of sexual brain sites and chemicals? Why are parentally neglected children disinclined to touch their genitals? Are their sexual brain chemistries at a low level and, if so, what has made them dwindle? These are just a few of many questions about human sexual development and all the other emotions as they emerge into tertiary-process thought and cultural awareness, which remain to be fully answered. There remains a gulf between neuroscience and psychoanalytic theories of psychosexual development, not to mention human cultural life. Modern neuroscience gives us good reasons to question classical psychoanalytic theories about component instincts, but it has not yet provided evidence, and probably simply can't, to fill the vast chasm between our knowledge of ancestral tools for living and the individual lives that spin out the endless varieties of human existence. This highlights that neuroscientific and more complex psychological and sociological analyses need to work together to obtain a fuller understanding of human complexities.

SUMMARY

In this chapter we have discussed some of the insights that modern neuroscience has shed on our understanding of sexuality in mammals. We have seen that sexual circuitry and sexual chemistry in male and female brains are different. In males, the anterior hypothalamus is the focus of sexuality and testosterone mediates the production of vasopressin, which accounts for much of male sexual behavior. In females, the ventromedial hypothalamus is part of the primary-process sexual locus of control and the main sexual chemicals are estrogen and progesterone. These hormones in turn mediate the activity of oxytocin, a neuromodulator that significantly governs female sexual responses. Although oxytocin has been popularized as the "love hormone," we conclude that this is a simplification of its role, because it may not *directly* produce much positive affect. Instead, oxytocin may enhance the activity of endogenous opioids, which produce much of the positive affects triggered by oxytocin administration. Nevertheless, it is clear that oxytocin does *promote* the generation of positive social affects, especially confidence and trust, that are important for competent motherhood. We expect opioids do the same, and at high doses even to the point where people do not need other people, leading to the social isolation of addicts.

Sexuality in mammals, at the primary-process level, is a product of the LUST circuitries. But is LUST a true *emotional* affect, or is it better classified as a homeostatic or sensory affect? We conclude that it deserves to be considered as an emotional affect because it directly produces complex instinctual sexual behaviors, along with the associated raw affects, from sheer eroticism to orgasms. And such psychobehavioral action tendencies are the hallmark of primary-process emotions. In females of many species, sexual arousal promotes the sexually receptive position of the lordosis reflex and in males it produces commensurate sexual behaviors of solicitation, mounting, intromission (successful thrusting), and ejaculation.

Sexuality is further complicated (especially at the tertiary-process level for humans) because the sexual body and the sexual brain develop along different trajectories in utero. The male brain is created when testosterone is converted to estrogen and the male body is created when testosterone is converted to DHT. All fetal bodies are initially female and if there is no interference, the female body will continue to develop. The female brain and mind, however, may be masculinized if the fetus is exposed to too much estrogen at crucial times in the second trimester.

The topic of sexuality, as all other emotions, is riddled with unanswered questions. What determines the diversity of reproductive strategies in closely related primate species? What determines different strategies within a given species? How do environmental influences determine sexual expression in human beings? What might be the chemical and neural correlates to these differences? And a crucial question for every psychotherapist is how does the sexual drive—how does LUST—develop during childhood? One can ask many similar questions, all of which remain for future researchers to clarify. And that is a most wonderful aspect of science. The work is never done. This is certainly the case in neuroscience. More precise knowledge can be harvested, endlessly.

CHAPTER 8
Nurturing Love

The CARE System

Tiny eyes that rode inside me,
Little ears that in my voice rejoiced,
Perfect cheeks and chin and fingers
And lips with early words
So dear and moist, . . .

Child of days escaped forever,
You laughed and cried for all your worth,
Ran and scrambled far afield
But always circled to my side,
For I was your first home on earth. . . .

—Anesa Miller, "Baby Love" (1995)

MAMMALS WOULD NOT EXIST ON the face of the earth unless their brains and bodies were prepared to invest enormous time and energy in the care of their offspring, who simply could not survive without such devotion. The investment of maternal attention has not been left to chance: It is grounded in a solid set of instinctual brain urges to nurture newborn infants and to bond with them. The miracle of human motherhood, including the role of extended families in nurturance, has long been extolled by anthropologists (Hrdy, 2009; Konner, 2010). In modern humans, of course, nearby relatives and extended families are not always part of one's immediate community, and such primal motives all too often must be pursued in a context of

insufficient practical and emotional support—causing these most essential and powerful of instinctual action tendencies to become mixed with diverse worries and feelings of insecurity that can undermine the maternal mission. Under such conditions, the joy of mothering can be overshadowed by a predominance of negative emotions.

When one is surrounded by supportive others, however, the affective symphony of motherhood must be deemed one of the great gifts of Nature. We all may feel the impression of this gift imprinted within ourselves, even those of us who will never give birth. Fathers of many species have latent maternal circuits in their brains, waiting for the right environments to amplify their potentials (de Jong et al., 2009). Thus, one easily can argue that the roots of human empathy reach deep into the ancient circuits that engender caring feelings in all mammals, where we identify our own well-being with the well-being of others (Decety & Ickes, 2009; Hein & Singer, 2008; Iacoboni, 2009a, 2009b). Feelings of PANIC/GRIEF in others (see the next chapter) may be one of the most powerful emotional resonances to promote empathic devotions.

In some species, CARE urges are so powerful that they readily extend to the young of other species. At present, one can find many remarkable examples of cross-species maternal devotion in mammals that have been photographically documented on the Internet. In some species, such as laboratory rats, where mothers do not bond specifically with their own young, one can cross-foster dependent young between different litters with impunity. In other species, however, especially ungulates who are 'born on the hoof' so to speak, mothers form an exclusive social bond with their offspring within hours of birth, and after that time frame they typically will accept no others. As we will see, though, an understanding of the neural and emotional mechanisms of bonding can allow us to "re-open" the bonding window and to foster the establishment of maternal bonds in such species using physical, pharmacological, and social interventions.

In this and in the following chapters focusing on PANIC/GRIEF and PLAY, we will examine three brain systems that generate nonsexual social bonds. This chapter will focus on the CARE system, which is epitomized by maternal devotion. We will discuss emerging knowledge about the ways mammalian brains generate nurturing impulses; then we will briefly discuss how these chemistries may control social learning and higher social cognitions. In the next chapter we will examine the other side of the coin,

namely from the infants' side: how young animals become emotionally bonded to parents. Then we will move on to older animals, and the ways in which juveniles and adults form positive nonsexual bonds, or friendships if you like. We originally called the primal emotion that promotes infant bonding to mothers the PANIC system. This unusual label was used to highlight the fact that when most young mammals and some birds are separated from caregivers, the feelings engendered may resemble a "panic attack"—a psychiatric episode that is quite distinct from garden varieties of fear and anxiety (see Chapter 5). We still think this is a good label for the primal affect generated by the "separation-distress" system. However, because this label has caused consternation and confusion, we here employ PANIC/GRIEF—or simply GRIEF. As a result of this relabeling, some arguments may be easier to understand. For instance, in older animals and humans, with well-established affection bonds, social loss can activate a fuller spectrum of distressing affects that are more easily described in terms of sadness and grief. After our discussion of the GRIEF system, we devote a chapter to the PLAY system, which urges people and animals, especially young ones, to engage in joyous, competitive interactions typified by rough-and-tumble ludic activities. This wonderful emotional energy allows young children to become friends rapidly, and it allows older people to do so as well but more gradually.

Much research remains to be done on the CARE system, as well as on GRIEF and PLAY. These primary-process emotional systems, so important for understanding social attachments and the bonding failures that can promote depression, are not yet widely acknowledged either in neuroscience or biological psychiatry. Nevertheless we now know enough about these nonsexual social systems to identify them in the brain, to understand some of the important ways they function, and to envision what roles they may play in human mental health and emotional disorders.

We also understand that these systems have intimate interrelationships. For example, CARE inhibits GRIEF while GRIEF reduces PLAY. Along with LUST, these basic social engagement systems are foundational for mammalian and avian social attachments. In humans, at the very least, these systems also underlie varieties of love (Panksepp, 1998a). We say "at the very least" because we would not wish to deny such higher emotions to other species. But the ethereal higher spaces of animal minds are not as accessible to scientific study, by using present methods of inquiry. All these

systems share many affect regulatory neurochemistries, such as endogenous opioids and oxytocin. Despite abundant recent work (Numan & Insel, 2003), most of the details of these neural mechanisms, and the evolutionary paths that led to the emergence of the CARE system, remain to be worked out in detail. Because of the shared neurochemistries and proximate anatomies, however, we should not ignore the controversial possibility that maternal CARE emerged over the long course of mammalian brain evolution, in part from the preexisting brain mechanisms and affects of female LUST.

MATERNAL URGES

Who can resist the enchanting ballet of emotions between a mother and her infant? Each is exquisitely attuned to subtle communications from the other (Hrdy, 2009; Konner, 2010; Reddy, 2008). The hint of a frown or an uncomfortable twist of the baby's body will evoke a mother's comforting ministrations, and her smile produces a burst of responsive joy from the baby. This finely tuned emotional interplay, one of the primal sources of human love, provides profound satisfaction to most mothers, and it is essential to the emotional and physical health of the developing infant.

Yet maternal nurturing is by no means universal in the animal kingdom. Many animals, including almost all reptiles, have little in the way of maternal impulses. They allow their young to fend for themselves in treacherous environments where many will die from predation. In contrast, essentially all mammals (and birds) look after their young, often at the expense of their own comfort and sometimes at their peril. Among mammals the primary-process nurturant urge is strongest in females. In birds, however, fathers are often as attentive as mothers; this phenomenon is also seen in a minority of mammals (de Jong et al., 2009). In fish, the job of tending a nest of eggs is often left exclusively to fathers. Such urges to nurture emanate from inherent brain circuits that we collectively call the CARE system.

Historically, the existence of the primary-process CARE system in mammals came to the attention of researchers with the discovery that blood transfusions from postpartum female rats would instigate maternal behaviors in virgin females; such behaviors included nest building, hovering over pups, and gathering pups that strayed from the nest

(Rosenblatt, 1990). We still do not know which maternal chemicals in the transfused blood interact with brain systems of the virgin females to promote nurturance, but we do know that oxytocin within the brain is a chemical that can promote such a transformation. Maternal behaviors can also be promoted by electrically stimulating specific regions of the brain; this stimulation is affectively positive and hence rewarding. It remains likely that such urges to provide CARE arise from SEEKING arousal; the SEEKING system is essential for much of what mothers must do in order for their offspring to thrive, including building nests and retrieving young. Thus, a great deal of the positive affect of CARE is probably due to the arousal of brain dopamine (see Chapter 3), in conjunction with opioids, as well as oxytocin, prolactin, and many brain chemistries yet to be identified.

NEUROSCIENCE AND THE ACCEPTANCE OF SOCIAL BRAIN SYSTEMS

Research on the primary-process aspects of the social brain is currently moving forward rapidly. The neuroscientific community is recognizing the importance of CARE and the other primary social systems in the brain. Most neuroscientists readily accept that the LUST and RAGE brain systems exist, because both emotions are clearly manifested in animal behavior and because these emotions are patently essential for survival. A solid start in this area of research was made soon after the discovery of the social functions of brain opioid system (MacLean, 1990; Panksepp & Bishop, 1981), followed more recently by brain oxytocin (Carter, 1998; Insel & Young, 2001; Panksepp, 1998a). Although neuroscientific work on the primary-process social systems has lagged behind research on the other primary affective systems, such as RAGE and FEAR, it is rapidly catching up. Still, sad to say, many investigators who work with humans and other primates do not even yet recognize the existence, much less the vital impact, of primary-process social systems, and envision maternal and play urges to be socially constructed.

Of course, science is quite conservative, as it must be. Consistent evidence accumulated across many studies and across many species must converge in order for general theoretical principles to be assimilated into standard neuroscientific thought and practice. New ideas have to prove themselves before they become accepted. For instance, as we have seen, the

field is only gradually moving toward a conceptualization of the dopamine-aroused SEEKING system that generates appetitive motivation, while still hanging on to long-favored concepts such as "the Brain Reward System," despite the latter's insoluble paradoxes. Fortunately, recently the CARE system has been the subject of an impressive amount of neuroscience research conducted by pioneers such as Allison Fleming of the University of Toronto and Michael Numan of Boston College, and their students. (Of course, the lion's share of the actual hands-on research work is typically done by postdoctoral fellows, graduate students, technicians, and the ever-present talented undergraduates.) In addition, social scientists have become very interested in the workings of the higher, more recently evolved social brain—the many facets of cooperation, empathy, and social mirroring—largely because of the advent of modern brain imaging. Thus, there are now abundant treatises on the higher social aspects of primate and human social brains (e.g., Cacioppo & Patrick, 2008; de Waal, 2009; Tomasello, 2009), although these investigators rarely mention the more ancient primary-process social urges that all mammals share. The social scientists will in time remedy this omission, as they must in order to forge a more comprehensive understanding of our ancestral roots. It is possible for instance, as alluded to previously, that empathy would not exist without the foundations of maternal CARE and the psychic pain of GRIEF (Panksepp, 1998a; Watt, 2007).

The next chapter will consider the separation-distress (GRIEF) system of young animals, which signals their need for maternal CARE by producing distinctive emotionally charged cries. We know from animal research that mothers dutifully investigate locations from which the distressed cries of their infants emanate, even when the cries are generated by a tape recorder. The adaptive logic underlying this reaction should be obvious. Distressed crying promotes an infant's ability to survive by arousing the protective attention of parents. A new layer has recently been added to our understanding of this reaction, however. Research has demonstrated that when parents listen to the crying of their infants, the separation-distress (GRIEF) regions of parental brains light up (Swain et al., 2007), with mothers being more responsive than fathers. Mothers also can often distinguish the cries of their own infants from those of strangers. An implication of recent brain-imaging findings is that parents, especially mothers, may directly experience the distress of their infants as the

corresponding emotional systems of their own brains are aroused by the infants' cries. This evidence is significant for two reasons. First, it appears to be an example of primal empathy between bonded individuals. Second, it strongly suggests that GRIEF (separation distress) arousal activates CARE (maternal-type nurturance). Thus, we begin to see the outlines of neural pathways in which the primal roots of human empathy find their origins in the CARE and GRIEF networks of the brain.

EVOLUTION OF THE CARE SYSTEM

How might the powerful mammalian urge to nurture have evolved from ancestral beginnings in the brains of reptiles, animals that are notoriously noncaring parents? Why are mammalian mothers eager to protect, cherish, and even sacrifice in order to ensure the well-being of their offspring? How might the CARE system have developed? We cannot be sure, but we make a case for the hypothesis that the evolution of CARE was intimately intertwined with the LUST circuits within the brains of mammalian forbears. Even though CARE can be deemed to be a nonsexual emotional system, the neurochemical controls of sexuality also lie at the core of nurturant behaviors, as already noted. In other words, vasotocin and related ancient neuropetides like mesotocin, which fuel reptilian and picine LUST and birthing systems, may have evolved into oxytocin, which facilitates not only female LUST but is also a key brain system that promotes maternal CARE (Uvnäs-Moberg, 1998).

Vasotocin is an ancient hormone that generates sexual urges and well-regulated birthing reflexes in both reptiles and birds. Reptiles do not generally nurture their young. Birds do, however, and this nurturing attitude is induced by vasotocin. For an illustration of how vasotocin mediates birthing behaviors, we might consider its trajectory in the well-known nesting pattern of marine reptiles such as sea turtles. At the conclusion of a migration that covered thousands of miles in order to arrive at the beach where her ancestors gave birth, the mother turtle lands on the beach and digs her nest; as she does so, her posterior pituitary gland secretes vasotocin in ever increasing amounts. The vasotocin levels rise still higher as she deposits one egg after another. Then, as her labor is finished and she covers her eggs, vasotocin plummets to insignificant levels. Thus, her maternal urge and duties end. She returns to the sea. Weeks later, when her progeny

hatch, they scamper quickly toward the surf, vulnerable to attack and without parental protection. Only a fraction of the offspring escape predation and advance the survival of their species.

In the LUST chapter, we mentioned that vasotocin has a calming effect similar to the role of oxytocin in mammals, and promotes nurturant moods in many species of birds (Adkins-Regan, 2009; Balthazart, et al., 1996; De Vries & Panzica, 2006). Indeed, vasotocin probably evolved into the mammalian chemicals oxytocin and vasopressin, which play major roles in controlling female and male sexuality, respectively, in mammals. Here we are especially interested in the oxytocin evolutionary link because, in addition to being an important chemical in generating female LUST (Caldwell, 2002), oxytocin is also an important maternal chemical. It plays a central role in labor by producing uterine contractions and, following the birth, by triggering the letdown of milk when nipples are suckled. In addition, oxytocin, along with other maternal chemicals, helps to promote maternal moods and behaviors when infused into the brains of virgin females (Pederson et al., 1982; Keverne & Kendrick, 1994).

An evolved chemical continuum from ancestral peptides to oxytocin suggests that the CARE system in the mammalian brain probably evolved, in part, from the reptilian LUST system. It is not uncommon for one function to evolve from another that seems outwardly quite different. François Jacob, the Nobel Prize winning molecular biologist wrote, "Natural selection . . . works like a tinkerer . . . who . . . uses whatever he finds around him . . . to produce some kind of workable object. . . . Evolution makes a wing from a leg or a part of an ear from a piece of jaw. . . . Natural selection . . . does not produce novelties from scratch. It works on what already exists" (Jacob, 1977).

Jacob was speaking about the principle of *exaptation*, which refers to evolutionary changes in which seemingly radical alterations in existing structures yield structures that are useful for new adaptive purposes. One example of exaptation is found in the arches that supported gills in fish, which evolved into the middle ear bones that serve hearing in mammals. Exaptation refers to the surprising, as opposed to the obvious, routes by which evolution fits physical structures to new uses, including the transformation of ancient brain processes into systems that perform new functions. Thus, while gill arches were an aid to respiration in fish, the resulting bones in the mammalian middle ear facilitate hearing. And despite

any squeamishness or other aesthetic objections we may feel, it violates no law of nature that the seemingly nonsexual CARE system may have evolved from the sexual LUST system of ancestral species.

Like all evolutionary steps, changes brought about through exaptation are either retained or abandoned depending on their adaptive value—the retention of the change will depend on how well it allows an animal to survive in its environment. Presumably, nurturing care for the young was adaptive because parental care and protection provided a decisive competitive edge for the survival of all mammalian species. If a young animal can only survive by initially obtaining food from another animal, there is nothing like social bonding and maternal devotion to ensure that such sharing takes place. As we shall see, the emotional health of all mammals is critically tied to the quality of such early care and devotion.

THE CARE SYSTEM AND PSYCHOANALYTIC THEORY

The discovery of the CARE system suggests that Freud was not completely incorrect when he famously hypothesized that sexuality was the fundamental impulse or drive from which all positive human social relationships emanated. Perhaps his idea hid an implicit relationship between LUST and CARE that he did not recognize. Yet other basic social-emotional systems were nowhere in his view of human nature. For instance, the PANIC/GRIEF and PLAY systems, which are very important for social bonds, have no obvious relationship to the LUST system. Freud hypothesized that all nonsexual love, even maternal love, was a sublimation of an underlying sexual urge. He maintained that sublimation—the channeling of a basic emotional energy into socially useful purposes—occurred when sexual urges were transformed into social values capable of serving nonsexual purposes (Moore & Fine, 1990). For example, most societies have an incest taboo. Freud argued that the moral prohibition against incest may arise from the mother's basic wish to obtain sexual gratification, shaping it into the nonsexual gratification of caring for her child. Thus, classical Freudian theorists believed, and no doubt some still do, that the maternal impulse (as well as other platonic attachments) is a sublimated variation of an underlying sexual urge. We can now look at this old supposition in new evolutionary ways.

Although CARE may have evolved from LUST, the two systems are now sufficiently distinct in the brain and they perform different functions. The LUST system generates the sexual urge while the CARE system generates nonsexual tenderness, even though there may be class similarities in some of the associated feelings, due to shared regulatory brain chemistries. In any event, we now know that many social attachments derive from other sources than the sublimation of sexual urges, including the GRIEF and PLAY systems summarized in the next two chapters. Of course, sexuality also leads to social attachments. One might wonder whether the institution of marriage is an expression of the urge for sex or perhaps a sublimation of sexual energies. We might reasonably conclude that marriage entails some of each: It can become a practical compromise among competing impulses and needs that include the joys of sexual partnership as well as the economics of reproduction and CARE. This may be a practical cultural compromise among urgent evolutionary dictates.

CARE CIRCUITRY AND CHEMISTRY

Oxytocin, one of the main maternal chemicals, is manufactured in greater quantities in female brains than in male brains (Jirkowski et al., 1988). Estrogen mediates production of oxytocin throughout the cell fields of the anterior hypothalamus, including the paraventricular nucleus (PVN) and the dorsal preoptic area (dPOA). The relative importance to CARE behaviors of these two oxytocinergic brain areas has been demonstrated in rat lesion studies. Lesions on the PVN can dramatically reduce maternal behavior in first-time mother rats, although not in experienced mothers. Lesions on the dPOA, however, can totally obliterate maternal behavior (for a full overview, see Numan & Insel, 2003).

Oxytocin, like all chemical messengers of the nervous system, would be useless if it did not bind with specific chemical receptors. Estrogen and progesterone levels control the number of oxytocin receptors in many regions where oxytocin is released, including the bed nucleus of the stria terminalis (BNST) and the ventromedial hypothalamus (VMH). The BNST appears to play a more significant role in regulating separation distress. In this brain region, oxytocin appears to fuel the anxiety felt by mothers when their babies are lost and crying: Mothers will experience a chilling,

horrifying feeling until they locate their babies, and this maternal experience supports infant survival.

Although the brain structures just mentioned are key CARE players, the system's circuitry extends widely throughout medial subcortical regions of the brain, linking up with many subsystems that are essential for effective mothering. For example, there is a distinct circuit that controls milk letdown. This circuit descends from the lateral midbrain area to segments of the spinal cord that innervate the nipples (Hansen & Kohler, 1984), physiologically preparing mothers to nurse. Although only mothers can nurse their young, nurturing circuits are not the sole domain of females. Both males and females are capable of nurturing their young. And, as already noted, many CARE circuits also exist in the male brain (de Jong et al., 2009).

One aspect of the CARE circuit deserves particular emphasis. A branch of this system extends through the hypothalamus from the dopamine-producing ventral tegmental area (VTA) (Numan, 1990) to the very heart of the SEEKING system. This segment of the CARE system most likely arouses SEEKING impulses, which surely promote goal-driven maternal foraging tendencies, which are especially important for nest building and retrieving pups. Indeed, injections of oxytocin into the VTA promote such maternal behaviors, indicating that the SEEKING system is sensitive to the practical and appetitive demands of maternal life. Again, we see highlighted one of the ways in which most other emotional systems utilize primal SEEKING to fulfill their affectively rich, action-oriented functions.

Maternal urges are also supported by various nearby emotional systems such as the separation-distress PANIC/GRIEF system. As noted, this system includes the BNST, which is also rich in oxytocin networks. As we will see in the next chapter, oxytocin is remarkably effective in reducing separation distress. Presumably, when the GRIEF system is aroused, the resulting negative affective state can intensify caregiving urges in nurturing adults. Perhaps this is why mothers who identify with the distress of infants are motivated to nurture and comfort them. This hypothesis deserves more experimental attention than it has yet received. In most species that have been studied, the separation calls of infants evoke intense attention and approach behaviors in mothers, and to a lesser extent in fathers. As noted, it is not known how this occurs in the brain, but the BNST linkage provides a promising start.

Although females may be more maternal and caring, males are also constitutionally able to nurture. For example, nurturing behaviors can be induced in young male rats as well as in virgin female rats (two groups that usually avoid babies) by simply exposing them to infant rat pups on a daily basis (Rosenblatt, 1967). This process is known as *sensitization*. Nobody knows why exposure to infant animals sensitizes the CARE system in juvenile rats, but we assume that it somehow facilitates and reinforces the chemical changes that are known to arouse the system, such as increased oxytocin activity. Sensitization is more successful in very young males, perhaps because the testosterone that washes over the male brain at the start of puberty promotes a distinctly aggressive affective tinge, which often counteracts nurturant feelings. (Remember, testosterone in males promotes vasopressin synthesis, which is not a nurturant chemistry, even though it may figure heavily in parental defense of the young.) By contrast, virgin females are more readily sensitized after adolescence, because pubescent female bodies produce estrogen, which promotes the production of oxytocin.

There is every reason to believe that the CARE system generates positive affects in nurturant caretakers, both female and male. In addition to being fueled by oxytocin it is also fueled by endogenous opioids. Indeed, endogenous opioids play a role in all positive social interactions. Both oxytocin and endogenous opioids are soothing "feel-good" chemicals that are known to inhibit aggression and irritability (McCarthy, 1990; Siegel, 2005). Caring mothers, in whom these chemicals are at a high level, exhibit confident can-do attitudes (Kinsley & Lambert, 2006, 2008), urges to "tend and befriend" (Taylor et al., 2000), and at times can even escalate to a kind of maternal ecstasy.

The aggressive effects of testosterone in the brains of adult males, however, can counteract caring impulses and even promote infanticidal tendencies. In the animal kingdom, males often kill the young of their own species, but not typically their own offspring. This is probably one reason why young animals typically exhibit much more fearfulness when in the presence of adult males than females—a tendency that can further diminish bonds between young animals and adult males. Oxytocin, however, appears to inhibit the male tendency to commit infanticide (McCarthy et al., 1992). As noted in the previous chapter, male rats are less likely to commit infanticide following mating. This peaceful tendency increases gradually

and peaks just at the time when the male's own offspring would be born (Mennella & Moltz, 1988). We know that sexual activity results in the production of oxytocin in the male brain. It may also be that this elevated activity in the oxytocin system of the male brain continues to increase as long as it takes for their offspring to be born. Might this be the reason that males are less aggressive toward young animals in the weeks following fertilization? We do not know, but it is a plausible hypothesis and is ripe for exploration by an enthusiastic affective neuroscientist.

THE NEUROCHEMICAL CHANGES OF PREGNANCY

We have highlighted the role of oxytocin in CARE arousal, which is epitomized by the maternal urge to nurture newborns but which also has abundant positive antistress effects in both the mother and the child (Uvnäs-Moberg, 1998). Oxytocin manufacture is controlled by estrogen, which remains at modest levels throughout pregnancy and increases as labor approaches. Thus, at the end of pregnancy there is a proliferation of estrogen-mediated oxytocin activity. It is interesting to note that for many years researchers did not believe oxytocin was an important maternal chemical. Many believed that the sole source of oxytocin was the posterior pituitary. When the removal of this gland from nursing mothers did not inhibit maternal behavior (Slotnick, 1975), scientists reckoned that maternal behavior did not depend on oxytocin. Only the discovery of oxytocinergic (oxytocin-using) systems deep within the brain itself led neuroscientists to consider that oxytocin may be secreted in the brain and may play a central role (a role in the brain and/or spinal cord) in generating maternal behaviors such as nest building, nursing of young, hovering over them to provide warmth, and so on.

Yet the exact role of oxytocin in maternal behavior took some effort to clarify because the effects were not always clear. Although oxytocin infused directly into the ventricular system of rats as well as sheep usually promoted maternal tendencies (Pedersen et al., 1982, 1992), sometimes such experiments did *not* produce maternal behaviors (Bolwerk & Swanson, 1984). Such apparent contradictions were resolved when researchers discovered that virgin female rats typically find the odor of newborn pups to be aversive, which by itself can counteract maternal

tendencies produced by the injections of oxytocin (Fleming & Rosenblatt, 1974). When a mother rat gives birth to her first litter, however, her accustomed aversion to the smell of young pups is replaced by attraction. This same phenomenon is seen both when virgin rats are transfused with blood from postpartum rats and when they are sensitized by daily exposure to pups. The complete array of factors contributing to this rapid reversal of disgust for newborns is not known, but clearly the change is not due to oxytocin alone. Thus, chemicals other than oxytocin must play essential roles in the creation of maternal impulses.

We can understand what some of these chemical factors might be by studying the chemical changes that occur during and after birth, which is the quintessential experience that induces caring behaviors. Giving birth produces a dramatic transformation in the brain. Progesterone levels, along with estrogen and oxytocin levels, are high throughout pregnancy. Progesterone levels plummet, however, as labor approaches. At high doses, progesterone is known to act as a sedative, almost like an anesthetic in the brain. Perhaps the ebb of this hormone highlights the fact that motherhood requires increased vigilance and attention to details. On the other hand, prolactin rises sharply as labor approaches, inducing the manufacture of milk and playing a role in the generation of maternal feelings and behaviors. Although such research has been carried out primarily with rats and sheep, mammalian similarities indicate that these findings most likely pertain to other mammals, including humans.

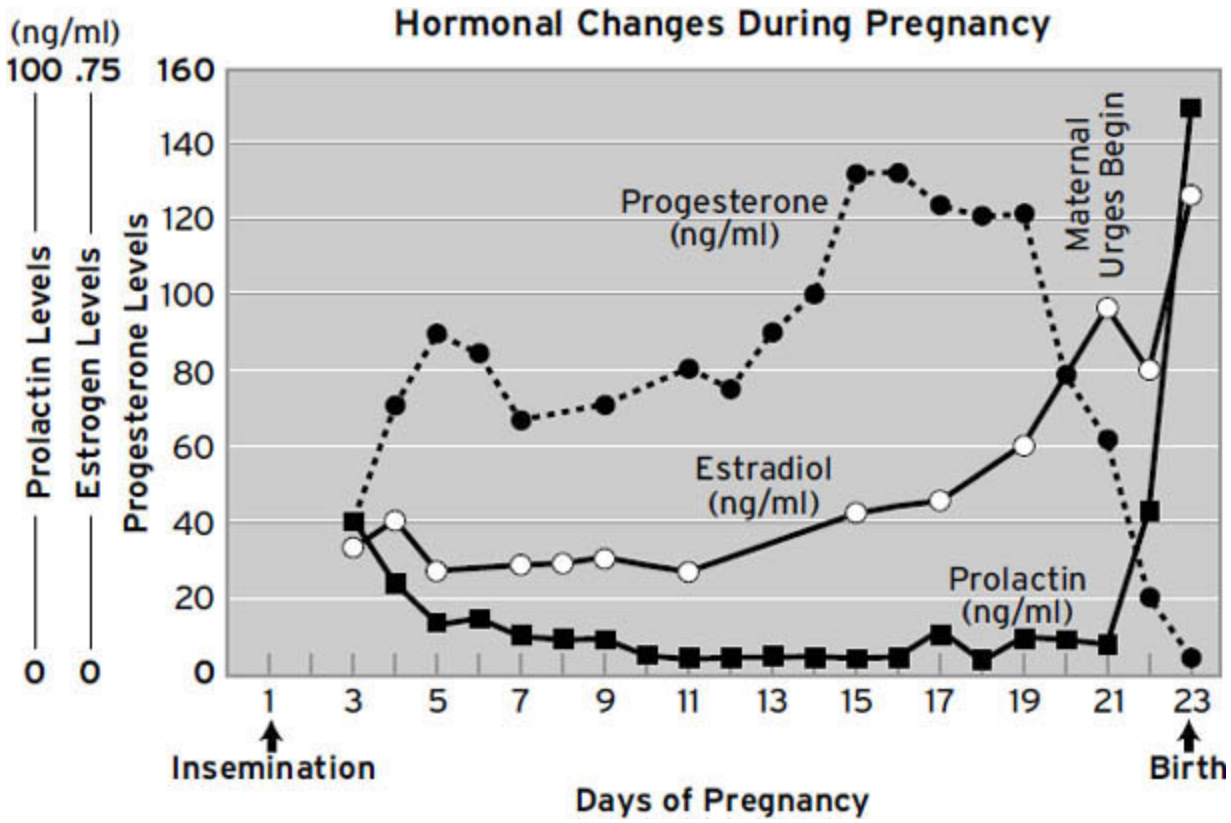

Figure 8.1. Circulating levels of progesterone, estradiol, and prolactin during pregnancy in the rat. The shifts in these hormones during pregnancy and then the rapid decline of progesterone and massive elevations of estrogen and prolactin a few days before birth establish the physiological conditions to promote maternal behavior. It is believed that the activation of oxytocin in the mother's brain is one of the most important effects that achieve this change in CARE motivation (adapted from original data by Rosenblatt, 1990, as depicted in Panksepp, 1998a; republished with the permission of Oxford University Press).

It is not known whether these chemical transformations account for the change in a female rat's attraction to the smell of pups. However, we do know that these chemical transformations play pivotal roles in the generation of many maternal behaviors and affects. Furthermore, these transformations can be artificially induced. One merely has to simulate the hormonal changes that precede parturition with the right pattern of hormone injections; this is done by rapidly increasing the levels of estrogen, oxytocin, and prolactin and by reducing the level of progesterone. This

pattern of chemical profusion and depletion reliably induces the brain transformations that produce maternal eagerness. Thus, oxytocin is only one of the maternal chemicals, and perhaps it is not all that critically important. In fact, oxytocin is not essential for the creation of maternal urges in rats. Mice whose oxytocin systems have been "knocked-out" by eliminating the relevant gene that manufactures oxytocin can still exhibit adequate maternal behaviors. However, their pups will not survive because milk production is nonexistent without oxytocin; these mice pups must have a "wet nurse" in order to live (Pedersen et al., 2006). Of course, it is possible that the maternal behavior of these animals is not as satisfying, devoted, and vigorous as mothers whose brains are full of oxytocin (Nishimori, et al., 2008).

Other research has helped define the limits of oxytocin in creating caring attitudes. For example, while oxytocin clearly contributes to the initiation of maternal urges, once maternal behaviors have been established, oxytocin is no longer critically important for competent maternal behavior. If, just as a mother rat's first pups are being born, one inhibits the influences of oxytocin within the mother's brain by administering a drug that blocks oxytocin receptors, the mother does not rapidly begin to show maternal eagerness. She seems resistant to the psychological and physiological enticement of infant pups. If this same manipulation is done a few days later, maternal behavior is not severely disrupted. It has become a habit. As already noted, a lack of maternal feeling also occurs when the PVN of the hypothalamus, a key area in the brain containing abundant oxytocin-secreting nerve cells, is damaged before parturition. This manipulation does not disrupt the birthing process itself (van Leengoed et al., 1987) but does interfere with initial maternal competence. If a first-time mother is allowed to have a few days with her pups prior to this type of restricted brain damage, however, her maternal competence is not disrupted at all (Insel, 1990; Insel & Harbaugh, 1989). Clearly, the maternal CARE system helps the brain to rapidly learn much about maternal competence. However, these nurturing tendencies quickly become so ingrained as habits that they no longer depend on the "magic" of oxytocin.

One wonders, however, if there is a long-term price to be paid for blocking the effects of oxytocin in experienced mothers. When mother rats have had past maternal experience, oxytocin is not essential in the *short run* for competent maternal behavior. But is oxytocin essential for maternal

competence in the *long run*? Under normal conditions, even after maternal behaviors have been learned, oxytocin is still released in the mother's brain. Presumably, this produces nurturing urges and associated emotional feelings that are highly rewarding for the mother. A long-term oxytocin block might, perhaps, interfere with a mother's pleasure in carrying out her maternal duties. This attenuation in subjective pleasure might lead to deterioration of maternal competence over time. This scenario has not been well studied in animal models. We also lack evidence regarding the extent to which these principles apply to humans.

Granted, the specific long-term role of oxytocin in maintaining maternal feeling and behavior is unknown. But brain chemistries frequently work by cooperation. Evidence suggests, for instance, that oxytocin enhances the effects of opioids. Thus, we find our way to a plausible hypothesis: Oxytocin may support maternal behavior in the long run because maternal competence, like all positive social relationships, is enhanced by very low doses of opioids (Panksepp, 1998a). We will see in the PANIC/GRIEF chapter that the brains of animals will secrete endogenous opioids when they are engaged in positive social interactions, such as mutual grooming.

It is not yet known whether maternal satisfactions are mediated by the brain's own opioids. It is known, however, that very low doses of opiate drugs enhance many positive social interactions, including maternal behaviors and play. On the other hand, even modestly higher doses of opiates induce a blissful but socially unresponsive sluggishness: In these cases, juveniles show less play, animals are generally less gregarious, and mothers show fewer maternal behaviors. Presumably when mothers are in a completely satisfied opiate state, they cannot experience changes in affective fluctuations that arise from and are needed for good maternal behavior. Given in tiny amounts, however, opiates promote the urge to interact in friendly ways. So it is no surprise that low doses of opiates can also enhance maternal behavior. Nor would it be surprising to discover conclusively that oxytocin facilitates such enhancement via endogenous opioid brain chemistry.

As we also discussed regarding the role of oxytocin in the LUST chapter, the power of oxytocin may sometimes be indirect, via enhanced endogenous opioid effects. With repeated exposure to high doses of opiates, the brain ordinarily becomes increasingly tolerant of, or insensitive to, these drugs. This tolerance is the main reason that addicts require ever-greater

doses in order to achieve the positive feelings they desire. Oxytocin decreases opiate tolerance, which means that small amounts of opiates in the presence of sufficient levels of oxytocin continue to exert a comforting, pleasurable effect (Kovács & Van Ree, 1985). The brains of nursing mothers secrete both oxytocin and presumably endogenous opioids, a combination that can sustain satisfying, comforting effects for a long period of time. This may be why nursing remains such a pleasurable experience for many mothers. If oxytocin were blocked, then the ongoing pleasurable effects of endogenous opioids might gradually diminish, leading to premature weaning. This could also diminish maternal performance overall, leading to various developmental problems for the young.

Certainly, one sees mothers who take care of their babies in a perfunctory rather than empathic manner. Possibly the brains of these mothers are less replete with maternal chemicals than the brains of mothers who exhibit more apparent devotion. It is possible that a deficit in primary-process maternal chemistries may lead to diminished emotional sensitivity. One might speculate that mothers with low levels of such chemicals would provide sufficient nurturing as long as their children do not experience unusual levels of distress, but certain mothers might be unable to provide empathic support under more extreme conditions. To the contrary, indifferent nurturance could be swept aside as GRIEF kicks in under more extreme emotional conditions, powerfully bridging this primal empathic gap. Again, these issues need to be resolved with more research.

Clearly, our understanding of the role of oxytocin and other social chemistries remains incomplete. We can be confident that oxytocin is important for the initial generation of maternal behaviors because an oxytocin blockade exerts decisive effects in reducing the onset of nurturance in animal models. Nevertheless, the fact that oxytocin alone does not obliterate a virgin rat's initial aversion to the smell of pups indicates that other chemicals are at work during these crucial initial phases of motherhood. A release of endogenous opioids is surely part of the feedback that sustains maternal urges: If one injects animals with very low doses of opiates, maternal behavior becomes especially vigorous, but slightly higher doses diminish it, probably partly because maternal motivation is regulated by natural fluctuations of brain opioid release. Higher than normal levels diminish social desire in general. Furthermore, although oxytocin is not essential for producing maternal behaviors in

experienced mothers, maternal behavior is weak if oxytocin systems are generally unresponsive (Nishimori, et al., 2008).

Researchers are currently investigating the roles of the many nooks and crannies of oxytocin circuitries to clarify the precise ways that oxytocin secretions generate maternal behavioral changes. We noted earlier that oxytocin feeds into dopamine neurons of the VTA and that these cells are very important in promoting SEEKING urges. Also, following birth, crucial oxytocin circuitry becomes synchronized by the development of *gap junctions*, which are direct protoplasmic bridges between adjacent neurons. Gap junctions allow for rapid, nonsynaptic coordination between neurons (Modney & Hatton, 1990). This helps oxytocinergic neurons to act in unison. For example, with such synchronization, an infant's touch quickly brings about the milk-ejection reflex. Much of oxytocin circuitry, however, remains a mystery, especially in the regulation of human social feelings and behaviors.

Less still is known about the other pregnancy-related chemicals: prolactin and the various steroids that change with imminent parturition. Both progesterone and estrogen are known, however, to promote the remodeling of certain brain oxytocin systems. We also know that prolactin promotes milk synthesis and concurrently promotes maternal behaviors in several species that have been studied in detail, especially birds. While prolactin is a very large moleule, it is actively absorbed from the bloodstream into the brain, and direct administration into the brain facilitates maternal tendencies in animals (Walsh et al., 1987). And, as explained above, decreasing levels of progesterone are also important for the onset of maternal behavior (Sheehan & Numan, 2002).

Most of this neurochemical research has been carried out on rats, sheep, and birds, but more and more information is becoming available about the operation of these systems in the human brain. Abundant work is currently being conducted with intranasally administered oxytocin (the only known way to get the neuropeptide into the human brain). The general finding from this research is that people tend to become more prosocial, that is, less aggressive, more trusting, and generally more confident in the conduct of their social affairs (MacDonald & MacDonald, 2010). The outcomes are in general agreement with our impressive understanding of these issues in animal brains. Thus, given the homology of mammalian emotional systems, and given that all mammals, including our own species, universally exhibit

maternal behaviors (with abundant differences in details among species), it is very likely that similar principles are at work in the brains of all mammals. In other words the same symphony of maternal chemicals promotes the activity of the CARE system throughout mammalian species.

Of course, in humans it is impossible to tell how much a nurturing behavior is impelled by the CARE urge or how much is guided by conscious cognitive decisions. As the most intelligent of all creatures, human beings can cognitively appreciate the importance of childcare by both parents. This fact led early investigators to entertain the idea that humans had no maternal instincts but rather that they became devoted to their children entirely through learning. Traditionally, human males have not taken care of infants. In the modern era, a conscious appreciation of the importance of nurturing the young persuades many fathers to participate in childcare. Mothers, on the other hand, have much stronger biological urges to engage infants and provide care. Thus, below the vast complexity of cognitive and cultural issues there are biological emotional motives for engaging in childcare. Because of such biological differences, most human fathers who participate in infant care probably nurture in a more routine and less deeply emotional and empathic way than mothers. Mothers typically exhibit more natural warmth and desire to be with infants. It is also usually the mothers who more persistently carry on sensitive affective communication with babies, especially with happy babies but also with those in distress.

There are abundant reasons to believe that many maternal chemicals play central roles in the maternal urges of human mothers. These neurochemicals encourage heavily pregnant women to “feather their nests” prior to the birth of their child. When babies are born, a spectrum of primary-process brain chemicals typically helps ensure that maternal care will provide joys that outweigh the burdens. Of course, because we are intelligent, thoughtful creatures, we humans also know enough to begin planning for our babies months before their arrival. We shop for clothes, cribs, bassinets, diapers, and so on. However, even human mothers seem to experience a period just preceding delivery when they engage in a flurry of compulsive preparation for their babies’ arrival. This is probably due to the many chemical changes that herald the birthing process. It seems that evolution does not rely entirely on learning in order to ensure that a mother prepares for crucial life events like the arrival of a baby. For instance, the brain consequences of

maternal chemistries even make females less prone to anxiety (Kinsley & Lambert, 2006). This makes for better mothers, and the extra attention that good mothers devote to their children produces life-long benefits for the psychological and neural strengths of their offspring. Learning, however, is part of every emotional system. This is particularly so in terms of the complex cognitive structures, unique for each individual, that buffer or exacerbate our basic urges and influence our behavior in response to these instinctual mandates.

Finally, from a clinical point of view, the chemicals that typically change levels during the end of gestation and parturition, which normally promote maternal competence, can go awry. The resulting psychophysical conditions are sometimes toxic. A few mothers fall into depression for reasons that are not fully understood. We do know that some cases of postpartum depression and psychosis have been correlated with high levels of circulating beta-casomorphin, an opioid peptide that is derived from milk. However, we do not know that this *causes* the depression. This condition, which can have catastrophic effects on mother and child alike, is usually treated by the administration of conventional antidepressants and/or by psychotherapy. If we better understood the pharmacological underpinnings of the CARE and birthing systems, we would perhaps be able to treat toxic maternal responses in more specific and effective ways. One study that needs to be done is the evaluation of whether intranasal administration of oxytocin is able to alleviate feelings of despair in those mothers that experience depression soon after the birth of their children. Such a project, in the context of psychoanalytic therapy, has been initiated by Andrea Clarici's group in Trieste (in northeastern Italy, by the beautiful Adriatic Sea). We eagerly await the results. Also, schizophrenia is often characterized by failures of social bonds, and intranasal oxytocin has recently been found to alleviate both the positive (e.g., hallucinations) and negative (social-withdrawal) symptoms of those who have descended into psychosis (Feifel et al., 2010).

VARIETIES OF MATERNAL BEHAVIOR AND MOTHER-INFANT BONDING

Although maternal brain circuits are similar among all mammalian species that have been studied, as with all emotional systems each species

obviously has unique traits that promote different intensities and patterns of maternal behaviors. Rabbits, for instance, parent briefly and infrequently, feeding their sequestered litters only once a day, spending the rest of the day grazing on nutritionally modest foodstuffs. Mother rabbits also seem to lack the motivational or neurobehavioral equipment to retrieve little bunnies that are dispersed from their sequestered nest-burrows. We don't know if rabbits actually bond with their offspring. Indeed, social bonding has only been studied in certain species, and surprisingly we do not even know whether common laboratory animals such as rats and mice actually bond with their offspring. They do not really need to bond during the first couple of weeks of life when their offspring are still "preemies" (motorically incompetent and hence incapable of getting lost on their own). In contrast, bonding is essential in herbivores, such as ungulates, whose offspring are ready to romp and run, and hence get lost, a few hours after birth. Mothers of such precocial species typically bond rapidly and exclusively to their own offspring, while those species whose babies are born immature (*altricial*) are happy to adopt.

Because of ecological factors, different species exhibit different *bonding windows*—the optimal time intervals during which mothers and infants can become attached to each other. When animals are born in an altricial state with their eyes and ears still closed (something that is common in predator species), the infants are unable to stray far from the nest. In these animals, the bonding window is large and can last for many weeks after birth. For herbivores such as sheep and bird species with precocial offspring (e.g., chickens and ducks that can forage with their mothers soon after birth), the bonding window closes within a few hours of birth (for sheep) or up to a day later (for chicks and ducklings). This short bonding window reflects the fact that maternal-infant bonding and CARE circuits are tuned to levels of infant mobility at birth. As already noted, such prey species are typically born highly mobile, so they can keep up with their mothers who are continuously on the move, foraging for food or fleeing predators. They are often surrounded by many others of their kind, in herds and flocks, which reduce dangers from predation but may bring other problems. In such circumstances, the young can easily get separated from parents soon after birth and thereby become lost in the mass of other animals. Hence, bonds have to be formed very rapidly in precocious species where the young can

easily get lost on their own. In both humans and creatures that have immature young, brief temporal windows of bonding are not so essential.

Among sheep, mothers bond to their infants very rapidly after birth and can identify their own young by odor. The downside to this arrangement is that if mothers lose contact with their young for a couple of hours soon after birth (as can be done experimentally), they commonly ignore the offspring following reunion, in fact rejecting them if they attempt to nurse. In other words, those unfortunate young who happen to get lost before mothers have the opportunity to recognize them as their own are treated as strangers upon reunion. Clearly we humans, and many other omnivores, behave much more like carnivorous species, whose infants are typically born very immature. As a result, we can more readily adopt strangers into our circle of CARE. We will return to how the short bonding window of ungulates has helped investigators decode the neurochemical nature of bonding, but for now let us focus on some key aspects of human childrearing that may have implications for our own comparatively open social-bonding systems.

UNIQUE ASPECTS OF HUMAN BONDING AND SOCIAL DEVELOPMENT

Human infants have a remarkably large and long window for social bonding. They also can readily bond to nonparental caretakers, even though initial social bonds, so essential for survival, are most commonly formed between mothers and biological infants. It is reasonable to believe that in our ancestral environments childrearing was much more of a spontaneous group activity than it is in many of our modern cultures. For instance, in traditional cultures it is not unusual for babies to be cared for by an extended family. Within the safety of a large tribal family, independence was also encouraged. Babies were often toilet trained by 1 year of age, and soon thereafter they were encouraged to behave as relatively independent members of their tightly knit, extended-family group (Hrdy, 2009; Konner, 2010). In our Western nuclear family culture, something that probably emerged during the past millennium, most parents of 1-year-olds are not yet beginning to think about toilet training. Children are allowed to retain infantile behaviors well into the first two or three years of childhood. And parents supervise them remarkably closely during these formative years. We also prize the exclusivity of the parent-child relationship and afford our

children little opportunity for independent action within the larger community. Obviously, social maturation is bound to be promoted by social circumstances where many people nurture children, as reflected in a famous African proverb: It only takes one woman to bear a child, but it takes a whole village to raise it. Our culture no longer seems to subscribe to such an adage, and this may only amplify the darker side of parenting, which we will consider now.

The maternal impulse is powerful but it is not absolute. Some pregnancies are unwanted. Some human mothers abandon their infants, leaving them in the care of extended family members, churches, or social service agencies. Often this occurs when mothers do not have enough resources to rear their children. Such practices also become more common toward older children, especially when children approach independence with the coming of adolescence. Child abuse has long been an aspect of many cultural landscapes down through history, when empathy was less of a cultural value (Rifkin, 2009). Whether such practices were less common in our prehistorical ancestral past is hard to say, but it seems likely when groups of humans were largely extended families.

Resource availability is always a concern for mothers. In times of resource shortages, it is not uncommon for rat mothers to eat their infant pups, especially if resource scarcity is so extreme that it severely compromises the ability of the mother to rear her offspring to adolescence. Another common behavior is for mothers of multiple offspring to neglect the needs of the weaker individuals, with no apparent remorse. This rarely happens in species that have a single offspring at a time. But it does occasionally happen with humans. In certain traditional societies, an infant's destiny was often dependent on economic or practical considerations rather than biology. For example, in the not-too-distant past, some societies, such as the Netsilik Eskimo of northern Canada, sanctioned infanticide, especially the killing of female infants, in order to avoid social problems in the future. Female babies who had little hope of finding an appropriate mate, because no male babies of comparable age had been born in the tribe, would be left to die in the snow, with little outward distress or remorse exhibited by the parents (Riches, 1974). Long-term social concerns overrode short-term emotional ones. To this day, in societies that prize sons over daughters, the likelihood that female, instead of male, infants will be killed is much higher. Antecedents for such practices are found in some

animal species: as we noted above, some mothers kill their weaker pups. When environmental resources are scarce, this practice can increase the probability of success for the surviving offspring. Thus, the amount of investment made in offspring is only partly an emotional issue.

However, the emotional issues in the formation of mother-infant attachments in humans, with our extended infancy and childhood, are enormous—particularly now that our children are not reared in extended families. The brain mechanisms of bonding in humans, just as in many other carnivores and omnivores, constitute a protracted course of events. Mothers in general appear to bond with their infants on an emotional level quite rapidly. Infants, on the other hand, have a considerably wider and more flexible window of bonding. Attachments in human infants are not typically fully formed until they are about one year of age, allowing them to be fostered to supportive families until then without much worry. Once formed, however, the security of attachment is all-important (as described in the next chapter). And it is critical to know how attachments are formed both in the infants' and mothers' brains. We have essentially no direct evidence regarding these neural processes in the human brain. Thus, it is necessary to generalize from research performed in animal models. A reasonable and balanced evaluation of the relevance of the data we have available can be achieved only if we recognize that we stand at the beginning of this area of discovery, with most of the frontiers lying ahead of us. We have only made a start in our understanding of these vital MindBrain functions. They have only been studied in some detail in a handful of species. And the best work has been conducted in species that show narrow bonding windows; these species may not be optimal models for illuminating human bonding.

SOCIAL MEMORIES, BONDING, AND MATERNAL CHEMISTRIES

Let us follow these threads of thought into the underlying neural mechanisms.

We do now know that both oxytocin (Popik et al., 1992) and vasopressin (Dantzer et al., 1987, 1988) strengthen social memories, because when the activities of these neuropeptides are blocked social memories are weak and slow to form in all species that have been studied so far. Because both

vasopressin and oxytocin promote positive social and sexual behaviors in animals (Chapter 7), it is perhaps not terribly surprising that these same chemistries help promote friendly social bonds and also participate in the creation of memories about those bonds. Surely, this is another example of nature's economical ways, giving us some hope that what we are learning from the other animals will also apply to us.

Starting at about the time that mammals deliver their young, most mothers have elevated levels of estrogen, which in turn promotes the production of oxytocin as well as the receptors needed to code emerging maternal urges. Since oxytocin promotes the creation of social memories, it is reasonable to suppose that it also enables mothers to remember their offspring. Research on postpartum ewes indicates that this is indeed the case. In addition to its role in creating physical and emotional maternal responses, oxytocin has been linked to the utilization of norepinephrine (NE) in social processes (Kendrick et al., 1992; Levy et al., 1993). NE is another brain chemical that plays an important role in the creation of olfactory memories in ewes. Positive olfactory social memories are formed when new neural pathways that promote positive social engagement with offspring are created in a ewe's olfactory bulb. If the NE activity is blocked (by the antagonist, propranolol), these pathways are not so numerous. Olfactory memories are thereby compromised. As a result, mother ewes who have received the NE blocker are significantly less able to discriminate between their own and other lambs (Levy et al., 1995). Under ordinary conditions, these brain mechanisms operate very rapidly in ungulates such as sheep, helping to ensure that mothers provide exclusive nursing rights to their own offspring.

NE appears to facilitate the creation of olfactory memories in the following way: When released in the olfactory bulb, NE reduces the activity of GABA, which is the main inhibitory neurotransmitter in the mammalian nervous system. When GABA's inhibitory influence is reduced in the olfactory bulb, relevant smell-coding neurons become more active, firing more vigorously. This rapid firing stabilizes the neural pathway that encodes the smell of the lamb. When a pathway is stabilized, it endures over time, which means that it becomes a memory pathway. It is apparently in this way that mothers become attached to the unique odor signature of their infants. Similar processes may be operating within infants, even in human beings, which is a species with a relatively modest sense of smell

compared to other mammals. Quite a few studies have shown that human babies develop a selective attraction to the smell of their mothers' breasts. Presumably this attraction is reinforced by the positive affective feelings engendered by oxytocin release in the brain, as well as by the arousal of associated "feel-good" neuropeptides, such as endogenous opioids. In many species, such social attractions are further reinforced by gentle touch, a stimulus known to promote both brain opioid and oxytocin release (Panksepp, Bean et al., 1980; Matthiesen et al., 2001).

Indeed, social bonding may be an addictive phenomenon, as first surmised by Panksepp and his students. The relationships between social attachments and the bonds that people form to opioid use have remarkably similar characteristics (see Figure 8.2). This is the idea that provoked us to begin the first neuroscientific inquiries into the nature of social attachments (see Chapter 9). However, the fact that there are many other chemicals involved is no surprise. Every brain function is mediated by a multitude of brain chemicals. Although the opioid hypothesis has withstood the test of time, as we have already seen, oxytocin is very influential in allowing a mother to make the transition to a sustained caring affair with her infant, yielding sustained nurturance.

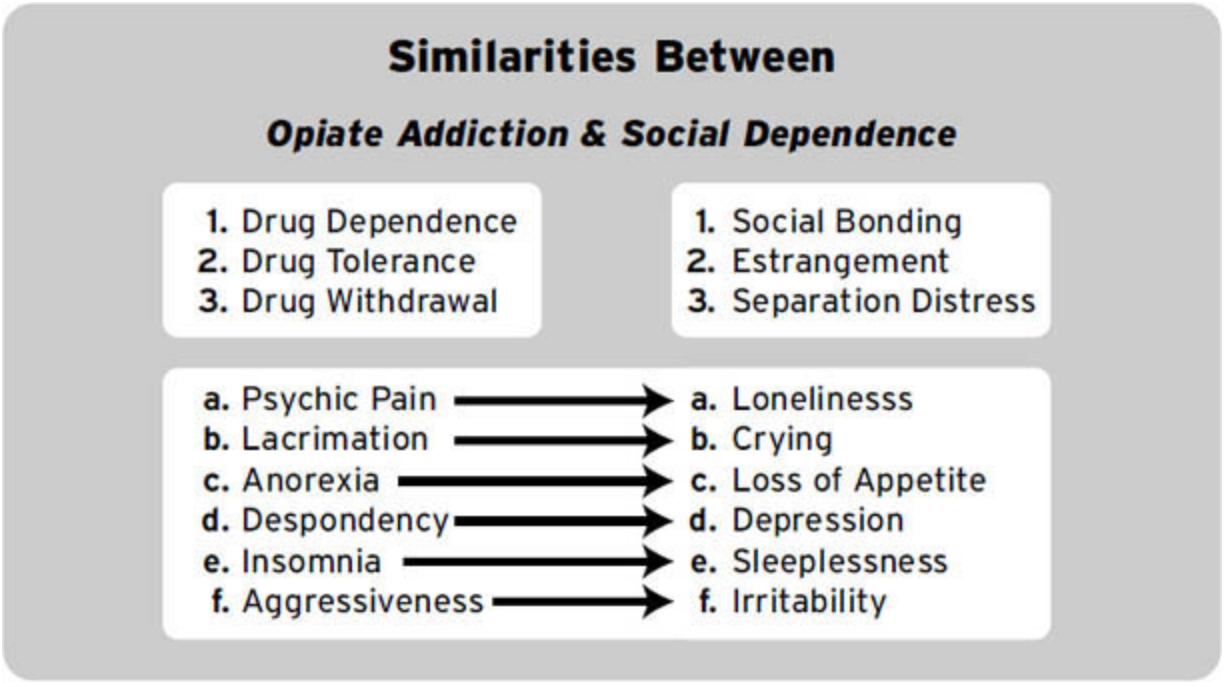

Figure 8.2. A conceptual summary of the first theory that social bonding is an addictive phenomenon, based on major similarities between the dynamics of opioid dependence and key features of social attachments. Both show very similar psychological dynamics, and this suggests that opiate and some other addictions are so affectively compelling because they utilize the same brain emotional systems. This idea was first developed in the late 1970s (from Panksepp, 1998a; republished with the permission of Oxford University Press).

We mentioned earlier that ewes have a very short bonding window, lasting no more than a few hours. If the mother does not have access to her lamb during this window of time, social memory pathways that promote maternal attractions will not form in her olfactory bulb and she will have no enticing imprint of her lamb's smell. However, researchers have found that the bonding window can be artificially re-opened for a couple of hours if the ewe is provided with stimulation to the vagina and cervix (Keverne et al., 1983; Kendrick et al., 1992) via a procedure straightforwardly referred to as vagino-cervical stimulation (VCS). VCS produces a number of responses in the ewe's central nervous system, one of which is a surge of oxytocin release (Levy et al., 1995). Because the normal mammalian birthing process stimulates the ewe's vagina and cervix, it seems likely that the oxytocin released by VCS helps generate a new ~2-hour social bonding window where the smell of a young animal becomes attractive again. In a finding that demonstrates a similar attachment response in infants, it has been shown in rats that administration of oxytocin into young rat pups can make the mother-associated odors more attractive (Nelson & Panksepp, 1996).

Although ungulates seem to bond primarily through olfactory mechanisms, as do many rodents, humans bond through sight, sound, and touch. We simply do not know whether bonding through these senses also operates through oxytocin mechanisms. We do know, however, that touch has been found to recruit opioids, which are as important as oxytocin in social bonding (see Chapter 9). Sound, as a source of bonding, has especially interesting implications, because the engaging intonations of mothers' voices may be a gateway to both the learning of language and our love of music (Panksepp, 2008b; Panksepp & Trevarthen, 2009). The

auditory system is incredibly rich in opioids, especially in the lower midbrain levels of the inferior colliculus (Panksepp & Bishop, 1981). Fetuses begin to integrate extrauterine sounds, even recognize their mothers' voices, before birth (Busnel et al., 1992; Kisilevsky & Davies 2007) and perhaps to imprint on those melodic intonations—the *motherese*—that will eventually open up the full potential for language acquisition.

OXYTOCIN AND THE AFFECTIVE POWER OF MUSIC

We have noted that many sensory portals promote social attachments. We think this occurs, in part, by boosting oxytocin activity in the brain. We have also discussed CARE sensitization, as exposure to very young animals can gradually evoke the emergence of nurturant behaviors in juvenile rats and adult virgin female rats. In the last section, we noted that VCS re-opens the bonding window in mother ewes, although we do not yet know if VCS is a universal mechanism that promotes oxytocin-driven bonding. There are probably many external stimuli that arouse the CARE system, but one that certainly should receive further study is music.

There are many reasons to believe that soothing music can release oxytocin in the brain. Farmers have long claimed that their cows yield more milk when listening to particular types of music (not a well-documented claim), and in our research on separation distress we have found that music can reduce separation-induced crying in young chickens (a very robust effect). While doing those strange experiments, we also found that when we play music to newborn chicks, we can evoke the same explicit behavioral profile as when we infuse oxytocin directly into their brains. During the first week of life, young chicks exposed to either music or oxytocin or vasotocin exhibit remarkably high levels of three very distinct behaviors: (i) very frequent lateral head shaking, (ii) vastly increased rates of yawning, suggesting a relaxed state of mind, and (iii) moderately elevated wing-flapping, suggesting decreased social inhibition (Panksepp & Bernatzky, 2002). Indeed, this last behavior, probably reflecting something like confidence, is vastly increased if animals are tested in small social groups as opposed to individually (Panksepp, 1992). Confidence is among the several key functions of bonding chemistries—they make animals feel socially comfortable as well as confident, because they provide an

affectively secure neurochemical base within the brain. As we will discuss in the next chapter, intracerebral infusions of oxytocin are remarkably effective in reducing separation distress in young animals.

The power of music to arouse brain oxytocin may be the implicit "force" that lies at the heart of a wonderful 2004 semidocumentary film by National Geographic entitled *The Story of the Weeping Camel*. This film shows how a family of nomadic shepherds in Mongolia's Gobi Desert induced a mother camel to bond with her newborn white colt after a very difficult, 2-day-long labor and delivery. Perhaps because of the prolonged stress of birth, which long outlasted an ungulate's typically short bonding window, the mother refused to accept and nurse her colt. The rejected colt wailed pitifully for many days, often at quite a distance from the mother. The shepherds had to provide milk by hand. In an attempt to solve this dilemma, the nomads conducted a traditional "reunion ceremony" for which they enlisted the services of a renowned Mongolian musician to render a moving background melody as the female head of the family sang a lullaby to the mother camel, all the while gently stroking her neck and body. Throughout this moving cross-species interaction, the young colt was coaxed to seek out his mother's teats. Mother and infant were encouraged to engage with each other. These shepherds, with the help of soothing music and touch, sought to open the bonding window once more. The deep sense of emotional harmony that was established slowly worked a spell, not only on the human audience (the movie was nominated for an Academy Award) but also on the mother camel. Ever so slowly, the camel's heart opened to the colt and she accepted him into the embrace of a lasting mother-infant bond. There is a lesson here for humankind.

PROMOTING MATERNAL FEELINGS AND BENEFITS FOR INFANTS

Animal research has clearly shown that once a mother has exhibited competent and devoted maternal behavior following the birth of her first offspring, her maternal abilities remain perpetually elevated. This is a striking example of life-long emotional learning. How these accruing benefits of maternal experience are coded in the nervous system is not known. But presumably they are due partly to lasting changes in both the underlying CARE circuits and in the associated memory networks that

encode various maternal skills, as well as the ability to deal with the world in more confident and effective ways. Motherhood makes animals more courageous in formal tests of fearfulness, such as the elevated plus-maze (see Chapter 5). They also remember locations of food better than virgin females, as evaluated by performance in an eight-armed radial maze where the speed and pattern of food finding can be easily quantified (Kinsley & Lambert, 2006).

Although the underlying neurobiological nature of such intensification of maternal urges is not well understood, a vast amount of important research has been conducted to evaluate the long-term benefits of maternal nurturance on the constitutional strength of the offspring. Infants who have received abundant and consistent tender loving care from mothers have been given a great gift. They are emotionally and physically benefited for the rest of their lives. It should come as no surprise that good mothering is good for infants, but the neuroscientific details that back up this fact are truly spectacular.

The key work has been done by Michael Meaney's laboratory at McGill University. Meaney and colleagues have evaluated how the amount of maternal touch in rats, particularly ano-genital licking—a prominent part of the maternal routine in mother rats—influences the emotional and cognitive abilities of young rats later in life (Meaney, 2001, 2010). In brief, Meaney and his colleagues have found that many life-long benefits emerge in the brains of rat pups that have been most abundantly licked and attended to by their mothers. Abundantly licked rat pups grow up to be less anxious, more resistant to stress, and more capable of exhibiting learning and other adaptive behaviors throughout their lives. These effects are accompanied by many demonstrable changes in their brains including (i) diminished stress hormones (i.e., corticotrophin-releasing factor [CRF] and adrenocorticotropic hormone [ACTH]), (ii) more GABA receptor sites, promoting reduced anxiety, and (iii) more receptors for glutamate and norepinephrine, which facilitate learning. Emotionally these animals are less anxious, showing more activity and fearlessness and better learning and performance in a variety of fear-inducing situations (Champagne et al., 2003; Zhang & Meaney, 2010).

In short, abundant maternal care sets in motion a series of epigenetic changes in gene expression patterns that make "well-loved" animals more resilient with robust, life-long resistance against various stressors. Animals

that did not receive abundant maternal devotion are more emotionally fragile, and hence they are more susceptible to being overwhelmed by stressful life events. In the next chapter, we will discuss in greater detail how the lack of secure bonding impacts the development of the minds and brains of human infants.

Some of the neurogenetic mechanisms that mediate these effects have also been brought to light through research. Social temperaments of animals can be modified by either promoting or diminishing the genetic expression of oxytocin or vasopressin (Donaldson & Young, 2008). Not only do these changing neurochemical tides modify primary-process social responsiveness, but they also percolate through the nervous system to regulate many higher brain processes such as social memories and cognitions (Ross & Young, 2009).

Although much of the focus of our coverage is on the primary-process aspects of mammalian emotionality, learning is always a feature of how these systems manifest themselves in the lives of animals. Thus, CARE in the real world is broadened by some experiences and narrowed by others. In a seminal study, Lonstein and De Vries (2000) documented how this happens in prairie voles where mothers and fathers typically share parental duties, and how the tendency to nurture young varies according to life experience. Among the findings were the following: (i) Virgin female prairie voles are more nurturant when younger; (ii) merely exposing virgin females to young pups for 2 days after they are weaned increases later maternal responsivity; (iii) young virgin females that grew up with their parents and siblings exhibited particularly outstanding parental behavior; and (iv) simply growing up with *both* parents was sufficient to increase CARE motivation in such females; but (v) this elevation was only seen if both parents were *present during early development*. These findings speak loudly to the role of intrinsic "family values"—of the most obvious sort—increasing nurturing motivations in the young. In more recent research, the effect of biparental early experiences that promote various caring behaviors in prairie voles, as well as long-lasting salutary effects on brain chemistries such as oxytocin and corticotrophin stress systems, have been documented (Ahern & Young, 2009).

CLINICAL IMPLICATIONS

Just as the story of life on earth has been illuminated by the insights of Darwinian natural selection, neuroscience has enriched our understanding of the adaptive mechanisms that Nature has built into mammalian brains through ages of evolutionary experience. The abilities of young animals to reach reproductive maturity and to provide nurturance to the recipients of their genetic legacy are critically linked to the quality of parental devotion. The ways in which motherly, and fatherly, CARE help nurture the brain are of great importance for understanding how altruism, compassion, and empathy became possible. The life-long benefits for brain and behavior initiated by motherhood are remarkable (Fleming et al., 1999; Kinsley et al., 2008).

In years to come we will undoubtedly learn much more about the CARE system. New therapeutic methods might alter brain CARE chemistry and related social-emotional systems. Such interventions could help parents experience nurturing affects more consistently, and display supportive behaviors more effectively. Facilitation of oxytocin activity may promote the kinds of accepting, positive, prosocial feelings that can increase confidence in one's capacity for greater emotional openness. Indeed, in a series of recent studies, it has been found that plasma oxytocin in mothers increases with the abundant affectionate contact with their babies (Feldman et al., 2010). Likewise, intranasal oxytocin facilitated the quality of fathers' play with their children (Naber et al., 2010). Many investigators studying this neuropeptide are beginning to suspect that oxytocin may have an important role in future psychotherapeutic interventions aimed at strengthening positive social-emotional feelings in people who are bogged down in personal doubts and insecurities (see Feifel et al., 2010; Panksepp, 2009c; Young & Wang, 2004). As brain science progresses, many other viable tools will become available to help with the healthy reintegration and recontextualization of the affective and cognitive dimensions of troubled lives.

It has long been known that the most effective psychotherapy occurs when clinicians know how to approach clients with unconditional acceptance, empathic sensitivity, and a full concern for their emotional lives. In a word, effective psychotherapists share their ability for CARE, along with the ability to recruit the healing power of positive emotions. And this lesson is not just for those whose professional focus is to help heal the mind, but also for those harried clinicians who are more involved with

bodily than mental health, and who, all too often, do not have sufficient time for the emotional concerns of their clients (Goleman, 2006). Of course, the loving touch does not need much time. But it does need consistency.

CHAPTER 9
Born to Cry

The PANIC/GRIEF System and the Genesis of Life-Sustaining Social Bonds

If guardian angels yet there be
then why am I alone
with troubled heart and vacant eye
—a lapse where once there shone
the light
of my now accursed life?

If love is all then what eclipse
dimmed care and let things fall?
What heartbeat skipped
and let rain down the blow
that took my all? . . .

—Anesa Miller, "Time of Grief" (1995)

ONE OF THE MAJOR SOURCES of depression is the psychological pain that consumes the mind as a result of unresolved grief. The first two stanzas of the above poem convey the misery of profound loss—in this case, the death of a child. This poem concludes, "*If time heals all then let it pass on wings I cannot feel or see . . . Companionship and kindness give moments of relief like angels, slow and silent, moving through the time of grief.*"

It was written in 1991 by my (JP) companion, Anesa, a few months after the death of my daughter Tiina and three other beautiful teenagers. All were

killed by a heavy drinker, nicknamed "Suds," whose blood alcohol level was so far beyond the legal limit that most men would have been unconscious. He was recently divorced and had been drinking all evening in an angry mood, partly because his wife would not provide access to his own children that Good Friday. He was very angry, and he hit the road, drunk to the brim. Around midnight I was called to the hospital: "An accident, perhaps my daughter was involved." I rushed there. Tiina and two friends were dead, one was dying. And one had survived, miraculously almost uninjured physically, from the side impact to their car.

I was alone the rest of that night; Anesa was in Washington, D.C., at a conference. She rushed home. My grief and anger seemed endless . . . for a long time. Three other families were also devastated. All because a reckless drunkard went speeding like a "bat out of hell" with a "cowboy" cop close behind, chasing Suds heedlessly down the middle of a lonely country road, at night, without his overhead lights or siren on. This, at least, was the testimony of a caring family who was having Good Friday dinner at their home that was situated at the intersection where the accident occurred. The community was polarized as law enforcement organizations tried to suppress the evidence of inappropriate police actions, and doctors refused to release blood alcohol results of the recovering drunkard, because of "doctor-patient confidentiality." For some time, our small university town was torn asunder by the taking of sides, as a result of intentional misinformation "from above." This tear in the social fabric was not easily healed by the authorities' actions.

That night, I cried for the first time since I was a child. For a long while I experienced deep grief and depressive sadness with little hope of resolution. It did not help, indeed it perplexed my mind, that this was happening to me, as I was a neuroscientist who was trying to empirically illuminate the ancient brain mechanisms of separation distress, one of the major emotional sources of our earliest social bonds. Without a loving companion and caring friends, chronic depression would surely have settled in. My descent into darkness was also partly relieved with antidepressants (used wisely, namely symptomatically, rather than at high continuous doses that can shift brain neurochemical balances that can create additional problems). The separation-distress mechanisms of the mammalian brain are believed to open the gateways to human grief (Freed & Mann, 2007) and then to sustained depressive despair, especially when the initial pain of separation

is due to the loss of parents occurring early in life (Bowlby, 1960, 1980; Heim et al., 2004; Watt & Panksepp, 2009).

What we will be talking about in this chapter is captured in a remarkable passage from James Saunders's play *Next Time I'll Sing for You* (1962):

> There lies behind every thing, and you can believe this or not, as you wish, a certain quality which we may call grief. It's always there, just under the surface, just behind the façade, sometimes very nearly exposed, so you can dimly see the shape of it as you can see sometimes through the surface of an ornamental pond on a still day, the dark, gross, inhuman outline of a carp gliding slowly past; when you realize suddenly that the carp were always there below the surface, even while the water sparkled in the sunshine, and while you patronized the quaint ducks and the supercilious swans, the carp were down there, unseen. It bides its time, this quality. And if you do catch a glimpse of it, you may pretend not to notice or you may turn suddenly away and romp with your children on the grass, laughing for no good reason. The name of this quality is grief.

THE PAINFUL SOURCES OF SOCIAL BONDS

This chapter examines the dark side of our capacity for love and play. A simple fact of life, with profound neural consequences and mental health implications, is that we become attached to—we love—those who nurture and befriend us. It is becoming increasingly clear that *mothers and loving others* are the ones who can offer us the gift of a happy life (see Chapter 8; Hrdy, 2009). The evolution of caring feelings, and social bonds, may have been the passage that also amplified our mammalian capacity for GRIEF. The separation-distress mechanisms of the mammalian brain are believed to open the gateways to human grief (Freed & Mann, 2007) and then to sustained depressive despair, especially when the initial pain of separation is due to the loss of parents occurring early in life (Bowlby, 1960, 1980; Heim et al., 2004; Watt & Panksepp, 2009).

Our earliest social bonds, when firm and secure, nourish our psychological health for a lifetime (Bowlby, 1980). A secure and warm maternal relationship is the primary key to a happy life. In humans, these life-saving bonds begin to form ever so slowly after birth, because we are born physically and psychologically immature—in a sense, we omnivores and carnivores are all born as preemies. Most herbivores are ready to run with their mothers soon after birth, and bonds are formed promptly and are solidified by nursing—the first suckling being especially important. By contrast, it is not until the first half year of life that we humans begin to

really cry in response to pure social separation, as opposed to just bodily distress—to exhibit separation-evoked distress vocalizations (DVs) when a mother, or any other primary caretaker, leaves us alone in a strange place. Mary Ainsworth and colleagues (Ainsworth, 1982; Ainsworth & Boston, 1952) first studied this type of crying and complaining in human children.

A few ethologists had already noted that young animals emit DVs when separated from their mothers, a fact known by all who have reared chicks and ducklings. Indeed, Konrad Lorenz (1935) demonstrated how young geese become attached to ("imprinted" on) their mothers, following closely behind her and persistently crying and searching for her if separated for just a few seconds. The study of the brain mechanisms of these panicky separation calls is perhaps the most rigorous neuroscientific gateway to understanding the brain mechanisms of the psychological pain and grief that seem to dispose many organisms to depression (MacDonald & Jensen-Campbell, 2011; Panksepp, 1981b, 1998a, 2010b, 2011a; Watt & Panksepp, 2009).

Without the succor of stable social care and secure bonds to loving others, human infants will pine away and die (Bowlby, 1953; Spitz & Wolf, 1946). The warm affective feelings of security that arise from loving attachments—the primary mechanisms of the "secure base"—are gradually transported to higher forms of consciousness around 2 to 3 years of age. Throughout the first six years of childhood, early social loss—excessive separation distress/GRIEF—sensitizes the child to chronic anxiety and insecurity, often heralding depression later in life. Loving social attachments, on the other hand, strengthen the positive affective powers of the brain, promoting healthy actions of PLAY (see the next chapter), which are fundamental psychological forces that helped make humans, indeed all mammals, the sophisticated social creatures that we are. We respond intensely to uncaring emotional gestures directed toward us; anything that hints at shunning or even milder forms of social exclusion is experienced as psychologically painful (Eisenberger, 2010). With the development of higher mind functions, namely with the developmental programming of the neocortex, we become profoundly intersubjective creatures who care deeply about the quality of our social networks (for a superb popular summary, see Goleman, 2006).

We are just coming to terms with the brain mechanisms of the profound sadness that arises from social loss. The wonderful imaging of the higher

brain mechanisms that are recruited during grief and sadness (Freed et al., 2009) often fail to do justice to the ancient subcortical mechanisms for separation distress (Panksepp, Herman et al., 1980), a neuroanatomical trajectory that was eventually confirmed with human brain imaging, as summarized in Figure 9.1 (Damasio et al., 2000; Panksepp, 2003a). Under comparable testing conditions, where volunteers were requested to generate four distinct emotions from their memories—namely GRIEF, JOY, RAGE and FEAR (see Fig. 12.1)—with strong feelings of sadness, the GRIEF system showed the clearest and most extensive arousal. The general anatomy of human GRIEF was the same as the system that mediates separation calls, as mapped in animals. This key system for feeling the sting of social isolation appears to have arisen evolutionarily from brain systems that mediate the affective intensity of physical pain. It is also noteworthy that this ancient subcortical brain system for affective aspects of pain is different than systems that loop to higher brain regions to mediate the cognitive-discriminative aspects of pain.

As noted earlier, we formerly called this the PANIC system because when young animals are abandoned, they experience a special form of alarmed anxiety—an agitated panicky state. We favored this term because there were good reasons to suspect that panic attacks stemmed, in part, from excessive arousability of this primary emotional system (for a recent overview, see Preter & Klein, 2008). However, many readers found the label confusing, probably because when older people are deprived of companionship, they tend to feel lonely and sad rather than panicky like little children. Of course, this only reflects the tertiary-process ruminations of adults, who have a lifetime of ways to cognitively adjust to social loss, lessons that young children have yet to learn (for a contemporary discussion of human loneliness research, see Cacioppo & Patrick, 2008). Nevertheless, because of confusions about our intent in using the term PANIC, we have simply decided to call this PANIC/GRIEF system GRIEF in this chapter.

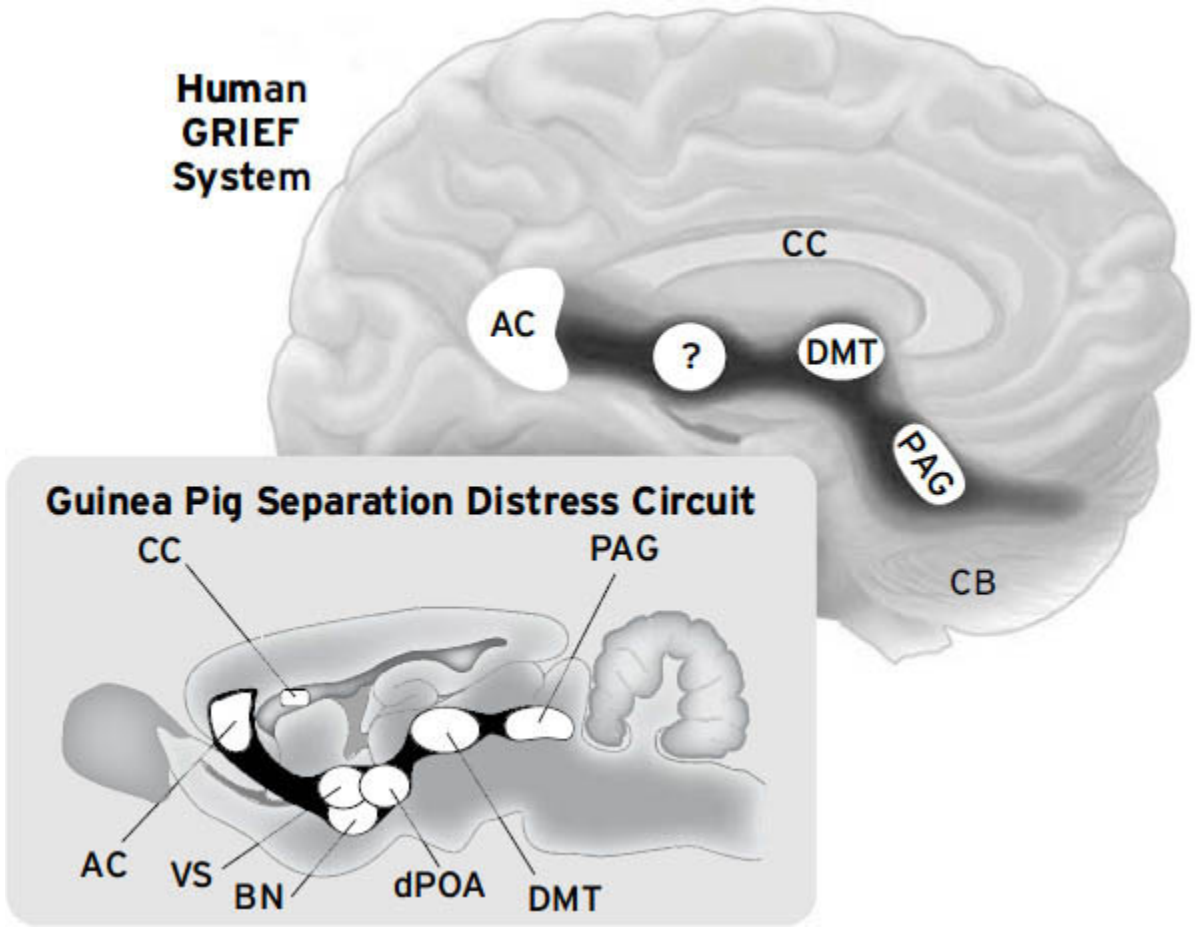

Figure 9.1. A schematic summary of guinea pig brain regions where one can readily evoke social separation–induced vocalizations in young guinea pigs (as mapped in Barbara Herman's doctoral dissertation, Bowling Green State University, 1980). Very similar anatomies were observed in domestic chicks (Paul Bishop's doctoral dissertation, BGSU, 1984). When Antonio Damasio and colleagues (2000) published PET scan images of human beings feeling very sad, a very similar anatomy was evident, suggesting that all warm-blooded vertebrates share the social cohesions that mediate the GRIEF system. The areas that lit up were in the anterior cingulate (AC), the dorsomedial thalamus (DMT), the periaqueductal gray (PAG), and also regions in the most ancient parts of the cerebellum (CB). Animals showed remarkably similar anatomies, including the ventral septal area (VS), dorsal preoptic area (dPOA), and the bed nucleus of the stria terminalis (BN), which are too small to be accurately identified on human PET images. Thus, the question mark is there in the human depiction (this figure was

first published in Panksepp, 2003; adapted with the permission of the American Association for the Advancement of Science).

This system has two prominent and opposing facets. In the first instance, arousal of the GRIEF system makes us feel bereft and miserable. But, when distress is alleviated—when we once again are emotionally enfolded in our secure attachments—we feel a deep sense of comfort and security, probably through the release of CARE chemistries such as endogenous opioids and oxytocin. Throughout our lives, we continue to feel complete and at ease when in the company of people whom we love and trust. With our emotional kin, we have the feeling that "everything is alright." It is this feeling, mediated substantially by the above social neurochemistries, that strengthens social bonds, as discussed in the previous two chapters.

The identification of the GRIEF system and its neurochemical controls by our research group in the mid-1970s provided the first neuroscientific insights into the affective mechanisms of our ancient social minds. It became clear that social bonding was in part an addictive process, obtaining its affective intensity from some of the same brain systems that also promote narcotic addictions. More recently, it is clear that the positive affect of SEEKING also contributes to adult social-sexual attachments (Insel, 2003).

While separation distress is clearly exhibited by most young mammals that have been studied, and it can be touching to witness in these animals, it is most poignant when we hear our own children cry. Any mother who frequents the supermarket recognizes the psychologically distressed screams of a child who has become lost in the mayhem of busy shoppers. These cries pull at our heartstrings. Unlike the demanding protest of a youngster who has been denied a treat, or even the vigorous distress of one who has fallen and been hurt, the cries of lost children have the unmistakable ring and urgency of panic. The feeling that is aroused appears to have little to do with the angst that can be generated by our FEAR system. The child does not hide or flee as it would from a source of danger. It does not freeze in an effort to avoid notice by a predator. Rather, the child is apt to run around frantically (perhaps a SEEKING response), crying and attracting attention. In many species, the crying gets more intense once the mother is in sight, or upon having momentarily been reunited with the

mother—a phenomenon called "maternal potentiation," which is probably a secondary, a learned, rather than a primary response of the system (i.e., one that requires prior social bonding). Species that do not show such specific maternal facilitation, like most laboratory mice and rats, presumably do not have *real* social bonding, the discrete bond between a mother and infant. They simply exhibit a more generalized social reward process.

It may disappoint many researchers to learn that those oh-so-convenient laboratory mice and rats are not ideal species for studying juvenile social-bonding mechanisms. They sometimes exhibit what has been called maternal potentiation (Shair, 2007), but it seems likely that their calls are potentiated by any female, not just by the pup's own mom. It may be simply a generalized dopamine-mediated SEEKING response, rather than an amplification of distress. In other words, it is by no means yet clear that the rebound in calling that has been occasionally observed by a brief period of maternal reunion is specific to one's own mother. Thus, it is of critical importance to determine the specificity of social attachments in other animals commonly used to model such social processes, to be sure they do in fact resemble the human condition. This is one reason we have spent much time evaluating the utility of another convenient small rat-size species —the *Octadon degu*—that shows a rich social repertoire, including real bonding, separation-distress, and social play at overlapping phases of life (Colonnello, et al, 2011).

It is pretty obvious why young animals cry when separated from their mothers. There is an adaptive value in such childhood misery, because when very young animals feel frightened and alone, their cries alert parents to come rescue them. Imagine the striking image of a young sea otter on the open sea, completely dependent on its mother for food and care. When a mother dives into the depths in search of sustenance, she must leave her infant unattended for many minutes. In her absence, the youngster becomes agitated and cries persistently. These DVs, which some scientists also refer to as "isolation calls," alert the mother where her baby is to be found. If there were no such emotional communication, a mother otter that has lost her bearings while diving might be permanently separated from her infant, who would then be lost forever. Thus, the security, indeed survival, of the infant is unequivocally linked to the audio-vocal thread of attachment that joins it to its mother.

The same is true for all mammals. At the outset of life, our dependency is complete and our survival rests on social bonds created with those who care for us. The all-important mother/infant emotional bond is easily monitored by the infant's cries of distress that typically occur when the mother is absent. Indeed, these DVs are the cardinal signs of aroused GRIEF. It was through the study of brain networks that evoke such cries of distress that we first came to understand the anatomy and chemistry of the psychic pain that can lead to depression. Likewise, when young children receive poor care—when abandoned, neglected, or abused—they endure an ongoing sense of insecurity and longing arising from these same networks that can promote lifelong personality problems. And the sustained arousal of GRIEF can promote chronic mood disorders (Watt & Panksepp, 2009), perhaps by eventually depleting the joy-of-life resources of the SEEKING system (Coenen, et al., 2011; Panksepp & Watt, 2011).

The capacity for GRIEF is not limited to mammals. Birds also have the affective imprint of social needs engraved deep within their brains, thanks to unknown remote ancestors shared by both birds and mammals. Although we know little about such distant relatives, we do know that the DVs of birds and mammals arise from very similar brain regions and are regulated by the same neurochemistries (Panksepp, Herman et al., 1980; Panksepp, Normansell, et al., 1988). When this GRIEF system is aroused, animals are intensely motivated to seek reunion. These facts dramatically indicate that GRIEF is an ancient affective system of the BrainMind of many vertebrates, one that mediates an intense affect that is well described as "psychological pain." While this emotion has very ancient roots, its influence is apparent in the way we construct our modern civilizations (Rifkin, 2009). And the functions of this ancient emotional system have a broad array of implications for feelings of well-being and misery throughout human lives (for a recent summary of the breadth and depth of psychological work in the area, see MacDonald & Jensen-Campbell, 2011).

The feeling of GRIEF, painful as it is, is essential for the survival of the young. Also, the alleviation of the acute pain of GRIEF—relief from the misery of social isolation—may tell us much about the nature of love. As already noted, some of the brain chemicals that fuel the positive affects aroused by social reunion are similar to opiates, which are the drugs that people readily abuse because of the powerful feelings of emotional comfort they provide. Brain opioids, in addition to oxytocin, and probably brain

prolactin as well, are secreted onto the chemical receptors of needy GRIEF networks when people and animals touch each other and form positive social bonds.

The action of these care chemistries is one major reason we derive emotional comfort from warm social relationships with family and friends. Mammalian and avian kin-groups become "addicted" to each other's company, thereby forming social bonds that allow them to live in harmonious societies (Panksepp, 1981a; Panksepp, Herman et al., 1980). Obviously, living in societies enhances the survival of all social species, so there is every reason to believe that our desire to live with each other, an affinity affirmed first through the mother-child relationship, is a natural process of our emotional brains. This feeling is not something we have to learn, but we do have to learn with whom we can relate in such open and "intimate" ways (Reddy, 2008). We also have to learn to build social structures that cultivate this better side of our nature (Rifkin, 2009).

HISTORICAL PERSPECTIVES ON SOCIAL-EMOTIONAL ATTACHMENT FUNCTIONS OF THE BRAIN

It is sobering to reflect that in the middle of the twentieth century behavioral scientists, and even psychoanalysts such as Freud, at least in his initial vision, believed that social bonds emerged purely as a result of being well fed, to put it bluntly. The idea was that young children loved their parents simply because the parents provided nourishment, shelter, and warmth. Presumably these "reinforcing" experiences were universal among human beings, because human babies are physically immature and rely on parents to meet their needs for food, water, and warmth. In other words, the behaviorists assumed that children love their parents through learned associations with conventional rewards—simply because parents provide life-sustaining necessities. Likewise, the behaviorists believed that children would not have a reason to bond with caretakers if they did not fulfill their physical needs. There was no thought that children or young animals had inherent needs for social attachments above and beyond the satisfaction of physical needs. Some of the most notorious advice was provided by John Watson, the father of behaviorism, who raised his own children with little affection. His most famous piece of advice for proper child rearing was to "never hug and kiss them, never let them sit in your lap. If you must, kiss

them once on the forehead when they say good night. Shake hands with them in the morning. Give them a pat on the head if they have made an extraordinary good job of a difficult task" (from his *Psychological Care of Infant and Child*, 1928, which sold over a hundred thousand copies within months after publication). All his own children had severe emotional problems, perhaps because of parental aloofness, including a daughter who attempted suicide many times and a son who succeeded in taking his own life.

The prevailing behaviorist view gradually lost ground when classic studies by Rene Spitz revealed that human babies failed to develop normally when reared in orphanages that provided good physical care but little affection (Spitz & Wolf, 1946). Without caring human contact, many babies died prematurely, while others exhibited severe emotional abnormalities as they grew up. In recent times we have observed this once again in orphanages such as those that existed in Romania before the dictator Ceausescu was deposed. As Spitz discovered in Germany 40 years earlier, such infants languish and fail to thrive without the balm of sustained human love. In order to flourish, babies clearly need emotional sustenance in addition to the physical necessities.

Syndromes similar to human failure to thrive and emotional stunting exist in other animals as well. Researchers in the middle of the last century came to recognize that social separation produces profound behavioral changes in young animals: agitation with persistent crying, accompanied by a massive release of adrenal stress hormones. These effects strongly suggested the existence of fundamental neural substrates that are designed to forge secure social bonds. From the 1960s to the 1980s insightful psychoanalysts, psychologists, and biologists began to assert that social bonds are created from our profound and innate need for each other. The psychoanalyst John Bowlby (1960, 1980), in his highly influential work in the area of human developmental psychopathology, stressed that poor emotional attachments between infants and parents can give rise to a variety of severe psychological difficulties as children grow up, which may persist and continue to impact the affected individual's well-being across the life span.

The results of animal research have been as robust and unequivocal as the orphanage observations of Rene Spitz. The psychologist Harry Harlow's (1958) well-known research on isolated infant rhesus monkeys, along with

work by the biologist John Paul Scott on lambs and dogs, demonstrated that when young animals are separated from their mothers they cry for hours, even days (Scott & Fuller, 1998). They no longer eat, and isolated monkeys fall into a despair that resembles severe depression. Maternally deprived infant monkeys will seek out any comfort they can find, including soft, inanimate "terry-cloth mothers," in preference to hard, wire mothers that provide nourishment but no solace. When this type of social isolation was sustained for a few months, the monkeys exhibited lifelong problems in social adjustment. And those severe deficits are only partly reversed if young monkeys are reared in the company of same-aged peers rather than their mothers (Suomi, 2006). And these effects were transgenerational. One of the most dreadful lifelong problems was found when emotionally deprived females grew up and became mothers themselves. As a result of their own childhood deprivations, these mothers were unable to respond adequately to their offspring. For example, female rhesus monkeys raised in isolation tended to be timid and overly excitable in relation to their young, and the mothers often neglected or abused their young (Harlow, 1958; Suomi, 2006).

We will try to show how far we have come in understanding the neural nature of GRIEF since Bowlby first published his insights on attachment and development. We no longer see an infant's need for nurturing care simply as a spinoff of the need for physical care. The early observations of Rene Spitz and colleagues (1946) indicated that simply taking care of bodily needs did not prevent the "failure to thrive" syndrome that arose from the lack of loving human contact. We finally understand that there are specific networks in the brain that generate our need for others. These networks are controlled by specific neurochemicals. And imbalances in such chemicals can promote mental distress and, if prolonged, illness. We will also discuss the crucial nature of the first social relationship, the relationship that an infant has with its mother or primary caregiver. We will see that this relationship, for better or for worse, is decisive in determining the way infants' brains develop.

DISTRESS VOCALIZATIONS AND VARIETIES OF INFANT ATTACHMENT

All nature abounds in the general principles of life that are clothed in an enormous diversity of details. We noted in the CARE chapter that mothers of different species bond in different ways. Herbivore mothers bond very quickly with their young after birth because, being born very mature, they can get lost from the beginning of life. Such mothers form rapid attachments to their offspring not only so they can flee predators together and so they may find and retrieve their offspring if they have become lost; mothers also bond selectively in order to provide resources selectively to their own "children." Carnivore mothers, whose offspring are born very immature, without either eyes or ears yet open—also other altricial species such as cats, dogs, and humans—have a much larger (longer) bonding window. These babies cannot easily get lost early in life, so there is ample time for mothers to bond to infants, and infants typically do not begin to form specific bonds to their mothers until they are approaching the age when they can get lost on their own. Thus, human infants show strong bonding to mothers only during the second half of the first year of life. Prior to that, infants seem quite content with anyone consistently taking care of them.

Thus, as already mentioned, scientists must be concerned that some of their favorite "artificial" species, such as lab rats and mice, that have often been bred for research purposes for hundreds of generations, may only have vestigial GRIEF systems (Panksepp et al., 1992; Panksepp, 2003b). This is one reason they were so convenient for behavioral research, which required "unpolluted" animals (especially in the behaviorist era), that survived well when housed all alone in sterile cages. Unlike infant guinea pigs, chickens, and primates, young laboratory rats do not pine away in depressive despair when housed alone. Of course, they are not as socially aloof as many reptiles. They do enjoy social companionship very much, and they get somewhat depressed if housed all alone for long periods. As we will see in the next chapter, they especially crave social interactions such as play, and social isolation strongly enhances playfulness (in strongly bonded species, it just promots huddling and clinging). Thus, if given the option young rats will always choose to be with friendly others. Indeed, social companionship serves as a strong reward in conditioned place preference studies, an effect that is stronger in females than males (Panksepp et al., 1997).

It is important to realize that infant rats and mice do not exhibit *true* separation calls, though they do make some small ultrasonic "clicking"

sounds when left alone outside their nest. These are not *real* separation calls for such pups are too immature to get lost on their own. They may simply reflect physical distress. Before two weeks of age, infant rats can't properly thermoregulate by themselves, and they need to signal mothers that they have been dragged out of their nests and are getting cold. These animals do not have a functioning separation-distress and social bonding system. Indeed, when they are old enough to get lost, at about 15 days of age, their physical distress peeps disappear, but they are not replaced by social separation calls. Although infant lab rats that are subjected to repeated sustained early isolations at a very young age (e.g., especially in the week before their eyes open at ~14 days of life) do show long-term depressive type behavioral and brain changes, these effects may be largely due to general stress (being cold and not licked by a mother) rather than specifically to social-isolation distress (Heim & Nemeroff, 1999). This is a big problem if one uses laboratory rodents to try to model human attachment and depressive processes.

Clearly, there is something peculiar in the social motivation of infant lab rats: They do not seem to require a particular companion—not even their mothers. For them, any other mother rat seems to provide enough comfort to eliminate indices of distress. It is far different in our own species. Once specific social bonds are formed, a human child may cry even when surrounded by many people, as in the supermarket example. Only the child's mom or a familiar caretaker can provide full comfort. Many other species also form these kinds of specific social bonds. But this is not the case for infant rats and mice. All they need is the presence of any other friendly animal to appear emotionally satisfied, at least until feeding time. Clearly, laboratory rats and mice have relatively weak separation-distress responses and there is currently no evidence that they have specific maternal social-attachment systems, as do many other rodents (see Colonnello, et al., 2011). Perhaps their social desires operate largely through the general-purpose SEEKING system that also helps mediate adult social bonds (see Chapter 7). It is becoming clear that adult social-sexual bonds are promoted by brain dopamine-mediated SEEKING behaviors (Insel, 2003). This is not the case for infant-mother bonds, where endogenous opioids and oxytocin are critically important (Nelson & Panksepp, 1998).

THE ANATOMY OF GRIEF

We usually think about misery and happiness in terms of external events: We are miserable because someone has betrayed us and we are happy because friends have lent their caring, even affectionate, support. But the GRIEF system, like all primary-process emotional systems, is initially "objectless"—there is a point early in life where it can be linked easily to any supportive and caring individual. Even an abusive individual is better than no individual at all. And when that individual is not available—when young animals are left completely alone—their distress becomes intense and they will cry for long periods unless rescued by a caretaker. Why and how do these patterns unfold?

The most informative neuroscientific evidence has emerged from the detailed analysis of one basic behavioral measure: the separation calls aroused by social isolation in young animals. We know much about this brain system because we can electrically or pharmacologically arouse specific brain regions, thereby evoking separation distress. Work on several species of animals has revealed the neuroanatomical locations of the emotional system that mediates DVs and the distinct social feelings that arise from social exclusion and loss. In the late 1970s, we identified specific brain regions from which DVs could be aroused with electrical stimulation: especially the periaqueductal gray (PAG) and surrounding midbrain regions, the dorsomedial thalamus, the ventral septal area, the dorsal preoptic area, and sites in the bed nucleus of stria terminalis (Panksepp, Normansell, et al., 1988). In higher species, DVs also can be aroused by stimulation to the anterior cingulate gyrus, along with a few sites scattered in the amygdala and in the hypothalamus. In other words, like the other emotional primes, the GRIEF system consists of a widespread emotional network concentrated largely in ancient medial brain regions below the cortical thinking cap. Brain imaging of sadness in human adults has highlighted very similar brain regions, accompanied by a reduction in brain opioid activity (Zubieta et al., 2003).

There are good reasons to believe that the GRIEF system evolved from primitive brain-stem pain networks. One is the fact that both physical pain and DVs are readily alleviated by opiates. And separation distress circuitry is concentrated in the more ancient medial brain regions, such as the PAG, that convey the deep affective feeling of pain, not the cognitive aspects of

pain that are mediated by higher regions of the brain. For instance, intense GRIEF responses (separation calls) as well as FEAR and RAGE responses are generated by the dorsal parts of the PAG, which is also a brain region that generates much of the affective intensity of physical pain. Thus, the psychic pain of GRIEF may have strong evolutionary linkages to the ancient affective messages of physical pain. That is the way evolution works, by using preexisting solutions for crafting new tools for living.

Clearly, a GRIEF-based social-need system exists in a wide range of vertebrate species, including our own. Relevant brain stimulation research, along with neurochemical analyses implicating endogenous opioids, was first conducted in guinea pigs (Herman & Panksepp, 1981) and then was replicated in an evolutionarily very distant species, the domestic chick (Panksepp et al., 1988). Sufficient work has now been done in other species, particularly primates (Jürgens, 2002), to give us confidence that this circuitry is a universal property of all vertebrates with strong motivations to form lasting social bonds. However, the reliance of animals on this system varies greatly across the life span. Young animals are very dependent on it, but with maturation the responsivity of the system diminishes, partly because of the inhibitory effects of sex steroids, which shift animals toward adult forms of socio-sexual gratifications.

MATURATION OF THE GRIEF SYSTEM

One must wonder what happens to these systems as young animals mature. Many adults, especially males, cry very little. Some men can go through most of their adult lives without crying. What has happened to these distress circuits? Have they atrophied and disappeared? Are they still there but have become remarkably insensitive from disuse or because of other intrinsic neurobiological reasons? Research with adult guinea pigs clearly indicates that the circuits are still there: Targeted electrical stimulation of the right places in the brain can still make adult males cry like babies (Panksepp & Miller, 1996).

With age, however, these circuits become much less responsive than when we were babies. How do we know this? As one artificially activates these systems in guinea pigs by using localized brain stimulation, it takes more and more electrical current to provoke crying as animals mature (Panksepp & Miller, 1996). The sensitivity of this GRIEF system gradually

diminishes as animals go through puberty, and it becomes more insensitive in males than in females. This suggests that the increasing levels of sex hormones during puberty play a critical role. Indeed, when young male and female guinea pigs are castrated (and the gonads and ovaries are removed), the sensitivity of the separation-distress system does not diminish as much as it does in intact animals (Sahley & Panksepp, Unpublished data, 1986). At puberty the males with intact gonads cry less when receiving brain stimulation than the intact females. The conclusion is clear: "Big boys don't cry," and not simply because they have been taught not to. They are less likely to cry because their maturing gonads secrete massive amounts of testosterone during puberty. Is this a key reason why boys typically become pushy and less socially sensitive and empathetic than girls? Perhaps. Recent work also indicates that testosterone given to women tends to rapidly provoke more male-like psychological attitudes (Bos, et al., 2011).

Likewise, adult humans are less likely to weep than children do, but they are still vulnerable to the sadness and misery that results from losing a loved one. In human studies, robust electrical stimulation of brain regions that contain GRIEF circuitry in animals can immediately shift people into a state of depressive despair that lifts rapidly when the stimulation ceases (Bejjani et al., 1999). Stimulation of brain regions such as the anterior cingulate is currently a method for treating depressed people who have received no relief from antidepressant medications (Mayberg, 2009). The success of such treatments may be due to disruption (inhibition) of some of the higher brain mechanisms of GRIEF, but that still needs to be ascertained by future work. Indeed, the various kinds of brain lesions that have been induced therapeutically to help people with treatment-refractory depression to feel better may all have their effects by damaging the brain networks for GRIEF and perhaps by facilitating the activity in brain SEEKING systems that normally promote feelings of enthusiasm and positive vitality in our mental lives (Coenen, et al., 2011; Schoene-Bake et al., 2010).

THE CHEMISTRY OF GRIEF AND SOCIAL BONDING

Arousal of the GRIEF system feels awful, while the effects of medications that can diminish its arousability feel good. We know enough about this system to conclude that artificial manipulation of its neurochemicals can

increase or decrease both distress and social motivation. When particular brain chemicals are at low levels, infants produce DVs and adults feel lonely. On the other hand, when these chemicals are at high levels, babies are happy and adults feel cheerfully self-contained. In everyday life, these contented responses occur when babies receive tender care and when adults are supported by family and friends. Thus, positive social bonding, or the feeling that we have a "secure base," is accompanied by high levels of social attachment chemicals.

Three neuropeptide brain chemicals in particular have been shown to *strongly* reduce GRIEF (while there is an abundance of minor players). The first, and perhaps strongest, of these neuropeptides are endogenous opioids, which, in their pharmacological forms (e.g., morphine and heroin), can be highly addictive. Two others that strongly diminish separation distress, oxytocin and prolactin, we have already encountered as big players in mediating CARE. In short, if brain opioids, oxytocin, or prolactin are elevated in distressed infants, DVs will diminish and the infants will relax and exhibit signs of comfort usually displayed when enjoying the soothing attentions of a mother (Panksepp, 1998a).

Experiments, starting in the mid-1970s, monitored social distress through the frequency of DVs exhibited by young animals (young dogs, guinea pigs, and chickens) that were separated from their normal social environments, usually mothers, for short periods of time (Herman & Panksepp, 1978; Panksepp, Herman et al., 1978; Panksepp, Vilberg et al., 1978). By using separation-distress calls as objective indicators of the underlying emotions critical for social bonding (Panksepp, Herman et al., 1980), it was discovered that all brain chemicals and pharmaceuticals that activated one of the three major opioid receptors, the *mu*-receptor, were incredibly effective in reducing DVs in all three species tested. And these effects were due to shifting sensitivities of brain GRIEF networks (Herman & Panksepp, 1981). These effects were subsequently replicated and extended to rodents and primates (Kalin et al., 1988; Kehoe & Blass, 1986; Keverne et al., 1997; Newman, 1988).

These findings suggested a relationship between opiate addiction and social attachment mechanisms. This may help explain why so many people, especially lonely people—those with too much psychological pain—become addicted to narcotics (Maté, 2008; Panksepp, 1981a). Stimulation of other opiate receptors called *delta*-receptors had similar but weaker

effects. A third opioid, dynorphin, which operates through the *kappa* receptor, generates feelings of a diametrically different kind. The feelings produced are usually very aversive, with disorienting, dissociated feelings that make some feel as if they are losing their minds. It now seems likely that dynorphins become overactive in depressed animals and people (Land et al., 2008; Watt & Panksepp, 2009).

What evolutionary purpose in the brain might the existence of endogenous opioid systems serve, when narcotic addiction, which depends on such systems, is the bane of society? An initial clue is that there are dramatic similarities between the dynamics of opiate addictions and positive social relationships. Initially, drug addiction starts with a period when the drug produces a powerfully euphoric pleasure or emotional relief response that drug users come to treasure and intensely crave as the feeling disappears when such drugs wash out from the body. When the drugs leave the system, there is a dissatisfied, even painful, affective residue (the "opponent process"). This leads to repeated use and in some, eventual drug abuse. Long periods of narcotic use are commonly followed by drug tolerance, during which increasing amounts of the drug are required to produce the now familiar and desired positive feelings. Now the individual is dependent on the drug to sustain any feeling of normality. If, however, the addict is deprived of the drug after tolerance has set in, he or she will endure a period of withdrawal that is attended by dysphoric feelings and misery, a feeling not dissimilar to the sadness you feel when you have lost a friend (for a summary see Fig. 8.2).

Social relationships follow a similar trajectory. There is the initial period of intensely attractive feelings of social bonding followed by a gradual diminution as one gets used to the other person, perhaps akin to opiate tolerance. If, however, the relationship is subsequently threatened or terminated, one endures a period of separation distress. The quality of this distress is akin to maternal separation-induced panic in the very young and to sadness or grief in adults. Thus, social bonds were initially hypothesized to be mediated by pleasurable feelings evoked by endogenous opioids (Panksepp, Herman et al., 1978). It was suggested that abundant brain opioids engender the contented feelings of warmly bonded social relationships. A sudden dearth of opioids might engender the panic and sadness that we feel when we are isolated or bereaved.

This hypothesis finds historical support in the fact that in psychiatry opiates were once used as antidepressants. Indeed, they were the only effective psychiatric medicines available to doctors before the modern era of psycho-pharmacology for emotional disorders that started in the early 1950s. And although their use often led to addiction, there was no question that they made unhappy patients feel better (Tenore, 2008). In addition, the psychological literature on opiate addiction indicated that the emotional effects of opiates are quite similar to the feelings of security that one obtains from supportive social bonds.

Many pharmacological studies have now affirmed the opioid hypothesis of social affect and social bonding—the first empirically supported neurobiological theory of social attachment ever proposed (Panksepp, Herman et al., 1980) and it has even been extended to the wonderful singing and social bonding of birds (Riters, 2011). The idea has also been extended to the pleasure we get from many other sensory affects, for instance, the pleasures of eating (Avena et al., 2008), as well as perhaps the highs that often accompany long-distance running (Strassman et al., 1989). The suspicion is that these diverse effects arise from opioid activity in many different brain regions. However, the finding that chemicals that stimulate mu-opioid receptors dramatically reduce separation distress is full of psychiatric implications. For instance, clinical depression may substantially reflect a pleasure deficit in the brain.

It has been gratifying that recent brain imaging has revealed that human sadness and related social processes are mediated by these same brain regions (Damasio et al., 2000; Lorberbaum et al., 2002; Swain et al., 2007) and that human sadness and depression are accompanied by low levels of brain opioids (Kennedy et al., 2006; Zubieta et al., 2003). It is especially important to note that a scarcity of brain opioids may contribute to several psychiatric disorders. Besides feelings of depression, it is becoming clear that the neurophysiology of panic attacks is promoted by administering receptor blockers that reduce opioid activity in the brain (Preter, et al., 2011). Presumably, depressed people and those who lack adequate social support have low levels of soothing social-affect molecules in their brains, making them more liable to abuse addictive drugs. Positive social activities, including motherhood (Ferris et al., 2005), tend to reduce the likelihood of cocaine and opiate addiction, at least in animal models. This could be due to the natural, but affectively comparable, satisfactions derived from positive

social interactions. We also now know that a variety of positive social interactions, such as play, result in the release of endogenous opioids in the brain, which may have further implications for natural ways to reduce addictive behaviors and other psychiatric problems (see Chapter 10).

The fact that endogenous opioids, so similar to opiate drugs of addiction, mediate social relationships indicates that mammals, especially the very young who are completely dependent on others, are literally addicted to social relationships. By their very nature, mammals and other vertebrates, such as birds, maintain family life through opioid-mediated social dependencies, which can become addictions when people satisfy these systems pharmacologically. Thus, one reason why some people are more likely to become addicted to opiate drugs may be that they are not getting enough positive satisfaction out of their social lives. Many may be self-medicating because they have chronic feelings of "psychic pain" that arise, in part, from chronically active GRIEF systems. They have learned that they can get relief more rapidly through the immediate consumption of molecules rather than through the development of positive social relationships (Insel, 2003; Panksepp, 1981a).

OPIOID ACTIVITY, LEARNING, AND OTHER POSITIVE EXPERIENCES

We now know that a variety of comforting environmental stimuli can unconditionally result in the secretion of the endogenous opioid β-endorphin and other comforting brain chemicals. Bodily warmth, familiar maternal odors, soothing voices, suckling, and even sweet sugar water will cause cries to abate in many young mammals, including humans. For example, after rat pups have suckled they exhibit a stereotypical stretch response and a mild insensitivity to pain (Smotherman & Robinson, 1992). Similar analgesic effects have been observed in human babies (Gray et al., 2002). This indicates that the infusion of milk induces relaxation and comfort, the typical responses to opiates. If one administers an opioid receptor blocker prior to milk infusion, these contented responses are inhibited.

As with other emotions, the influences of this primary-process emotional system are expanded through learning as people and animals mature. Opioid release surely also becomes subject to conditioning and various

learning experiences. For instance, one learns more easily to love people who resemble people one has loved before. Even infant rats show later preferences for smells associated with their mothers (Nelson & Panksepp, 1998). The primal emotional systems are among our most essential value-coding tools, each distinctly valenced through eons of brain evolution. These systems yield evolutionary experiences that guide the construction of all the rest of the mental apparatus—the secondary and tertiary cognitive strategies through which our brains layer adaptation upon epigenetic adaptation.

We may readily imagine examples of how conditioned secondary learning occurs from the earliest stages of life, linking opioid release to a variety of conditioned stimuli. For instance, if a mother regularly plays music while nursing her baby, the sound of the music alone might in time cause the release of opioids in the infant's brain, thereby producing soothing affects. Thus, music, as a conditioned stimulus, may take on durable, affectively driven meaning in the course of the infant's neural development. Of course, certain types of soothing music, being special forms of "touch" (audition is a skin vibrational sense), may have intrinsic abilities to release opioids, and the lower auditory relay stations, such as inferior colliculi, are rich with opiate receptors (Panksepp & Bishop, 1981).

A moment's reflection suggests that the conditioned stimuli, the cognitive strategies that arise in response to them, and the relationships that develop among these attitudes over the course of a lifetime are staggering in their quantity, scope, and complexity. These forces are of great importance for personality development and cognitive maturation. Because of their long-term influences, we often have little choice but to follow the affective dictates that well up from ancient regions of our brains. As in so many aspects of life, emotions lead the way.

As well-bonded children grow up into adulthood, they learn social skills that keep them close to friends and relations. This learning process must surely involve the development of higher order social feelings—from shame and shyness to empathy—that play their roles in these relationships. For example, if a child appears foolish in the eyes of others, she may feel ashamed. If she is unsure of the response of others she likes, she may feel shy, and, of course, a child's desire to feel close to those she loves encourages empathy. Children also develop skills that enable them to cope with inevitable periods of loneliness—perhaps by distracting their attention

or by engaging in gratifying fantasies and games. All these strategies relate to our level of and need for social engagement. And from a neuroscientific point of view, all these strategies reflect reactions to the essential need to maintain affective balance, in some cases the regulation of opioid secretion in our brains (Panksepp, Siviy et al., 1985).

Poorly bonded children often fail to develop these capacities and, as adults, they can easily become depressed or even panicky if they are obliged to fend for themselves. Even when social networks are available, relationships can be compromised by their fragmented and often aggressive qualities. When such negative tendencies emerge in human personality development, various psychological and pharmacological therapies can effectively promote affective homeostasis (Chapter 12).

Our capacity for loving attachments may also take many less obviously social forms, ranging from the love of music, as we have seen, to the squalor of drug addictions. It is even possible that much of the emotional attraction and power of religion is based upon social attachment systems, no doubt supplemented by other social feelings of the brain (Thandeka, 2005, 2009). Consider the fact that the hunched, self-clasping posture of socially isolated infant monkeys is fundamentally similar to a prayer posture—a bodily posture of despair and supplication, head down, arms and hands clenched, expressing the most primal need for solace. In humans, this often transforms into the cognitive longing for CARE from a higher power. Another common liturgical posture, as seen when clerics give sermons, with arms uplifted gracefully toward the heavens, resembles the eager seeking posture of an infant reaching upward toward a caretaker such as a mother.

OPIOIDS AND THE SENSE OF TOUCH

Opioid secretion is especially sensitive to the soothing effects of touch. We all know that we can comfort domestic animals by petting them. One easy way to study such effects objectively is to monitor crying in young animals when they are held or not held. The effects are, of course, dramatic. Animals stop crying rapidly when gently touched. There is compelling evidence that this contact comfort is mediated, in part, by the activation of brain opioid systems. For instance, young chicks agitated by separation from the flock will quickly settle down and even close their eyes in comfort when held by humans (Figure 9.2). Opioid receptor blockers reduce the

effectiveness of this contact, increasing the amount of time needed to calm the chicks. However, even with complete blockage of opioid systems by chemicals like naltrexone or naloxone (which block the effects of opioids), socially isolated birds held gently in this way do eventually settle down and cry much less than control birds that are not held. Clearly, neurochemistries other than opioids contribute to feelings of contact-comfort.

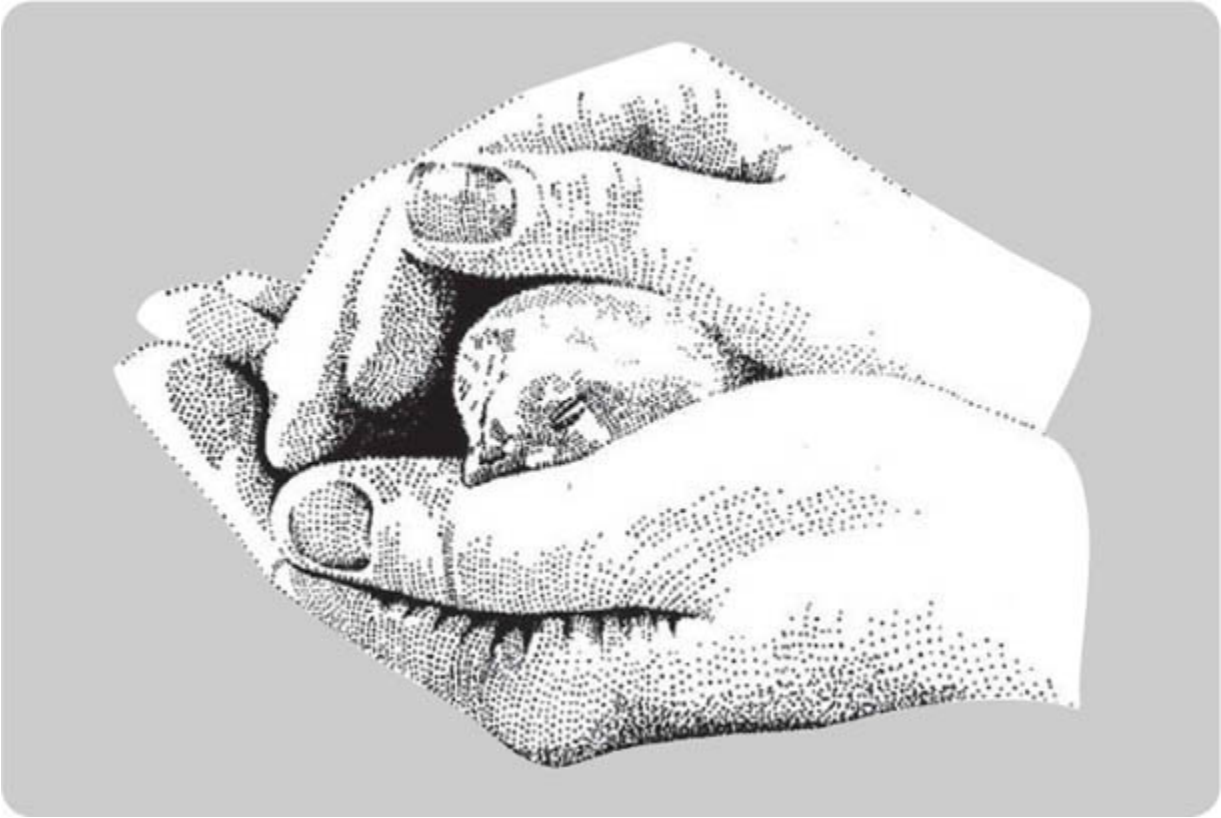

Figure 9.2. When held gently in human hands, newborn chicks exhibit a comfort response consisting of the cessation of vocalizations and eye closure. These effects are attenuated by opiate receptor blockade with naltrexone and amplified by low doses of opioids (drawing by Lonnie Rosenberg; from Panksepp, 1998a; republished with the permission of Oxford University Press).

The fact that touch can release opioids in the brain has also been confirmed in primates (Keverne et al., 1989). Indeed, administration of the opioid receptor blocker naloxone tends to increase grooming in primates,

perhaps in an attempt to counteract the negative affect of reduced opioids in the brain. Primates groom each other in order to obtain feelings of social comfort. In neuroscientific terms, this means that grooming is rewarding, at least in part, because it facilitates the secretion of opioids in the brain. By the same token, the administration of opiate drugs decreases the desire to be touched, perhaps because opiates induce a sense of opioid self-satisfaction in an animal's brain. Indeed, the most dominant monkey in the above study did not wish to be groomed by others, and she had the highest baseline brain opioid levels. She did not need to be groomed, but she was quite willing to groom others.

Two Other Comfort Chemicals That Soothe GRIEF

Soon after the discovery of the role of endogenous opioids in regulating GRIEF, it was discovered that oxytocin and prolactin were comparably robust inhibitors of this system and that they were capable of strengthening social bonds between infants and their mothers. We have noted above that oxytocin and prolactin, in addition to opioids, will quell DVs in infant animals. However, much research still needs to be done. In particular we need to learn more about the mechanisms through which these chemicals influence the human GRIEF system. Although it is now generally accepted that social-emotional processes are regulated by opioids, oxytocin, and prolactin, we do not fully understand how these systems interact.

Rather little is yet known about the role of brain prolactin in affect regulation. Oxytocin, however, has been subjected to extensive investigation (Insel, 2010), and has demonstrated roles in quelling DVs and in generating social attachments. In addition, evidence for the role of oxytocin can be found by examining the brains of animals at various times in the life cycle when they exhibit different proclivities for social engagement. In the brain of an infant rat, when social bonding is crucial to survival, one finds high levels of oxytocin receptors in the brain structures of the GRIEF system. Oxytocin receptors are less dense in these systems during adulthood, when social bonding is not so crucial for survival. When animals are young, oxytocin may play a more decisive role in their largely primary-process social and emotional lives than when they are physically mature and have abundant cognitive backup mechanisms and strategies to sustain emotional homeostasis. Still, it seems to be the case that oxytocin is

released by comforting social touch for a lifetime. For instance, it is known that animals stroked on their stomachs release more oxytocin into their circulation, as do humans who have been massaged (Uvnäs-Moberg, 1998).

One also finds different densities of oxytocin receptors in species with different social temperaments. For example, the brain of the strongly socially bonded prairie vole has a dense distribution of oxytocin receptors more similar to the brain of infant animals. Montane voles, on the other hand, with their tendency to live alone except when sexually motivated, not only have fewer overall oxytocin receptors but these receptors are differently distributed in the brain. Similar differential patterns of oxytocin distributions have been seen in solitary and pair-bonded monkeys, as well as in wild mice (Donaldson & Young, 2008; Ross & Young, 2009).

Although oxytocin research has become a hot item in recent years (Carter, 1998; Insel, 2010; Nelson & Panksepp, 1998; Ross & Young, 2009), how this chemical works to generate affective change is by no means clear. As noted in the previous chapter, oxytocin can enhance the effects of endogenous opioids. Animals usually become habituated to (tolerant of) opioids, which means that, after a while, the drug will lose some of its potency. This is why addicts need an ever-increasing amount of drugs and why the initial euphoria of a new friendship may fade into a more sedate companionship over time. Oxytocin can reduce this kind of opioid habituation, rendering opioids more potent for longer periods of time. Perhaps oxytocin enhances the activity of opioids such as β-endorphin (Kovács et al., 1998). If so, much of the affective comfort—the sense of trust and security—that can be produced by oxytocin may yet turn out to be opioid-mediated.

This relationship between oxytocin and opioid sensitivity may be especially important for new mothers whose brains are awash with oxytocin. In addition to providing maternal moods, oxytocin probably prolongs the effects of opioids, which may be one reason why maternal moods are so intense and persistent. The same principle may be at work in social bonding. Oxytocin may increase the potency of endogenous opioids, allowing them to provide a more intense sense of comfort—especially to youngsters who are still completely dependent on care.

Opioids and Other Social Neurochemicals in Autism

The substantive link between social bonds and endogenous opioids led Panksepp to consider that the asocial symptoms of autism might be caused and/or exacerbated by high levels of endogenous opioids. Indeed, almost half of socially aloof autistic children exhibited prosocial clinical benefits from low doses of naltrexone (Panksepp, Lensing et al., 1991). While excessive endogenous opioids are unlikely to be the main cause for autism, atypical opioids may be one factor in some cases (Bouvard et al., 1995). This work on autism marks one of the rare instances in which basic research on brain emotional systems in animals has generated an effective psychiatric intervention. However, this has not become a standard treatment, because sufficiently extensive double-blind placebo-controlled studies have never been conducted, even though a number of studies have confirmed the initial results (Green & Hollander, 2010; Kolmen et al., 1997).

More recently, oxytocin has loomed large in the autism and child development literature (Bartz & Hollander, 2008; Carter, 2007; Insel, 2010; Yamasue et al., 2009). Studies have observed lower than normal plasma levels of oxytocin in autistic children as well as some modest social benefits from intranasal administration of the neuropeptide (Green & Hollander, 2010; Heinrichs et al., 2009; Rossignol, 2009). However, as is common in such areas of research, failures to replicate and extend these findings are bound to occur (Tansey et al., 2010). Autism is a complex conglomeration of several brain-body problems, with many genetic contributions, and there is currently no way to reliably distinguish the many subtypes. The current epidemic of autism, where about 1 of every 120 children could be diagnosed with an autism spectrum disorder, suggests that something is happening in our postmodern social or physical environment that promotes a failure of children to thrive socially. As we emphasize in the next chapter, one big problem may be that we no longer allow children to be children—to have robust physical play be part of their daily social diet.

STRESS CHEMICALS THAT AROUSE THE GRIEF SYSTEM

We have seen that a dearth of endogenous opioids and oxytocin may result in feelings of loneliness and even panic in the very young. When GRIEF is aroused, other brain chemicals become more active, especially the stress

neuropeptides, corticotrophin-releasing factor (CRF) and glutamate, an excitatory neurotransmitter that participates in every emotional response. Indeed, when these chemicals are administered to the brains of animals, they very strongly promote separation calls (Panksepp, 1998a; Panksepp & Bekkedal, 1997). CRF produces normal calls (see Figure 9.3), while the vocalizations after glutamate activation are often acoustically abnormal (Normansell & Panksepp, 2011). CRF is the hormone involved in the classic stress response, which activates the pituitary-adrenal system. Stress activates neurons in the paraventricular nucleus (PVN) of the hypothalamus, which contains an abundance of CRF neurons.

These PVN axonal projections descend to the anterior pituitary gland, causing the release of adrenocorticotrophic hormone (ACTH) into the bloodstream. ACTH is the trigger that stimulates the adrenal cortex, which lies just over the kidneys, to release the hormone cortisol. This steroid helps the body to use energy in ways that will allow it to cope with many kinds of stressful situations, including separation distress. It is not known exactly how cortisol might help animals to cope with separation distress, but one possible option lies in the fact that the hippocampus has many cortisol receptors. The hippocampus is responsible for the creation of episodic memories, particularly memories about spatial relationships and personal autobiographical experiences. Perhaps as a result of periods of separation distress, the elevated cortisol secretion allows young animals to develop stronger memories of old familiar and comforting places, such as their homes, as well as their love of parents when they are reunited. A little separation anxiety may help solidify social memories and social bonds. Another related hypothesis is that cortisol facilitates cognitions that can facilitate finding home and the benefits of social reunion.

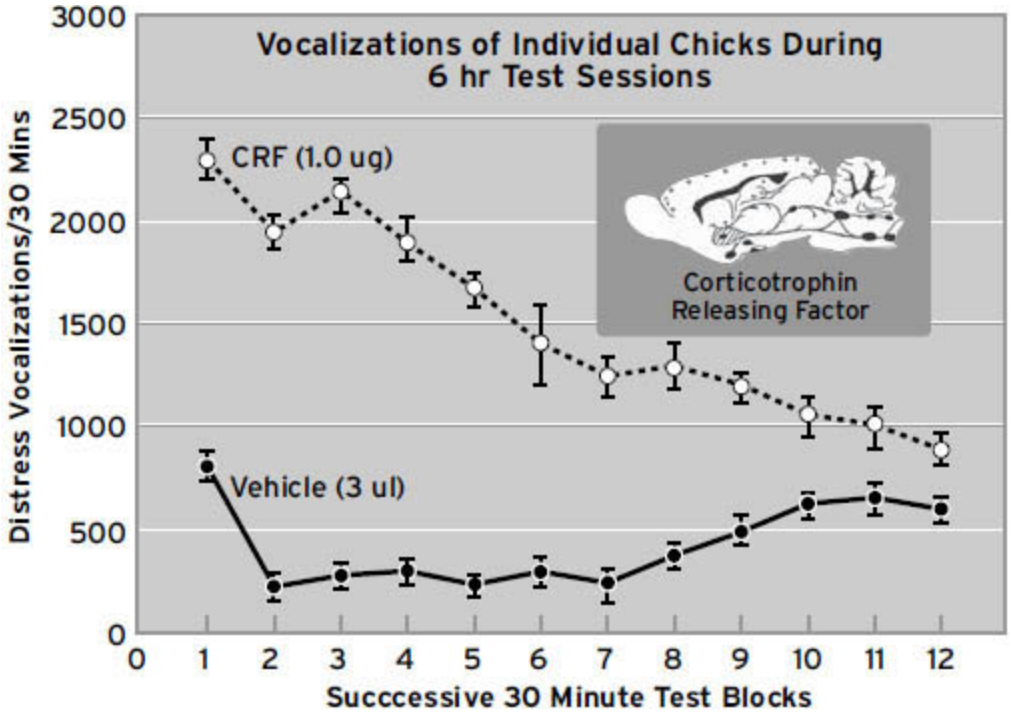

Figure 9.3. Corticotrophin-releasing factor (CRF) is a major brain activator of the pituitary adrenal stress response, but it also has an extensive subcortical circuitry, as depicted in the insert. When a small amount of CRF is injected into the cerebrospinal fluid of the young 3-week-old chicken, it no longer shows many separation distress vocalizations (peeps) when separated from the flock for 6 hrs. (Unpublished data, Panksepp, 1984.)

When the stress system is working well, cortisol is fed back to the abundant receptors in the PVN, and this feedback action causes the PVN to stop pumping out CRF to the anterior pituitary, which stops making ACTH. Without ACTH to facilitate the release of cortisol, the entire stress response winds down. If this self-regulatory feedback effect is impaired, however, the production of cortisol continues unabated and eventually exerts a deleterious effect on the body and on the brain, creating chronic feelings of stress (sometimes manic excitement). In susceptible individuals, depression and even hippocampal damage eventually result; such negative effects can sometimes be reversed with antidepressants.

In extreme cases, prolonged high levels of cortisol released into the circulation cause the hippocampus to become overstressed to the point of being permanently impaired. Excess cortisol can eventually injure and even

kill neurons in the hippocampus, resulting in memory loss. Because of ethical considerations, detailed neuroscientific evidence is lacking for human beings who could be studied in this way, but those who have experienced war trauma or other atrocities (such as Holocaust survivors) often exhibit some hippocampal shrinkage with structural brain imaging, although evidence of actual damage has been hard to demonstrate. However, controlled research shows that monkeys that have been raised without nurturing relationships can experience prolonged high levels of cortisol that do injure the hippocampus (Nelson & Bloom, 1997). Indeed, it has been found that abused children as well as adults who have chronically suffered sexual abuse or soldiers who have experienced excessive wartime stress also tend to have smaller-sized hippocampal areas than nonstressed individuals have (Conrad, 2008; Irle et al., 2009). Because the hippocampus is essential for the creation of many types of memories, including all our episodic (autobiographical) memories, these studies indicate that prolonged social deprivation in the early years can compromise the developmental maturation of the mind. In this way, we can imagine how early-life stressors, as well as those later in life, may impair cognitive functions that are mediated by the hippocampus.

STRESS AND DEPRESSION

Imbalances in the pituitary adrenal CRF-ACTH-cortisol system are also followed by a depletion of chemicals known collectively as biogenic amines, especially norepinephrine (NE), serotonin (i.e., 5-HT or 5-hydoxytryptamine), and dopamine (DA). Initially, the release of CRF powerfully arouses these neural systems. But when CRF release is sustained, the synaptic chemical resources of these systems can become exhausted. This leads to many secondary changes in brain biochemical systems, including reduced levels of neural growth factors and increases in inflammatory processes in the brain (Cirulli et al., 2009; Harro & Oreland, 2001; Miller et al., 2009). When the brain's biogenic amines are depleted, people and animals are also prone to depression, which can follow on the heels of sustained excess CRF release. Depressive symptoms in animals and people can be induced experimentally by establishing such patterns of physiological change in the body. Indeed, prolonged administration of CRF along with the depletion of the biogenic amines robustly promote

depressive responses in animals, while administration of CRF receptor blockers counteracts depression in humans (Holsboer & Ising, 2008).

We will now provide a general synopsis of the relationship of the above ideas to the understanding and treatment of depression. We will do this largely without citations, since a coverage of stress-related instigation of depression as well as traditional pharmacotherapies of depression have been very widely covered in the available literature already cited as well as in various psychiatric textbooks (e.g., Panksepp, 2004). This synopsis simply provides a bridge to discussing the role of the GRIEF system in depression and some potential novel interventions such as attempts to directly replenish positive affective chemistries, while the influence of negative affective chemistries is diminished.

We do not yet know precisely how converging, stress-related brain changes ultimately lead to the persistent psychological changes that characterize clinical depression. But we do know that medications that counteract the low availability of biogenic amines (i.e., serotonin, norepinephrine, and dopamine reuptake inhibitors) tend to have antidepressant effects. Most of the antidepressant medications in current use facilitate the availability of biogenic amines by blocking their reuptake mechanisms. The antidepressants thus allow the biogenic amines to linger for longer periods at the synapses, which are the gaps between neurons where these chemical messengers are released and received. The most widely used antidepressants are selective serotonin reuptake inhibitors (SSRIs), such as Prozac. As their name implies, the SSRIs have selective effects that make serotonin alone more available at synapses. However, these agents, used for prolonged periods of time, also downregulate serotonin receptors, which may set in motion brain adjustments which may eventually have undesirable consequences when chronic medications are stopped.

Other types of reuptake inhibitors that influence the availability of all three of these crucial amines are most effective, at least in the short-term, while efficacy is less among those that act as single amine facilitators. The fact that these medications take weeks to show efficacy suggests that the therapeutic benefits are secondary to long-term brain changes such as the gradual buildup of enhanced neural growth factor activities, which should help repair stress-damaged brain areas like the hippocampus. However, it should be noted that short-term use of SSRIs can markedly alleviate

separation distress in animals, which has led to the use of agents such as fluoxetine (trade name Prozac) for the treatment of separation-distress problems in pets.

Still other antidepressant medications, such as the monoamine oxidase (MAO) inhibitors, increase availability of the biogenic amines by inhibiting the effects of the enzymes that degrade these neurotransmitters at the synapses. The MAO inhibitors and other older, pre-SSRI antidepressants are no longer as widely used in psychiatry as they once were, due to their higher levels of side effects compared to newer medications. Regardless of their specific mechanisms, however, the effect of both the older and newer antidepressants is to make biogenic amines available for longer periods of time in the synapses, thus providing increased stimulation to the neurons where these chemical messengers are received. Again, the sustained long-term consequences of brain adjustments arising from such medications remain to be adequately studied.

A bit of elaboration on the ways in which such drugs give rise to antidepressant effects is in order. As noted earlier, biogenic amines (neurochemicals such as norepinephrine, serotonin, and dopamine) often act as neurotransmitters, or chemicals that cause neurons to fire or to be inhibited from firing. Soon after a neurotransmitter is released into a synapse, it is eliminated from the synapse in one of two ways. First, an enzyme can degrade the neurotransmitter, breaking the molecules into inactive components. Second, the neurotransmitter can be taken back up into the neuron from which it was released (“reuptake”), thus taking the molecules out of the action. When biogenic amines act as neurotransmitters, their availability at the synapses can be facilitated by either of these mechanisms—that is, either by inhibiting their chemical inactivation or by inhibiting their return to their neuron of origin. However, as already noted, the strong antidepressant effects of these drugs may take weeks to occur, a delay that implicates the many “downstream” changes being activated in the brain. These more remote changes may be most beneficial, including many repair processes such as reduced inflammation in brain tissues, as well as both the proliferation of new neurons and increased growth of neurons in the hippocampus, a region essential to normal and healthy brain functioning. Others, such as the development of receptor under-sensitivity may not be as desirable in the long term.

Although we have no definitive answer to the causes of depression at the level of brain function, there are numerous neurochemical candidates. These potential causative agents are, unfortunately, often proposed without any supporting affective data. One of the currently favored chemicals being vetted is a brain "fertilizer" known as brain-derived neurotrophic factor (BDNF), but there are many other growth factor candidates. These are all "transcription factors," which means they exercise influence at the juncture of gene regulation where many downstream genetic pathways are turned on. A remarkable finding is that many of these growth factors are also turned on by healthful activities that have antidepressant effects, such as exercise. Even physical play in young animals can promote BDNF availability in the brain (Gordon et al., 2003), and can increase levels of other growth factors such as insulin-like growth factor (Burgdorf et al., 2010). We will emphasize these and other functions of play in the next chapter. However, it is clear that perceived social support is one of the best indications that an individual will be able to bounce back from a major depressive illness (Leskelä et al., 2006).

BRAIN OPIOIDS AND DEPRESSION

Current antidepressant medications do help reverse some of the ravages of stress. We know, however, that when GRIEF is aroused, which is a key factor in stress-induced depression, there is a depletion of comfort chemicals such as endogenous opioids (Kennedy et al., 2006; Zubieta et al., 2003). We also know from a wide array of research that endogenous opioids and opiates can have rapid antidepressant-like effects. These facts suggest that there might be another approach to combating depression, namely by augmenting brain opioid activity. As already noted, in the days before modern antidepressants, enhanced opioid system activation was often achieved by administering opiate drugs. Due to their notorious addictive qualities, however, the administration of opiates was abandoned in favor of the kinds of comparatively weak modern antidepressants that have been designed to intervene in the underactivity of biogenic amines (although, as noted earlier, this does not mean that depression is largely due to a deficit of these transmitters).

Today neuroscientists should reconsider the possibility that depression is largely due to deficits of pleasure chemicals in the brain, particularly those

that support the security of social bonds. For instance, if periods of intense and sustained grief promote depression, in part, by precipitous drops in brain mu-opioid activity, we finally have ways to address such imbalances. Modern neuroscience has discovered drugs that can promote mu-opioid activity without severe dangers of addiction. One example is the mixed opiate receptor agonist/antagonist buprenorphine (which is now widely used to *treat* narcotic addiction). This medication only promotes opiate activity at remarkably low doses, whereas at high doses it blocks receptor activity. At those low doses buprenorphine has been found to be a strong antidepressant in people who have failed multiple other drug therapies (Bodkin et al., 1995). The mixed agonist/antagonist action means that people cannot become strongly addicted to this drug so it can be safely used as a medication for recovering opiate addicts. The drug affords sufficient opiate activity, and hence psychological relief, to prevent the painful withdrawal effects of opiate detoxification. Thus, buprenorphine could perhaps also be more widely used as an effective, fast-acting antidepressant, especially among individuals that have had no relief from traditional therapies. However, without proper double-blind studies and ensuing governmental regulatory approval, its widespread use in standard psychiatric practice is unlikely. Clearly a few well-controlled studies are needed before any general medical acceptance of such approaches can emerge.

In this context, it is notworthy that placebo effects are very common in anti-depressant trials, often being so strong that the benefits are as strong as those obtained with widely used SSRIs. This partly reflects the fact that everyday mild depressive responses are often self-limiting conditions. In addition, from the present social-brain analysis, anti-depressant benefits are to be expected from placebos, which operate partly through endogenous opioid release (for summaries, see Panksepp, 2006c, 2011a). Positive social interactions release brain opioids, providing positive social feelings, and placebo effects may reflect, in part, the perception that mental health professionals and other significant others are caring about one's depressive feelings. This perception of care may increase the release of brain opioids, which makes depressed people feel better. In other words, placebo effects in depression reflect, at least in part, the capacity of social support to activate brain opioid systems. Surely, this is also one reason why the affective

qualities of relationships are so important in the outcome of psychotherapeutic interventions.

Mu-opioid depletion, however, is not the only depression-related opioid imbalance that neuroscientists are exploring. The shift from intense grief to despair and depression is also accompanied by the diminished arousal of the SEEKING system. Elevated kappa opioid activity, caused by the augmenting effects of dynorphin, has been identified as a chemical change underlying this involvement of the SEEKING system. As mentioned above, dynorphin binds with kappa opioid receptors. There is currently quite a bit of excitement that the discovery of medically safe antagonists that can block kappa opioid receptors would inhibit the effects of dynorphin and prove very useful as antidepressants (Knoll & Carlezon, 2010). In fact, this also is one of the pharmacological properties of buprenorphine, which at the same doses can block dysphoric kappa-receptors while facilitating positive hedonics via mu-receptors.

The role of diminished SEEKING arousal may highlight a poorly understood "adaptive" effect of depression. Evolutionary psychiatrists have considered the possibility that even though severe depression can have various deleterious effects on the psychic economy, some level of depression when social support is lost may promote survival (an idea first introduced by John Bowlby and neuroscientifically developed by Watt & Panksepp, 2009). For instance, after a period of intense separation distress with vocal protest, indicative of an initial panic response, which helps parents find their lost offspring, it may be adaptive to regress into a behaviorally inhibited despair or despondency phase in order to conserve bodily resources. Such a depressive state might serve to discourage helpless organisms from wandering even farther from safety. Silence would also minimize detection by predators. In a parallel vein, another negative emotional process that is very effective in reducing distress vocalizations is FEAR. In sum, if the initial protest does not achieve a reunion, a silent despair response could be a useful secondary strategy to optimize the likelihood that parents would eventually find their lost offspring alive.

THE DIFFERENTIATION OF GRIEF AND FEAR

Modern neuroscience now allows us to distinguish between anxieties aroused by GRIEF and FEAR—between isolation panic and the fear one

feels when anticipating injury, death, or some other impending aversive event. Of course, the two systems interact. For example, children who have frequently been left alone will experience separation anxiety, but they will also be afraid of the prospect of being left alone and again feeling miserable. In other words, they can be afraid of GRIEF at a higher cognitive level.

These two systems also share some overlapping neuroanatomies and chemistries. In fact, anatomical and chemical overlap exists in many affective systems (e.g., all emotional systems involve the evolutionarily ancient PAG of the midbrain, the very heart of primary-process emotional life, while the neurotransmitters GABA, norepinephrine, serotonin, and perhaps dopamine as well modulate all the emotional systems). Nevertheless, the FEAR and GRIEF systems can be distinguished on both anatomical and chemical grounds. CRF can activate both systems, but the two systems are also chemically different in many other respects. For instance, at very low doses opiates strongly reduce separation cries. But it takes much higher doses to modestly reduce the behavioral indicators of anxiety or anticipatory fears. On the other hand, the classic benzodiazepine antianxiety agents, such as chlordiazepoxide (Librium) and diazepam (Valium), effectively diminish FEAR responses at low doses, but they are not as effective in diminishing the separation calls that are indicative of GRIEF. In terms of behavior, we just noted that separation distress may lead to learned anxieties: One can easily become fearful of feeling panicky. A parallel causal pathway leading from fear to despair is not readily apparent, because whenever animals are intensely afraid, distress calls typically diminish. Of course this makes good evolutionary sense because animals are afraid when they are in danger, and if they cry out at these moments of peril they are more likely to attract the attention of predators. Still, at a higher cognitive (tertiary-process) level, it would not surprise anyone if chronic anxiety also makes humans and animals more prone to depression.

In any event, there are many reasons to believe that the mysterious pangs of insecurity that psychiatrists call “panic attacks” may also arise substantially from the sudden arousal of the social separation-distress GRIEF network rather than from the FEAR network, as many theoreticians currently believe. The pharmacological distinction between neurochemistries that control panic attacks and those that control general anxiety was first revealed by the careful work of the psychiatrist Donald

Klein in the early 1960s (see Panksepp, 1998a for details). Klein found that the newly discovered benzodiazepine-type antianxiety (anti-FEAR) agents such as Librium and Valium had little beneficial effect on the incidence of panic attacks. The tricyclic antidepressant imipramine, however, was very effective in quelling such attacks. Although patients with anxiety initially claimed that the tricyclic had no benefits for them, nurses reported that these patients were complaining less frequently about panic attacks. Indeed, when the count was in, the patients did have many fewer panic attacks when medicated with imipramine. Apparently, the patients had not focused on those improvements because the drug did not diminish the anticipatory anxiety associated with the disorder—namely, the fear of the attacks themselves. These patients were still afraid of having panic attacks; perhaps they needed medication for FEAR as well.

In this context, it is important to note that imipramine is quite effective in reducing separation distress in many species, including dogs and primates (see Panksepp, 1998a). This indicates that panic attacks, like separation calls, are functions of aroused GRIEF, rather than of FEAR. Bolstering this hypothesis is evidence showing that physiological aspects of panic attacks can be promoted by diminished opioid activity (Preter & Klein, 2008). There is further clinical evidence suggesting that panic attacks are related to social loss: Those who suffer from panic attacks often have a history of childhood separation anxiety. Furthermore, panic attacks and separation distress both make one feel as if the center of one's comfort or stability has been abruptly removed. And both are accompanied by feelings of weakness and shortness of breath, often accompanied by a choked-up feeling as if one had a lump in one's throat.

PSYCHOPATHOLOGY AND THE GRIEF SYSTEM

Imbalances in the GRIEF system play a pivotal role in a wide array of emotional disorders because so much mental illness is rooted in the incapacity to enjoy the security of warm interpersonal attachments. We have already mentioned conditions as disparate as panic attacks, depression, and autism. Perhaps an array of social phobias and personality disorders will also be included under the umbrella of GRIEF pathology. We cannot begin to examine all these conditions. Instead we will focus on the very first and most crucial of social bonds—the infant/mother relationship. The

psychiatric consequence of child emotional neglect, and even moreso abuse, are enormous (Heim, et al., 2010).

During the past several decades, developmental psychologists have constructed a coherent theoretical view of the nature of a child's social attachment to its mother or primary caretaker. They have observed that children exhibit a variety of attachment "styles," or temperaments, that have strong environmental antecedents. Securely attached children are confident of receiving social support from their parents or caretakers. They are generally outgoing and tend to confront life with optimism and enthusiasm. These children grow up to be well-adjusted adults because they start out with a "secure base." Generally speaking, they have good relationships and they are successful in their life pursuits.

However, when a mother fails to nurture her child, the child grows up to be poorly attached. Insecure children exhibit two major emotional and behavioral patterns. Some are excessively clingy and seem to need more than the usual amount of attention from their caretakers. Others choose to distance themselves, avoiding social situations, presumably because they are not confident of receiving the positive support and feedback they crave (Ainsworth, 1982). Over the past 30 to 40 years, abundant clinical research on human infants has highlighted the need for a mother to provide empathetic attention to her child's moods (Beebe & Lachmann, 1988). When mothers are emotionally attuned to their infants, emotional health is promoted.

Of course, these theories rely on behavioral observations and descriptive psychological inferences about what goes on in children's minds. Only in recent years have neuroscientists been able to translate attachment theory into concrete changes that happen in the brain. Neuroscientists understand these brain changes in terms of *epigenesis*—gene expression that takes place as the result of experience. We noted earlier that gene expression is a process whereby a dormant gene becomes active. Epigenesis involves experience-dependent gene expression; it is the gene expression that happens after birth as a result of the child's experiences in the world.

Epigenesis may seem an odd concept because we are accustomed to thinking that the genetic endowment we are born with will precisely and indelibly determine the attributes we will exhibit across our life spans. Some genes do just that. For example, the capacity to exhibit DVs are surely determined by brain networks that become active soon after birth,

and this is why virtually all young mammals and birds cry if they are left alone. Thus, the information for the construction of such brain systems is surely genetically determined. However, certain experiences during the lifetime of a human being or an animal can cause genes to be expressed more vigorously or less so, by epigenetic mechanisms that have revolutionized our understanding of nature and nurture (Szyf et al., 2008). So, when a previously dormant gene is expressed in particular brain circuits, it can produce proteins and neuropeptides that the brain cells have not previously produced. Many of these aroused neurochemical pathways surely modify affective BrainMind functions. Thus, we see how epigenetic processes can influence emotional behaviors and feelings.

When young animals receive the care of their mothers, these experiences result in epigenetic changes in the activity of genes that influence brain function. Such variable brain functions can generate characteristics and behaviors specific to the individual—such as the styles of attachment mentioned above. Epigenesis can create healthy brains in infants who have been well mothered, but it can also engender various types of mental problems. Research suggests that if a mother is depressed and consequently unresponsive to her infant, one sees abnormalities in the child's behavior as well as its brain organization (Meaney, 2001; Tronick, 2007). Perhaps such children develop unresponsive brain oxytocin or opioid systems. As noted earlier, perhaps the failure of oxytocin to soothe a distressed mother, and hence provide comfort for her youngster, may arise because her oxytocin system can no longer complement and enhance the effects of endogenous opioids. There are many possibilities to consider.

One well-known social problem is the poor development of maternal urges in adult females who had poor care and insecure attachments when they were young. In part, the experience of being badly mothered probably creates epigenetic changes in the brains of young females, rendering them unlikely to be the best mothers when they have their own children. One result of poor early mothering is a large number of behavioral changes that arise, in part, from epigenetic changes in the way brains respond to stress (Meaney, 2001; Szyf et al., 2008). Most of the work on this topic has been done in rats, but recently it was reported that the brains of maltreated children who eventually committed suicide as adults exhibited epigenetic changes quite similar to those observed in rats that received less maternal care (McGowan et al., 2009). Our earlier discussion regarding cortisol's

deleterious effects on the hippocampus probably reflects another example of epigenesis. This research helps us better understand why mistreated youngsters often become poorly performing parents who perpetuate cross-generational cycles of child neglect and even abuse. Insecure attachments, perhaps due to low opioid responsivity, may be passed on through the generations. As a population, people who commit suicide have brain indices of low opioid activity (Gross-Isseroff et al., 1998), and it is possible that pharmacological boosts of brain opioid satisfaction, as with low doses of "safe" opioids such as buprenorphine, can reduce suicidal thoughts. Of course, this could be also obtained by the more consistent positive emotional regard of caring others.

Most biological studies on attachment highlight changes to the most recently evolved higher brain regions: the neocortex, especially the functions of the two cerebral hemispheres. These studies, extensively summarized by the clinician Alan Schore (also, see McGilchrist, 2009), point to the fact that many parts of the brain are not fully formed at the time of birth, and the development of these areas is mediated by experience-dependent epigenetic changes. Along with an ever-growing number of neuroscientists, Schore has investigated how the quality of mother-child interactions controls the development of the brain, for better or worse (Schore, 2001).

A focus on the higher brain regions has indicated that, at birth, only the primary somatosensory cortex is metabolically highly active (Chugani, 1996). The rest of the neocortex is still in the process of development. Schore has focused attention on the right cerebral hemisphere, which exhibits a more vigorous growth spurt in the first 18 months of life than the left hemisphere. The right hemisphere remains dominant for the first 3 years of life (Chiron et al., 1997). Obviously, this is the time when infants and young children start to form relationships with their parents. The right hemisphere sustains a more emotional, wholistic attitude toward life, as compared to the later maturing left hemisphere, which eventually provides more analytic cognitive skills, far removed from social sensitivities. Schore focuses on the right side of the brain because research indicates that this side is especially emotionally responsive to external stimuli like nurturing tactile experiences early in childhood (Kalogeras et al., 1996). These experiences are intensely molded by the quality of maternal nurturing, and at least in rats have permanent brain effects (Meaney, 2001). The

development of a child's brain is experience-dependent and is directly impacted by inputs from the mother-child relationship.

Schore further argues that the orbitofrontal cortex (OFC), an old cortical structure, undergoes a critical period of maturity from the last quarter of the first year to the middle of the second year. Again, this is a period when the mother-child relationship burgeons. Experiences with the mother in this time frame generate epigenetic changes that contribute to development or underdevelopment of the orbitofrontal cortex, which plays a critical role in processing interpersonal signals and their emotional significance. When this region of the brain is damaged, people are likely to exhibit poor social regulation, moving toward a more emotionally impulsive, even sociopathic, pole of temperament (Adolphs et al., 2003). A well-developed orbitofrontal cortex also regulates many aspects of the autonomic nervous system, which produces physiological components of emotional experience (Porges, 2009b). Thus, the orbitofrontal cortex plays a critical role in affect regulation (Schore, 1994). If poor mother-child interactions interfere with the maturation of this brain area, the child likely will experience difficulties in regulating affects throughout life. Because affect regulation is a cardinal feature of mental health, good functioning of the orbitofrontal cortex is essential. Although we presently do not understand the details of the epigenetic changes involved in these crucial aspects of brain maturation, we can assume that such changes provide critical mechanisms that result in healthy or unhealthy development of the brain.

Although the specter of compromised functioning of the neocortex, the orbitofrontal cortex, and the hippocampus is ominous, animal research suggests an even more dire scenario. Experimental evidence indicates that human beings or animals subjected to extreme experiences can develop chronically increased or decreased sensitivity of primary-process limbic emotional networks. These changes to subcortical emotional systems are also epigenetically mediated. For example, if an animal has had many frightening experiences, its FEAR system may be permanently sensitized; such animals are likely to be frightened quite easily (LeDoux, 2002). Such epigenetic changes may lead to a pathological oversensitivity and overresponsivity of various other emotional systems, especially those that regulate separation-distress/GRIEF responses.

If we consider the possibility that subcortical emotional systems exert a decisive influence on the maturing neocortex, then long-lasting

developmental changes in deeper emotional regions of the brain assume even greater importance. It has been proposed that early attachment difficulties can result in the attenuation of neural connections between limbic emotional regions and the neocortex (Schore, 1994). This may mean that subcortical regions of the brain make fewer contributions to the development of the cortex. Conversely, because the neocortex usually inhibits limbic expression, this may also help explain why people who have endured attachment difficulties are often emotionally disinhibited.

GRIEF may be the most powerful affective network of the human brain, one from which we can never be fully shielded by the many safety nets of modern cultural institutions. Indeed, the many artistically rich and productive careers of practitioners of cultural forms such as Blues music are testament to the power of this essential human experiential trait. And in the best brain-imaging work, sadness "lights up" our brains more spectacularly than any other emotion (Damasio et al., 2000). The patterns we see in these images match up well with what we know about the neuroanatomy of the separation-distress/GRIEF system through the study of our fellow animals (Figure 9.1). When we have secure attachments to loving others, we are granted a lifelong gift. When attachment processes are impaired, the diverse manifestations of psychic pain within the higher mental apparatus can lead to chronic feelings of distress throughout life. This distress often encumbers the way in which we can relate to others. Still, despite such vicissitudes, humans are a remarkably resilient species, so all of the above influences on emotional well-being are highly variable across individuals.

THE GRIEF SYSTEM AND PSYCHOTHERAPEUTIC TECHNIQUES

Psychotherapists view transference as crucial to their work. Transference refers to the tendency of patients to recreate their established ways of relating to important others within the therapeutic setting. The opinions and feelings that patients have about the therapist will often reflect the feelings and opinions that patients have had about their family members, and particularly about their parents (Pulver, 1995). To put the matter somewhat differently, all patients, indeed all people, develop habitual emotional ways of responding to others, and these emotional habits are largely shaped by early relationships. If you have been brought up in a wholesome

environment, you will approach the world at large in an open and receptive way. This is a transference response, because you may be open even to unscrupulous people. In fact, the first time that you encounter manipulative people you might have difficulty understanding them. Conversely, children who were raised in treacherous environments will often view everyone with suspicion and hostility, and they may not understand or believe that anyone has benign motives. Transference is a universal phenomenon; we often identify positively with other people we admire, and it is useful, probably essential, in psychotherapy because it gives the patient and therapist the opportunity to work through the emotional and behavioral vestiges of troublesome past experiences.

In the early days of psychoanalysis, therapists were urged to conceal aspects of their personalities. The idea was that the therapist's personality should be a "tabula rasa," or a blank slate onto which the patient could project his or her transference reactions without fear of contamination by the therapist's character. There is still some virtue in this model in that therapists should not burden patients with their own problems. After all, one must bear in mind that one of the two is a client, often a fee-paying client.

However, the notion that therapists should be emotionally neutral is not only impossible but also deleterious to the therapeutic endeavor. It is impossible to be neutral because all people (all mammals) have an inherent need for positive relationships. So a therapist who is unsmiling and/or unemotional will be perceived in a negative light. This kind of therapeutic stance will repel most patients and attract only those who have developed masochistic tendencies or who are anxious to "graduate" from psychotherapy training without creating a fuss. It is normal and necessary for therapists to have a positive attitude about their patients and to behave accordingly. Of course, there is always a danger that a positive disposition might blind a therapist to negative traits in a patient's personality. However, when therapy is successful, these negative traits should be addressed without rupturing the overall positive therapeutic relationship. And this positive alliance can provide the patient with a safe haven in which to recognize that the baggage he or she transfers onto others is in fact a collection of habits, beliefs, and feelings that belong to an altogether different relationship, or likely even a different era.

Throughout this book we have stressed the hope that, in the future, psychotherapy and psychopharmacology can work hand in hand. Indeed,

some emerging agents are already available to facilitate standard psychotherapeutic approaches—for instance, D-cycloserine, which promotes therapeutic change on the neural level when administered during exposure therapy (Norberg et al., 2008). One can imagine that short-term pharmacologically induced affective homeostasis, achieved by modest doses of safe opioids that promote positive affect and confidence, might facilitate the therapeutic process, especially if used in the context of reconsolidating troublesome memories with more positive affective contexts (see Chapter 6). Likewise, much more research is needed to see if rapidly acting affective agents such as buprenorphine could be used to promote long-term homeostasis in GRIEF disorders. Of course, these pharmacological aids to psychotherapy could only produce beneficial results if the therapy itself were sound. However, these kinds of medications might provide motivation in the short run that would allow some patients to get over an emotional "hump" that psychotherapy alone would have difficulty in surmounting.

It is especially urgent to have properly designed studies that attempt to promote consolidation of benefits when a client has begun to gain insight into his or her problems. It is possible that a mild dose of a GRIEF inhibitor such as intranasal oxytocin (a strictly experimental agent at this time) or very low doses of imipramine, and perhaps even the mild but rapid antistress psychic effects of many other common antidepressants or antianxiety agents, judiciously combined in temporally advantageous ways, may be useful in promoting lasting therapeutic change. It is also possible that the reconsolidation of memories into a positive self-image can be facilitated by the creative use of sensitively combined psychopharmacologic and psychotherapeutic maneuvers. And this might also steer us away from the current standard practice of "throwing drugs at clients" for prolonged periods of time, without dealing with their mental lives. These are critically important lines of future inquiry, with some promising precedents: For instance, as has been repeatedly shown, the combined use of antidepressants and psychotherapy is more effective than either intervention alone (e.g., Holtzheimer & Nemeroff, 2006).

SUMMARY

The GRIEF system is of paramount importance to mental health and is probably one of the most important systems in generating both the misery of depression and certain types of chronic anxiety. The other is SEEKING (Panksepp & Watt, 2011). All mammals need to bond with others and the quintessential mammalian bond of love is between a mother and infant. It is in this relationship that the mother's CARE system nurtures and provides sensitive responses to the infant's GRIEF system—to the infant's need to feel close and attached. This is not simply the homeostatic need to be fed and sheltered. It is an emotional need to be securely and warmly attached to the mother or caregiver.

Grief teaches us a great deal about love. We may feel sadness or alarm when we merely imagine the prospect of separation from those closest to us. This feeling tells us that we are bonded to those loved ones with emotional ties that are likely to withstand the slings and arrows of disappointment, loss, and many of life's changes. As soon as we realize that the best antidepressant chemistries will be those that recruit the power of positive social-affective systems, like endogenous neurochemistries such as oxytocin that can melt the psychic pain of separation distress, we will make more progress in developing new antidepressant medications, such as glutamate receptor antagonists (Machado-Vieira et al., 2009; Skolnick, 2009) and more indirect modulators (Burgdorf et al., 2011), and along the way we may learn to use highly effective medicines such as "safe" opioids (Bodkin et al., 1995). Of course, the best medicine is the warmth and comfort we gain from our loving relationships. They satisfy the human soul.

Let us reemphasize that, although the GRIEF system can generate a form of "anxiety," this anxiety is not the same as the dreaded anticipation that emanates from the FEAR system. FEAR and GRIEF are supported by different brain structures and by overlapping but also distinct sets of brain chemicals operating in different parts of the brain. We have yet to fully understand the role that the GRIEF system plays in clinical depression and chronic anxiety. We also have a long way to go in utilizing available agents and devising new effective drugs to treat these conditions. Indeed, we do not really know how current antidepressant drugs work; it clearly is not due to their short-term pharmacological effects. Some believe the drugs set in motion growth factors that help mend broken brain functions, as reflected in the ability of many antidepressants to promote neural proliferation in the hippocampus (Boldrini et al., 2009).

On the other hand, we are well aware that modest doses of opiates are effective antidepressants that take rapid effect, in contrast to the matter of weeks that current medications generally require to become fully effective. Such rapidly acting antidepressants are desperately needed. Unfortunately, the fact that opiates can be drugs of abuse when taken in large quantities has prompted researchers to overlook their great potential for psychiatric medicinal use. Besides the potential of safe opioids such as buprenorphine to serve that need, the emergence of glutamateric blockers as rapid antidepressants (perhaps by blocking the psychic pain of GRIEF) is spearheading the discovery of safe, nonhallucinogenic treatment strategies that also take positive affective processes in animal brains seriously (Burgdorf et al., 2011). Hopefully a better understanding of the GRIEF system will also encourage psychiatric researchers to turn their attention to the possibly beneficial effects of these medications.

Likewise, the more that painful memories can be recontextualized in the context of positive affective attitudes the better off clients may be in the long term (see Chapter 12). As we will see in the next chapter, playfulness, which is the source of one of the most positive social-affective feelings our brains can generate, is not yet systematically or well used in psychotherapeutic contexts. There are surely ways to make this robust positive affect a more common aspect of therapeutic interactions. We may be wise to remember Norman Cousins's (1983) famous idea: Laughter may be one of our best medicines.

CHAPTER 10

PLAYful Dreamlike Circuits of the Brain

The Ancestral Sources of Social Joy and Laughter

What daring it takes
to play for eternity,
to play as ravines sweep down,
to play as a river flows.
—Boris Pasternak, "Bacchanalia," translated by Anesa Miller for Mikhail Epstein (1993)

IN HIS QUEST TO BE free, Boris Pasternak, the great Russian novelist and poet (1890–1960; winner of the Nobel Prize for Literature, 1958), wrote the above lines about deep play in "Bacchanalia." The philosopher Mikhail Epstein (1993) proceeded to reflect that "Pasternak's lines . . . convey the play of nature, that is the ideal of what culture might do—to play, not as chess players but as a river plays." All mammalian youngsters discover within their minds, in Epstein's words, "a wild, naughty, rambunctious creature" resonant with the spontaneity of "all nature—like a mischievous child."[1]

Ask children what they enjoy more than anything else. Their almost invariable answer is "to play!" Playful activities, in their many forms, bring all young mammals great joy. And at the earliest ages, physical rough-

housing—also known as rough-and-tumble play—is the most fun of all, as indicated by the abundant laughter that accompanies such activities, almost equally in boys and girls (Figure 10.1). Psychologists have written a great deal about play, but they do not know how many primary-process PLAY systems exist. It could be a single one—the one for physical play described in this chapter. Playfulness in the maturing human mind, however, extends to the farthest reaches of our imaginations in the stratosphere of our higher mental apparatus, to the point where we can tickle each other with jokes most clever and outrageous. We will not focus on those higher positive psychological issues of the human mental apparatus, which have been explored in various works on positive psychology (e.g., Sheldon et al., 2011).

Physical playfulness is a birthright of every young mammal and perhaps of many other animals as well. Two recent books and two that are somewhat older provide excellent summaries of playfulness across species, much more than could be summarized in this short chapter (Aldis, 1975; Burghardt, 2005; Fagen, 1981; Pellis & Pellis, 2009). There is also a classic monograph about the play behavior of rats, still well worth reading (Groos, 1898), as well as a follow-up on human play (Groos, 1901). It is now certain that a genetically determined PLAY network that mediates positive affect exists in mammalian brains (Burgdorf et al., 2007; Panksepp, 1998a), although many details remain to be scientifically analyzed. So far most of the neuroscience research has been done with laboratory rats, so we cannot be sure how well these lessons translate to humans. Indeed, we know almost nothing about primary-process rough-and-tumble play (henceforth called the PLAY system) in humans, although there is some relevant work on laughter. But because the PLAY system is concentrated in subcortical brain regions, just like all the other basic emotional systems, we can anticipate that many general principles, especially about the neuroanatomies, neurochemistries, and raw affects (social joy), will translate across all mammalian species. How we translate playfulness into humor within our tertiary-process networks of our minds, will not.

It is hard to define play, but you know it when you see it. Perhaps the best general definition has recently been suggested by Gordon Burghardt (2005), consisting of five criteria: (1) The adaptive functions of play are not fully evident at the time play occurs; (2) play is a spontaneous activity, done for its own sake, because it is fun (pleasurable); (3) play is an exaggerated

and incomplete form of adult activities; (4) play exhibits many repetitive activities, done with abundant variations, unlike serious behaviors that are not as flexible; and (5) animals must be well fed, comfortable, and healthy for play to occur, and all stressors reduce play. Burghardt (2005, p. 82) sought to put all these qualities into a single sentence: "*Play is repeated, incompletely functional behavior differing from more serious versions structurally, contextually, or ontogenetically, and initiated voluntarily when the animal is in a relaxed or low-stress setting*" [emphasis in original].

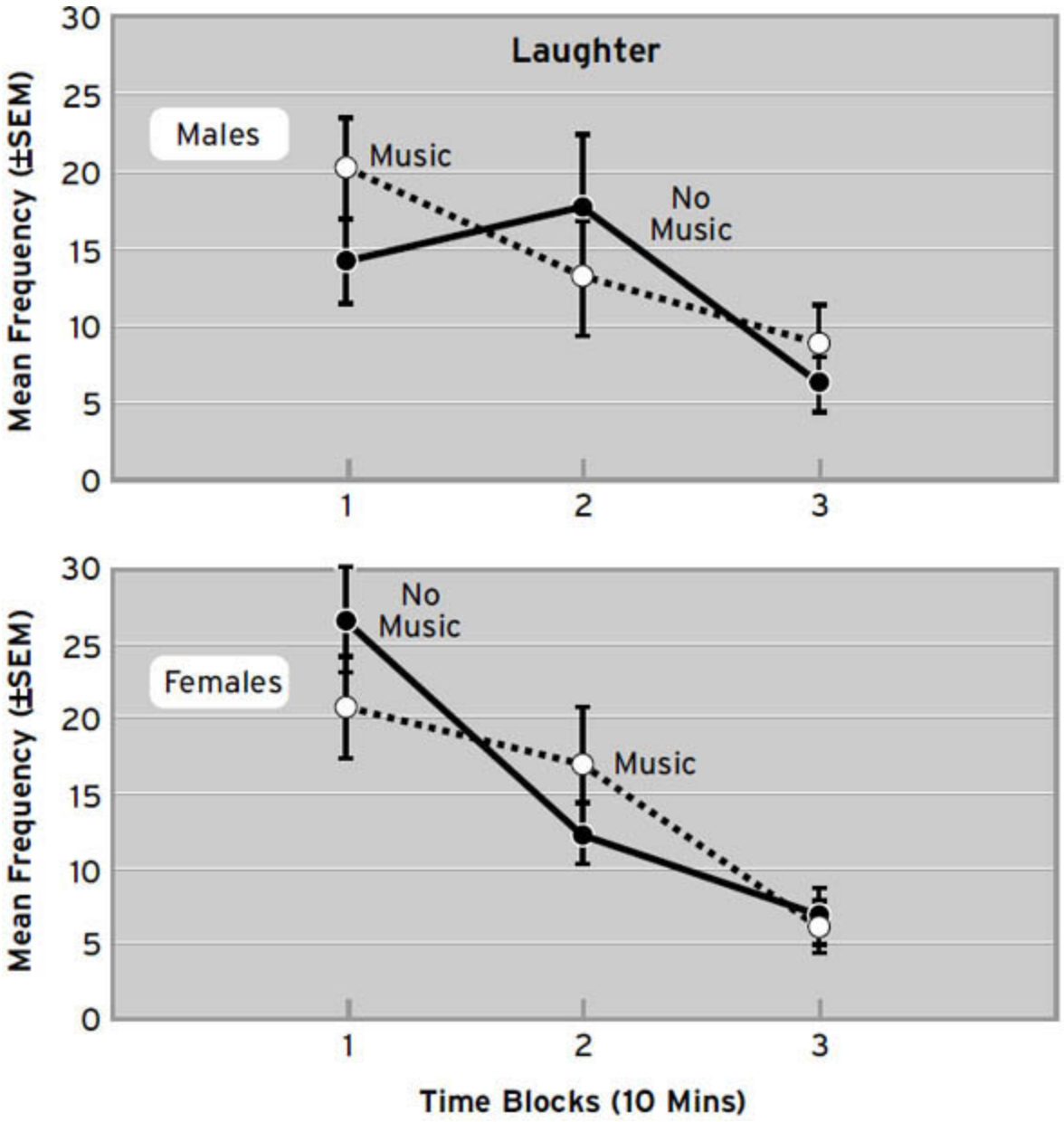

Figure 10.1. Time course of play in pairs of young (4- to 7-year-old) girls and boys during a half an hour of free physical play with no toys available; however, every 5 minutes there was music (Irish jigs) and no music during the intervening 5-minute blocks of observation, and video coding of behavior. Here we see that the amount of laughter diminishes systematically during the play sessions, with no major differences between boys and girls. Also, a total of 19 other play gestures were scored, and practically none showed a gender difference, except for "pushing from the front," which girls did less than boys. The conclusion is that there is no substantial difference in the urge of girls and boys to exhibit physical play, and that gender differences seen in the past are probably due to learning (data adapted from Scott and Panksepp, 2003).

As one can see, dynamic social interaction was not included as a criterion, allowing Burghardt to include exploratory fun under the play concept, which may be largely due to animals using their SEEKING system for personal fun. However, for us, it is the social form of play, often taking the form of "play fighting" that is the most dramatic and joyous form of play, with dedicated brain systems, that also incorporates the exploratory-SEEKING urges into its domain. Also, for us, primary-process PLAY has to be partly defined by social neural circuits, in addition to SEEKING, that generate the dynamic quality of young animals playing with each other. One of our original descriptions of social playfulness was when two juvenile rat pups "are placed together in a non-threatening environment, they rapidly begin to exhibit vigorous fighting: Animals chase and pounce on each other, sometimes unilaterally, sometimes mutually with rapid role reversals. They repeatedly poke and nip at each other, often at the nape of the neck but also on the ventral surface when one animal is pinned" (Panksepp et al., 1984, p. 466). This is the kind of play we will focus on here, for it is the one that generates the most fun, as highlighted by the abundant high-frequency laughter-type sounds—50-kHz chirps—that rats emit when spontaneously indulging in this activity or when tickled playfully by a resilient human being (Knutson, Burgdorf et al., 1998; Panksepp & Burgdorf, 2000).

These chirps are intimately linked to the dopaminergic rewards of the SEEKING system (Burgdorf et al., 2007), helping to explain the pleasure of

exploratory play, which takes the form of predatory practice (such as when a kitten toys with a ball of yarn). We now know that predatory behavior, and hence the playful chasing and pouncing form in young animals, is a developmental outgrowth of the SEEKING system (see Chapters 3 and 4). However, as we will discuss later, this wonderful laughter-type chirping sound can now be used as a direct measure of positive affect in rats (Panksepp & Burgdorf, 2003), which has opened up doors to even understanding the euphoric delights of addictive drugs (Browning, et al., 2011; Burgdorf et al., 2001; Panksepp, Knutson et al., 2002).

Although exploratory object play can be great fun, there is nothing that quite matches the outright euphoria of fully engaged social play, a state that is quite evident to even an untutored eye. One can observe it almost daily when looking into a yard and seeing squirrels leap and chase each other on the grass.

Why does a PLAY urge exist? It probably enables the young to learn nonsocial physical skills like hunting, foraging, and so on. It is also surely important for acquiring many social capacities, especially nascent aggressive, courting, sexual, and in some species, competitive and perhaps even parenting skills. It may be an essential force for the construction of the many higher functions of our social brains. Playful activities may help young animals learn to identify individuals with whom they can develop cooperative relationships and to know who they should avoid. They surely learn through play when they can dominate social interactions and when they should gracefully disengage, submit or accept defeat. Play can also have a darker side. When animals play, they may learn whom they can bully and who can bully them. In short, the brain's PLAY networks may help stitch individuals into the stratified social fabric that will be the staging ground for their lives, and these networks may also prepare them to handle various unexpected events that life will surely throw their way (Spinka et al., 2001).

The PLAY urge is both robust and fragile. It is fragile because a great number of environmental manipulations can reduce play—including all events that evoke negative emotional states such as anger, fear, pain, and separation distress; it is especially sensitive to species-typical fear stimuli such as the smell of predators for rats (see Panksepp, 1998a, Fig. 1.1; Siviy et al., 2006). For instance, if a laboratory researcher has a pet cat at home, and he is not careful to change his clothes before going to work, he will

have a difficult time studying the play of rats because the odor of cats intrinsically scares rats, and fearful rats simply do not play. Likewise, rats are scared of well-lit open spaces; they play in safe burrows, away from the attentions of predators. In addition, hunger is a powerful inhibitor of play (Siviy & Panksepp, 1985), as are many other bodily imbalances, including, of course, illness. This is a general principle: Play only occurs when one is safe, secure and feeling good, which makes play an exceptionally sensitive measure for all things bad. PLAY, however, is also a robust system: If young animals are healthy and feeling good, they almost invariably play together when given the chance.

Currently, some of the more boisterous forms of rough-and-tumble play in human children tend to be discouraged by parents. Few of them consider the developmental fact that diminished opportunities for physical play may have undesirable maturational consequences, such as poorly controlled hyperactive urges that can become so severe as to be pathologized, and are all too often given labels such as Attention-Deficit Hyperactivity Disorder (ADHD; Panksepp, 2007b). Children who exhibit excessive activity become more manageable when given medications such as amphetamines, the same drugs that dramatically reduce playfulness in rats (Panksepp, Burgdorf et al., 2002). Conversely, in rat models of ADHD, abundant daily play can reduce the symptoms of hyperactivity (Panksepp et al., 2003). Further study of play in young humans may allow us to help children diagnosed with ADHD by giving them more play opportunities, as opposed to the drugs that dampen the play impulse. But before we proceed to complex socio-cultural issues, let's examine the evidence that indicates PLAY is an ancestral gift of the mammalian BrainMind.

THE DEVELOPMENT OF SOCIAL PLAY IN YOUNG ANIMALS

Research indicates that the desire to play systematically increases the longer young rats have been deprived of an opportunity to "boogie." Laboratory rats have relatively weak GRIEF systems (probably due to selective breeding to live well by themselves); this very weakness seems to render them especially useful for the study of PLAY. In order to increase an animal's urge to PLAY, investigators must keep them for a period of time in social isolation, a condition that causes many animals, especially primates,

to feel lonely and miserable. Primates have highly developed GRIEF systems and they are intensely bonded to one another. After prolonged isolation, young monkeys become despondent and, following reunion, they huddle together and are initially disinclined to play (Evans, 1967). Apparently, their basic needs for social warmth, support, and affiliation must be fulfilled before they feel playful again. After spending some time with conspecifics (others of the same species), their social confidence is restored and the urge to indulge in carefree play reemerges (Chalmove, 1978; Novak, 1979). It is to be expected that human children would behave in much the same way.

Juvenile rats, however, are not so emotionally dependent. Because they do not suffer as much from separation distress, the urge to play is immediately apparent. After a period of separation (with even as little as 3 to 8 hours, but maxing out at a full day), their PLAY systems are in overdrive, and they quickly engage in rough-and-tumble play as soon as a partner enters the arena. Even when young rats have been kept in total isolation from the time their eyes and ears open at about 2 weeks of age, until the age of 25 days (a time when socially housed rats begin to exhibit the play impulse), they do not become clearly depressed. Instead, their urge to play has been building up, and they play normally, with great enthusiasm, within seconds of being paired with another rat (Ikemoto & Panksepp, 1992). Thus, we can conclude that the urge to play is not learned. It is innate. The evidence indicates that PLAY is one of the primary-process, genetically determined social urges.

Just as the urge to PLAY builds up systematically during social isolation or other periods of play deprivation, the desire for play systematically diminishes when pairs of juvenile lab rats are allowed free play for half an hour (Burgdorf et al., 2006; Panksepp & Beatty, 1980). This indicates that the increased urge to play is like a kind of hunger—a specific hunger for play, and not simply a general social need. This is highlighted by the fact that when young rats are housed in ways that allow them to touch each other through a screen, they still become hungry for play. A similar situation occurs if rats are housed in a busy “jungle-gym” type of living environment, where they can have very intimate bodily contact but the cramped quarters prohibit rough-housing. When released into an open arena, they play eagerly. Likewise, the desire for PLAY builds up in young rats that live continuously with adults that are not very playful. Even though

they have had full body contact and opportunities for many other physical and social interactions, they will play with gusto when given the chance (Hole & Einon, 1984; Panksepp et al., 1984).

Although there are many differences in specific play patterns exhibited by various mammalian species, there is a dynamic similarity in rough-and-tumble play across species (Burghardt, 2005; Pellis & Pellis, 2009). It is an activity of joyful social exchange with a strong competitive edge.

Thus, the evolutionary roots for such activities probably go back to ancient homologous PLAY circuits shared by all mammals. Even though the precise details of play episodes may vary widely among different species, there are hints of practice for future needs. For instance, predatory species such as cats enjoy object play, as exemplified by their batting about the proverbial ball of yarn, important practice for behavior patterns they will need as adults (Byers & Walker, 1995). In contrast, prey species such as antelopes exhibit abundant running with rapid twists and turns, skills they need when evading predators (Byers, 1997). It is also likely that creatures other than mammals, especially birds, exhibit social play, but avian play is less predictable; it requires large free and open spaces, and hence it is more difficult to study scientifically (Aldis, 1975).

Rats show a balanced mixture of mock attacks and eager evasion, which are flight/escape behaviors. In rat play, one typically sees rapid spurts of activity toward and away from a play partner. Sometimes one animal "bowls" the other over, which leads to a flurry of playful chasing. Taking turns, the animals pursue each other, with rapid pivoting, wrestling, and role reversals. They often pounce on each other's backs as if they are soliciting vigorous interactions. These *dorsal contacts* can easily be quantified and have commonly been used as an explicit measure of play solicitations that indicate an urge to play. Usually the recipients of play solicitations respond either by running away or twisting laterally; a bout of wrestling ensues, in which one animal winds up on its back with the other animal on top (for very detailed frame-by-frame analyses and many other fascinating facts about play, see Pellis & Pellis, 2009). This *pinning* posture can also be easily quantified and is the clearest measure of the consummation of a particular bout of play activity (see Figure 10.2). Also, rats exhibit an abundance of joyous 50-kHz ultrasonic chirps during their play; they start with relatively "flat calls" when just getting to know each other and more joyous "frequency-modulated" calls in the midst of vigorous play. As will

be discussed later, there are many solid empirical reasons to believe that this is a form of ancestral laughter, related to SEEKING urges (Panksepp, 2007c; Panksepp & Burgdorf, 2003).

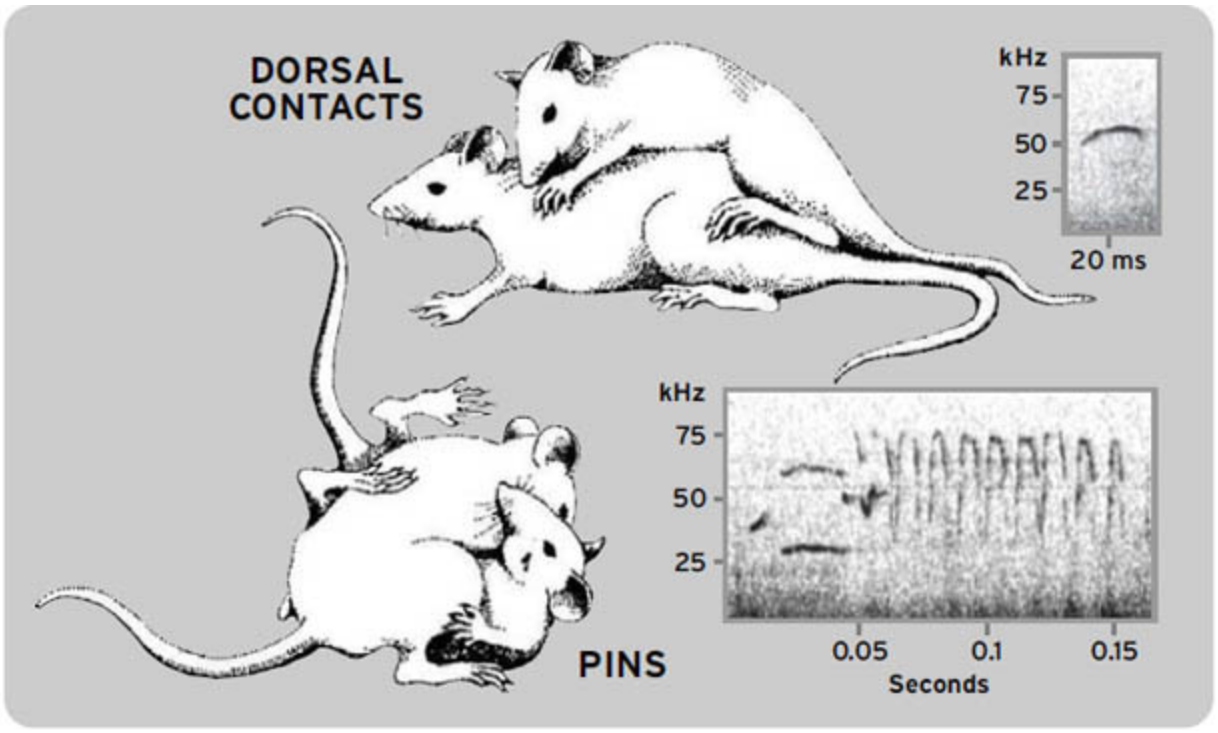

Figure 10.2. Two major play postures that are used to quantify rough-and-tumble play in our work. When animals initiate play, they start by pouncing on each other, especially at the nape of the neck (dorsal contacts), and when they are just exploring a new place they usually emit a modest number of "flat" 55-kHz ultrasonic vocalizations (also called 50-kHz USVs, or chirps) as depicted in the "sonogram" (sound frequency by time graph) in the upper right. However, when rats really get into rough-and-tumble social play, they run around, chasing each other and wrestling, with the easiest measure of play being the number of "pins." During such joyous play, there are abundant frequency-modulated (FM) USVs, which are a direct indication of positive affect in these animals. A sonogram at the lower right depicts a classic example of those, even though there is considerable variability in the exact acoustic wave forms (drawing by Lonnie Rosenberg, and published in Panksepp, 1998a; republished with the permission of Oxford University Press).

Rat play exhibits a characteristic developmental course during the lifetime of the animal, with the amount of play increasing during the early juvenile period, remaining stable through youth, and diminishing as animals go through puberty (Barrett & Bateson, 1978; Panksepp, 1981c; Thor & Holloway, 1984a, 1984b). We presently know little about the neurobiological factors that control this inverted U-shaped maturational function. Presumably it is related to presently undetermined neurochemical shifts that accompany brain maturation across development (Panksepp et al., 1997)—maturation that is, in part, promoted by neurotrophic factors released during play (Burgdorf et al., 2010; Gordon et al., 2003), which can even promote neurons sprouting in areas such as the hippocampus (Wöhr et al., 2009). Also, as with other emotions, neocortical development and the emergence of many higher brain functions tend to inhibit subcortical processes such as those giving rise to PLAY. This developmental trajectory makes sense. As animals become more dependent on learned behavioral strategies, primary-process urges become better regulated by higher brain functions. For example, in adult rats large frontal lesions, as well as lesions in septal regions where many higher and lower brain influences communicate, substantially increase the urge to play (Panksepp et al., 1984, 1994). This suggests that these brain areas participate in the developmental processes that normally diminish play as animals mature.

Play dominance emerges if two rats are allowed to play together repeatedly (Panksepp, Jalowiec et al., 1985; Pellis & Pellis, 1987). After several play episodes, one rat tends to become the "winner," meaning that it ends up on top more often during pins. The average difference is that "winners" end up on top about 70% of the time, while the "losers" achieve less success, ending up on top in about 30% of the total number of pins. Interestingly, the continuation of play appears to require a willingness on the part of the stronger partner to handicap itself. If the stronger animal does not exhibit this kind of reciprocity—if it becomes a "bully" and aspires to end up on top all the time—then playful activity gradually diminishes because the less successful animal begins to ignore the solicitations of the winner. Nobody wants to play with a bully.

MISUNDERSTANDINGS ABOUT PLAY

Developmental and social psychologists have divided human play into several categories: exploratory, relational, constructive, dramatic/symbolic games, and the kind of rough-and-tumble play that one most readily sees in young animals (Slade & Wolf, 1994). Embedded in these psychological taxonomies are two common problems: First, psychologists often confuse PLAY with mere curiosity—with the arousal of investigatory activities promoted by the SEEKING system (Welker, 1971; Weisler & McCall, 1976). Second, many misinterpret PLAY as a form of aggression, as reflected in the common label "play-fighting" for rough-and-tumble play (Aldis, 1975). Although few would go so far as to view PLAY as a manifestation of the RAGE system, there is probably considerable truth to the view that the types of jousting for dominance commonly seen in many species, especially when sexual readiness is high, are somehow related to behavioral refinements that are honed during juvenile play. However, as we will discuss shortly, rough-and-tumble PLAY has no relationship to any angry type of aggression, though prolonged play bouts do often end with one animal complaining more than the other.

Let us consider the first of these problems, the confusion of PLAY with curiosity—with the mere arousal of the SEEKING system (which, we should note, clearly promotes and is active during play). There is robust evidence that PLAY and SEEKING are distinct, albeit interactive, systems. When placed in new environments, animals typically exhibit strong exploratory activity with little tendency to play until they are familiar with their surroundings. Neurochemical evidence may also be taken to support the distinction between PLAY and SEEKING. We have seen that dopamine fuels the SEEKING system, and psychostimulants such as amphetamines strongly increase brain dopamine activity. Increase in dopamine activity produces vigorous exploratory behavior while markedly *reducing* play (Beatty et al., 1982). Blocking dopamine receptors, however, also reduces play (Siviy, 2010).

Although psychostimulants decrease play, dopamine systems are nevertheless aroused during normal PLAY (Panksepp, 1993). Recent work indicates that the high frequency (50-kHz) ultrasonic chirping sounds that rats make during play are vigorously promoted by brain dopamine arousal (Brudzynski et al., 2010; Burgdorf et al., 2001, 2007). Further support for the role of dopamine in play (perhaps through SEEKING arousal) is the fact that various dopamine receptor blockers are quite effective in reducing play

(Siviy, 2010). This is to be expected, however, because rough-and-tumble play involves a great deal of to-and-fro activity and frequent moments of pleasurable anticipation, an emotional state that is fuelled by dopamine. In other words, the fact that dopamine participates in PLAY arousal does not mean that dopamine causes play. Dopamine is secreted in response to many positive incentives, including opportunities to play. Therefore, the secretion of dopamine during play may simply indicate that the animal is engaged in an activity that entails a great deal of positive anticipation and euphoria.

Even if it turns out that dopamine does actively arouse the PLAY urge, however, research has surely not yet determined whether the same populations of dopamine neurons are active during social PLAY as during nonsocial exploration. It may be that some types of dopamine activity arouse the PLAY system while others arouse the SEEKING system. These questions will only be resolved through further research.

The role of dopamine presents us with a bit of a dilemma: Some dopamine activity correlates with play, but when animals are given psychostimulants, which greatly enhance dopamine activity, the urge to play diminishes. How, then are we to understand the fact that dopamine helps to fuel PLAY, when high levels of dopamine arousal reduce play? One possibility is that psychostimulants might arouse the affects that dynamically drive the PLAY urge *tonically* (without fluctuation) to a very high and sustained level, thereby reducing the neurochemical flexibility needed to express play. This could have the effect of inhibiting PLAY by "freezing" its ability to function normally. At these high levels of arousal, the PLAY system may not be able to respond dynamically to *phasic* fluctuations of dopamine. An analogy might be trying to play music through speakers that are emitting a broad spectrum hum. Under normal conditions, the speakers transmit the different tones of music. However, the hum is an overriding steady signal that does not permit the speakers to convey clearly the flexible tones that compose a melody. The speakers would be analogous to the PLAY system, and the broad-spectrum hum would be analogous to the high and continuous arousal caused by psychostimulants. Under these conditions the ludic melody, leading to dynamic playful activities, might be thwarted. But this is just an idea, currently without clear support. Thus, all possibilities need to be considered. Another is that the psychostimulants simply shift animals into

more intense exploratory SEEKING modes that compete with play motivations.

Indeed, this may be a general problem in many drug studies. Clearly, the mere application of certain neuroactive drugs cannot simulate how the relevant brain neurochemical systems actually operate during normal behaviors. For instance, psychostimulant drugs like amphetamines are typically administered peripherally—by injection or by mouth—which has much the same effect on all of the many brain dopamine systems. This does marginally increase happy 50-kHz chirping sounds. However, if amphetamine is placed directly into motivational regions of the brain, especially some of the main projection pathways of the SEEKING part of the dopamine system (into a subregion—"the shell"—of the nucleus accumbens, which is a target region for the mesolimbic dopamine system, see Figure 3.2), chirping increases dramatically (Burgdorf et al., 2007; Brudzynski et al., 2010). In contrast, there are very modest effects in the adjacent "core" of the accumbens, and there are practically none at all from other nearby dopamine-rich areas such as the olfactory tubercle and the dorsal striatum which generally mediates habitual skilled behaviors.

In addition to these ambiguities, as already noted, rough-and-tumble PLAY seems quite easily confused with aggression, especially by untutored observers. Adults may view the shenanigans of young children as aggression, even though the kids see it as unadulterated fun. Despite the fact that play and aggression may be superficially similar, careful scrutiny of the two behavior patterns reveals many differences. For instance, in a real fight, rats often exhibit boxing, consisting of standing on their hind legs and paddling each other with their front paws. Aggressive rats also exhibit a laterally directed aggressive posture called "side prancing," and piloerection, accompanied by many 22-kHz "complaints."

Animals do not initially emit such sounds while playing. However, sometimes play does end up in a real fight, leading to some 22-kHz ultrasonic vocalizations (USVs). When this happens, playful signs—the frantic hopping, darting, and pouncing—immediately stop. Indeed, play always declines systematically during prolonged observation, and this is partly due to the fact that complaints, as signaled by the 22-kHz USVs, begin to increase, and the positive chirps decrease accordingly (Figure 10.3). Indeed, if one had allowed little rats to play on one side of a test chamber for the first 15 minutes, and the other distinct side for the second

15 minutes, each animal shows a considerable preference for the side where they started the play session. This is no doubt due to the fact that the second half of the play session was not as delightful for them.

The distinction between play and aggression is further supported by the fact that testosterone promotes aggression between adult males, while having little effect on their urge to play, except that in some animals increased testosterone levels reduce play as a result of the animals more readily getting into real fights (Panksepp, personal observation, 1985). In other words, after several days of high-dose testosterone treatment, juvenile rats play less, apparently because bouts of play quickly devolve into outright aggression, whereupon social interaction quickly loses its carefree quality.

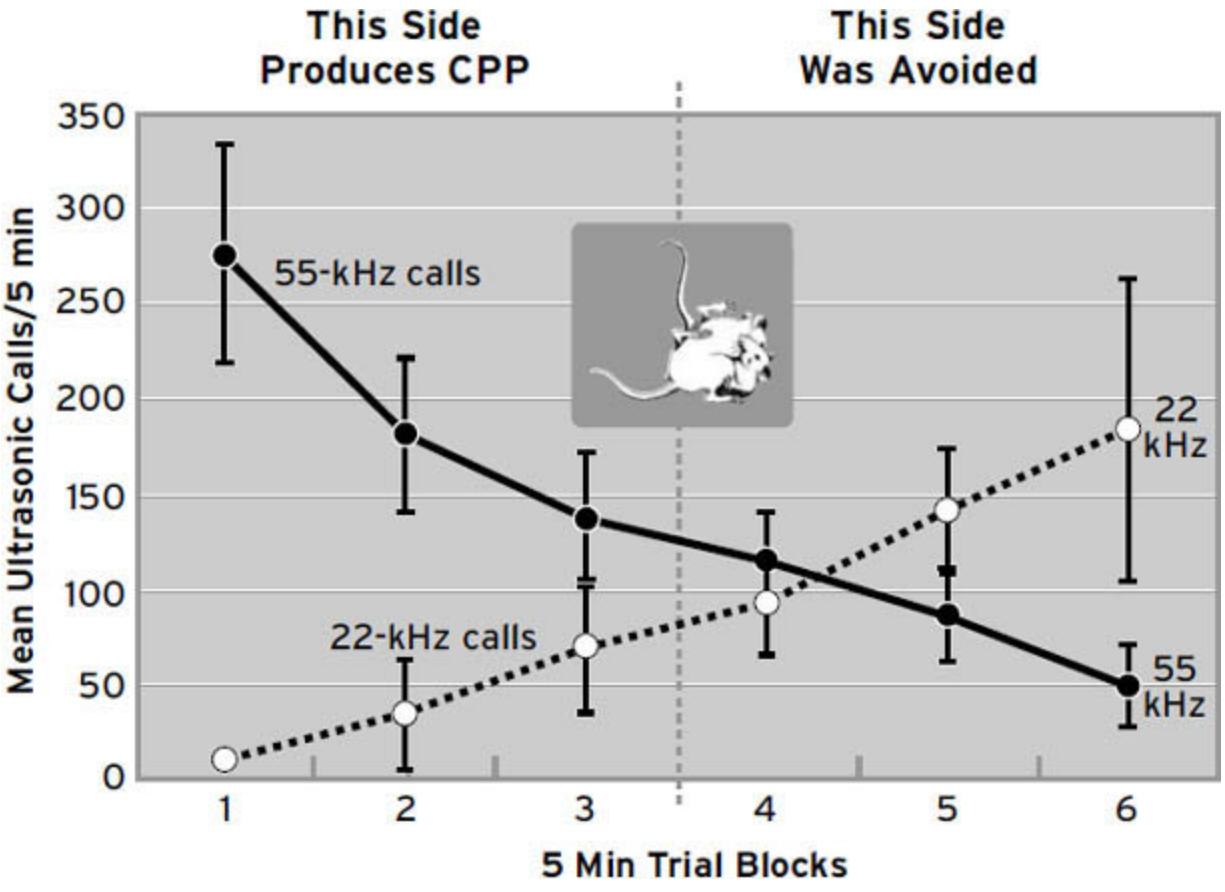

Figure 10.3. This depicts positive and negative emotional vocalizations in rats during a half-hour free-play period similar to the one shown for boys and girls (see Figure 10.1). During such a session, the positive 55-kHz calls decline systematically, as play diminishes. But the 22-kHz USVs, which

can be considered to reflect negative affect (i.e., they are complaints), increase systematically. If the first 15 minutes of play were allowed on one side of a box with distinct walls, while the second 15 minutes were on the other side, the animal consistently showed a place preference for the side where the play started and lots of happy USVs were seen (data adapted from Burgdorf et al., 2007).

It has also been observed that different social rules apply to play and aggression. For example, when adult males fight for dominance on the home turf of one of the animals, the resident male invariably wins. This is not the case with play fighting—the resident "wins" no more than if the animals were tested in a neutral play chamber (Panksepp et al., 1984). During play, there are no sustained defensive or aggressive postures in which one animal lays on its back while the other maintains a menacing top position for extended periods. But in adult fighting, such postures are common. In play, a joyous pattern of motor activity moves along gracefully and rapidly for quite a while. As already noted, however, play does not always remain emotionally positive. Just as among children, disputes can develop that interrupt play. Then rats will "complain" with the 22-kHz distress call as play grinds to a halt. But typically this happens only for a short while, since the rats usually regain their good spirits rapidly. However, these complaints in rats increase gradually during a prolonged play session, as the positive vocalizations decline and the overall amount of play diminishes, perhaps because more negative neurochemicals are being released in the brain (Burgdorf et al., 2006). As we will see, this is an important issue to focus on in the midst of childhood play, especially if we want to promote the development of a sensitive, socially intelligent BrainMind.

Although play can end in tears with children, and vocal complaints with rats, it is important to emphasize that rough-and-tumble PLAY is highly rewarding for all participants; in real fights, only winners get temporary gratification. How do we know PLAY "fighting" is affectively positive? Both "winners" and "losers" of play "fights" rapidly learn instrumental tasks, such as making fast and appropriate choices in a T-maze, *in order to gain the opportunity to play* (Normansell & Panksepp, 1990; Pellis & McKenna, 1995). The only difference is that winners barge quickly into the

"playground" without a pause, while losers are a bit more hesitant in entering the field of play. Also, the playing field is full of joyous chirps that have been validated to reflect positive affective arousal within the brain (Burgdorf et al., 2007).

THE NEUROANATOMY OF PLAY

Neuroscientists have found that ticklish stimulations of certain regions of the body are especially liable to arouse playfulness—for instance, on the back of the neck and the shoulder regions of rats. When these parts of the skin are touched in just the right way, rats initiate play. If these same parts of the skin are anesthetized, then the playful moods of animals do not seem to coordinate, and we see less and less play as the level of skin anesthesia is increased (Siviy & Panksepp, 1987a). However, anesthetization does not decrease the number of times that rats pounce on each other's backs—the number of dorsal contacts, which are a good measure of the urge to play, is not diminished.

The urge to play is reduced, however, by lesions to certain nuclei in the thalamus that process touch information. It is important to note that most sensory systems, including touch, divide at the level of the thalamus on their way to the higher regions of the brain, with some of the "information" heading to the neocortex, while the rest influences lower reticular regions of the brain. These latter regions appear to convey the affective impact of sensory inputs. The touch components that promote play do not go primarily to the neocortex but rather to more ancient midline thalamic regions such as the parafascicular complex and the posterior dorsomedial thalamic nuclei. Both of these are included under the heading of nonspecific thalamic reticular nuclei. Bilateral lesions to these brain areas, especially in the parafascicular nuclei, reduce dorsal contacts as well as pinning, indicating that this brain damage really does reduce the desire to play. Animals with very small amounts of brain damage still engage normally in other types of complex motivated behavior such as foraging for food (Siviy & Panksepp, 1985, 1987b). At present, these nonspecific reticular nuclei are as close to specific substrates for PLAY as we have. It is vital to keep in mind that we can remove the entire neocortex, approximately a quarter of a young rat's brain, and the rat will still play quite normally.

Obviously, play motivation may involve parts of the brain that govern the movements of rough-and-tumble play. Brain areas such as the cerebellum, the basal ganglia, and vestibular systems generate and regulate movement. Lesions to these areas can disrupt the flow of playful activities. But these lesions also compromise virtually all complex motor activities, so there is little chance of evaluating which of their functions are *specific* to play. Other brain lesions arouse emotional states that inhibit play. For example, lesions to the ventromedial hypothalamus (VMH) cause animals to become pathologically aggressive, which curtails playful activity. This does not indicate, however, that the VMH normally facilitates play. It only indicates that aggressive animals are not playful.

There is some controversy regarding the involvement of the amygdala in play. In our estimation, its role in play motivation is secondary and not critical. The amygdala is embedded in the temporal lobes, and when monkeys and cats have had their temporal lobes removed, a condition known as the Klüver-Bucy Syndrome results. Animals with this syndrome are apt to be hypersexual, hyper-oral (they mouth, and often eat, anything), and they exhibit little fear (Klüver & Bucy, 1939). Yet these animals are quite eager to play, even though many other social capabilities and nuanced social responses are lost. Pellis and Pellis (2009) report, however, that larger-brained mammals generally tend to play more than smaller-brained ones, and the most playful tend to have relatively large amygdaloid regions. Still, major deficits in play have been hard to find with modest lesions of the amygdala (Panksepp et al., 1984).

Likewise, we (and Pellis & Pellis, 2009) have noted that neocortical participation is not essential to the functioning of any of the other six emotional systems, nor is it essential for PLAY. Decorticate animals play abundantly, although they are pinned less frequently than control animals. This does not indicate that they are less playful. They rough-house just as much as controls, but they simply are less likely to get turned over, to be pinned, during their rollicking about. This may be a motoric side effect, because decorticate animals appear to keep lower to the ground in general. But it may also be due to the relative social insensitivity of these animals. Intact animals are sensitive to the fact that other animals will not want to play unless they have a chance to win part of the time. As mentioned above, stronger animals handicap themselves in order to keep the fun going. Presumably this sensitivity requires a level of higher cerebral participation

that decorticate animals obviously do not have. Still, if one looks at play dominance when such animals are pitted against each other, the neurologically intact rats do not invariably prevail over the decorticate ones. Indeed, the outcomes are basically even, with each kind of animal winning about half the time (Panksepp et al., 1994).

Although our understanding of the neuroanatomy of PLAY circuitry remains in its infancy, we can be sure that this primary-process emotion is organized at subcortical levels of mammalian brains. This does not mean that higher brain regions have no function in play. Of course they do, most especially in the symbolic play that leads many of us to love to play musical instruments, to act in theatrical productions and movies, and above all to invent games that can add endless delight and excitement to life. All of which is profoundly dependent on a cortex that listens intently to the ancestral messages from below. Thus, there is growing evidence that the primal urge to play is an important influence in helping program higher brain regions—to become happy adult brains with abundant creativity and zest for life.

ROUGH-AND-TUMBLE PLAY, TOUCH, AND LAUGHTER

Because most young mammals exhibit the urge to engage in rough-and-tumble play, we take the view that this is the most fundamental form of play. The more sophisticated human forms of play may be secondary-and tertiary-process variants of the primary rough-and-tumble urge that we share with other animals. Adult human play impulses can be manifested in many ways. As individuals mature, a great deal of human play comes to be focused on verbal interchange. The persistent verbal repartee that often characterizes friendly teasing has apparent parallels to the dorsal contacts and pinning of rough-and-tumble play. One tries to arouse the other individual with some provocation, at times even sharp and biting comments; then, if others respond, there is often a desire to "sock it to them" with an especially clever response. If successful, this yields peals of laughter among the young and chuckles among the elderly. This type of repartee may be repeated many times, with each trying to best the other—to be the cleverest—until it is clear that one prevails or until each is satisfied that he or she is a match for the other. When the latter happens, the

individuals presumably have a high potential to establish a special respect and friendship.

Before returning to some of the higher functions of our PLAY urges—which allow many social interactions to feel especially positive—let us pause to reconsider how the various senses control the basic urge to play. For example, rats that are blind play vigorously. Although rats do not need sight, blindness does curtail the playful activities of visually oriented creatures like ourselves. Still, blind children enjoy play as much as those who can see, and they laugh readily when playing. Rats whose olfactory capacities are compromised play almost normally. And children with stuffy noses also play well, no doubt, although no one has researched this formally. In short, neither sight nor smell plays an essential role in the urge to play.

Deaf rats play somewhat less than hearing rats, perhaps because they are insensitive to the ultrasonic rat "laughter". However, as already noted, the main sensory system that instigates and sustains play is touch. To reiterate, there are two major touch pathways. The *specific* pathways up to the neocortex, which carry cognitive information about tactile stimuli (e.g., where you have been touched), are not critically important for play. However, pathways that run through the *nonspecific* reticular nuclei of the thalamus carry the affective feelings aroused by touch, and they are very important for rough-and-tumble play (Siviy & Panksepp, 1987a, 1987b). Animals whose play-instigation areas are anesthetized simply can't coordinate their play urges any more. The fact that touch and PLAY urges coincide suggests that certain kinds of touch generate affective experiences that are very important in arousing the urge to play. We see this most clearly in tickle games.

These findings suggest that rats have specialized skin zones that send signals into the PLAY system when they are touched. Also humans can tickle rats more easily around the neck and shoulders than toward their hindquarters. In other words, rats appear to have "play skin" or "tickle skin," with specialized receptors sending information to specific parts of the brain that receive communications of playful intent between animals. Obviously, humans also have tickle skin, which is situated at the back of the neck and around the rib cage. Of course, this is one of the easiest spots to tickle young children, producing a playful mood.

Apparently, the PLAY system is also tuned to the perception of stimuli that are unpredictable. For instance, one cannot tickle oneself. Also, the underlying neural systems are designed so that children can't easily be their own rough-housing play partners (although there are solitary forms of exploratory and fantasy play). Tickling requires others to participate in the arousal of playfulness. This is not merely a sensory phenomenon but is very much an internal brain function.

Many people believe that laughter is a human phenomenon and that it is invariably associated with humor, such as the punch line of a joke. However, laughter does not require much in the way of cognitive complexity. For example, children love to stage skits and shows, but as they attempt to perform seriously, all too often they end up giggling with glee. The highest levels of childhood laughter occur when children are physically playing. Clearly, even human laughter is rooted in the ancient PLAY systems that generate joyful social engagement in other mammals.

There is now strong evidence that laughter-type vocalizations are emitted by many other mammals besides humans. The strongest evidence comes from humans tickling other animals. Just as physical tickling is one of the easiest ways to provoke laughter in young children, the same turns out to be the case for many other animals, ranging from all great apes to laboratory rats. It has long been known that a cyclical pattern of panting and grunting vocalizations, similar to human laughter, can be induced in chimpanzees and gorillas by tickling them (Provine, 2000). Indeed, a recent study (Ross et al., 2009) directly contrasted tickling-induced vocalizations in all of the great apes, leading the authors to assert that

> at a minimum, one can conclude that it is appropriate to consider "laughter" to be a cross-species phenomenon, and that it is therefore not anthropomorphic to use this term for tickling-induced vocalizations produced by the great apes. This term has been used in previous work on tickle- and play-related vocalizations in several nonhuman species . . . and the current results provide clear support for such usage. (p. 1109)

For many, it is surprising that the concept of laughter can be extended to mammalian species as lowly as the rat. Many serious-minded neuroscientists were unprepared to accept this discovery (for an overview, see Panksepp, 2007c, 2010d; Panksepp & Burgdorf, 2003, 2010). To evaluate whether rats laugh, we used a simple tickling approach—basically, human hand-play—which induced sustained peels of high-frequency

chirping (about 50 kHz, well outside the human hearing range, and hence requiring special sonographic measurements). Indeed, this chirping is most effectively evoked by tickling those same body regions where rats normally solicit play—especially on the nape of the neck. Of course, full body tickling is even more effective. And just like children, rats love it! Because these same chirps are very abundant during the natural play of rats, we would be hard-hearted to assume they have no relationship to our own laughter. Indeed, just like after one has tickled a little child, one can get peals of laughter just by "threatening" a tickle. A very similar response is seen in rats: Laughter will begin to be generated just by approaching finger movements (Panksepp & Burgdorf, 1999).

But we need not rely merely on behavioral research in identifying this connection to human laughter, as serious brain research has been conducted on the laughter of rats. Laughter circuits have been mapped in rats using localized brain stimulation to evoke laughter-type chirps. The circuitry runs along the meso-limbic SEEKING system and is strongly controlled by dopamine; wherever we find a laughter spot, juvenile rats readily self-stimulate—voluntarily "juice up"—those brain sites (see Figure 10.4 summarizing the mapping work of Burgdorf et al., 2007). Although we still have much to learn about the subcortical regions that generate primary-process human laughter, abundant research findings suggest that the brain regions associated with rat laughter also play an important role in generating human laughter (Black, 1982; Chen & Forster, 1973; Poeck, 1969; Sterns, 1972; Wild et al., 2003). Thus, existing evidence is consistent with the likelihood that human and rat laughter are generated by evolutionarily related subcortical circuits.

In the previous chapter, we said that distress vocalizations are useful indicators of aroused GRIEF. Likewise, laughter-type chirping is a prime indicator of aroused playfulness in rats. Laughter, like playfulness itself, is an unconditional instinctive response that arises, under the right social-environmental conditions, from ancient regions of the mammalian brain. It is not learned by imitation, because blind and deaf children laugh readily (Eibl-Eibesfeldt, 1989). We have easily bred for the tickle-induced chirping response in rats (Burgdorf et al., 2005). And the animals that exhibit abundant chirping are happier by all measures we have taken, while those who don't chirp much exhibit a negative affect and a susceptibility to depression (Brudzynski et al., 2010; Burgdorf, Panksepp et al., 2008;

Harmon et al., 2008). If these vocalization patterns are truly homologous across mammalian species, we may eventually come to understand much about the primal nature of human joy by studying the circuits that generate euphoric chirping in rats (Panksepp & Burgdorf, 2003; Panksepp, 2007c).

Of course, the bottom line of such an analysis will be whether we can identify the major genes that contribute to the construction and action of specific neurochemical circuits that allow animals to be playful. We are making progress on that front in the rodent model, and we will relate some details of this progress later in this chapter. This kind of work can provide rigorous neuroevolutionary evidence, pro or con, for the evolutionary continuity of laughter and the nature of social joy across distant species (Panksepp, 2007c). In short, the study of rats may tell us more about the primal nature of human PLAY and laughter than any other strategy available to scientists.

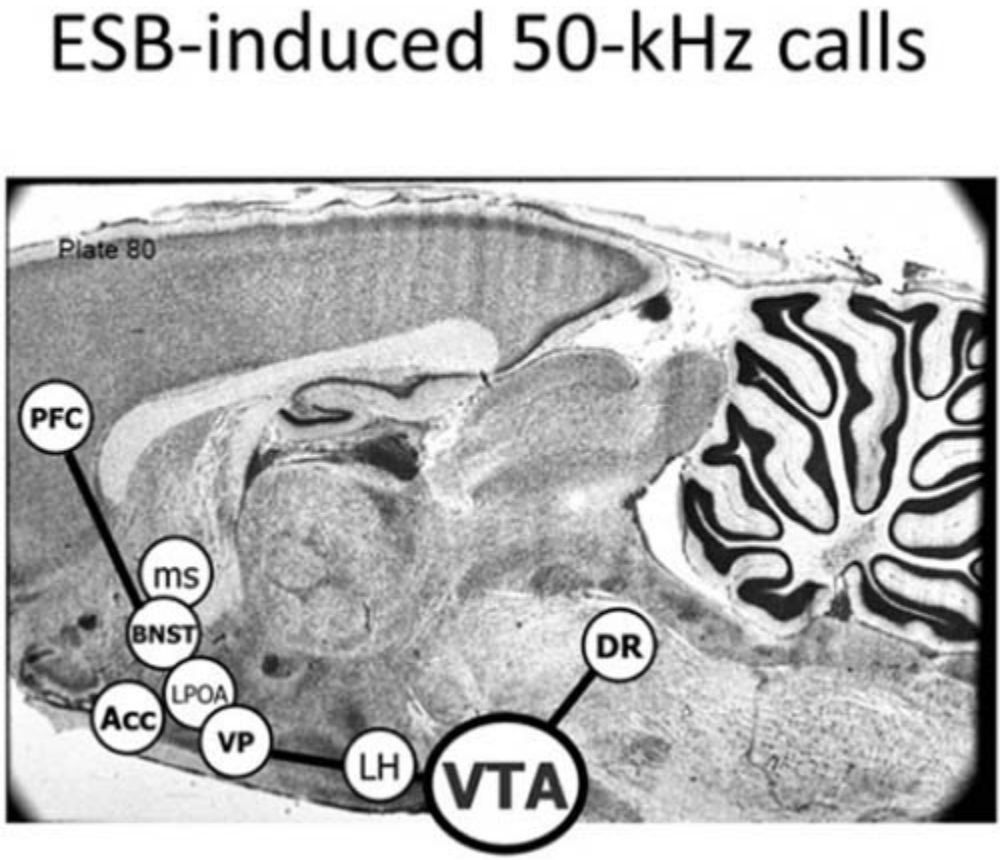

Figure 10.4. A summary of brain areas from where one can evoke abundant frequency-modulated (FM)-type 50-kHz USVs during localized electrical

stimulation of the brain in rats. These tend to follow the trajectory of the SEEKING system. In every place one can evoke such calls, animals will also self-stimulate the electrode sites, and dopamine-blocking drugs selectively reduce these calls. Anatomical areas depicted from the bottom up are the dorsal raphe (DR), ventral tegmental area (VTA), which along with the lateral hypothalamus (LH) may be the most effective sites, ventral pallidum (VP), nucleus accumbens (Acc), lateral preoptic area (LPOA), bed nucleus of the stria terminalis (BNST), the medial septal area (ms), and the medial prefrontal cortex (PFC) (summary of data reported in Burgdorf et al., 2007; we thank Jeff Burgdorf for sharing this summary).

More About Rat Laughter

We now know an enormous amount about rat laughter, certainly much more than we know about primary-process human laughter. Just as with little children, tickling is a positive incentive for young rats. They seek out this kind of stimulation and rapidly begin to chirp when they receive cues that are associated with tickling. One can also evoke chirps in response to the direct stimulation of a number of brain sites. It is important to emphasize the fact that every brain location from which chirpy sounds can be evoked in rats supports self-stimulation—animals readily turn on brain stimulation, indicating the experience associated with those sounds is positive (Burgdorf et al., 2007). The readiness to self-stimulate in this way indicates that these sites provide affective pleasure and that chirping is a signal that rats are having a delightful experience. In short, just like our own kids, young rats readily learn to enjoy tickles. The fact that this laughter system primarily follows the SEEKING system also helps explain why this happy sound is present when animals and children are anticipating non-social treats (Knutson et al., 2002).

Thus, it should come as no surprise that 50-kHz calls are abundant when animals are getting addicted to affectively desirable drugs (Burgdorf et al., 2001; Knutson et al., 1999). In fact, this measure can be used as a self-report of drug desire (Browning, et al., 2011; Panksepp, Knutson et al., 2002). Likewise, during sexual arousal in both male and female rats, 50-kHz calls are emitted, especially during the “courting-soliciting” excitement preceding copulation (McGinnis & Vakulenko, 2003). As we noted earlier,

it is somewhat surprising that when copulation is finished, male rats begin to exhibit 22-kHz alarm-type calls, only slightly different than those produced in really dangerous situations; that is, the post-copulation calls are not as monotonously flat but have a ripple of frequency modulation (Burgdorf, Kroes et al., 2008). We mentioned earlier that the male rat may simply be informing the female to stay away now that he is quite satisfied and ready to attend to grooming himself. More interestingly, perhaps this vocalization, usually used as an alarm call, may help keep other males at bay. In promiscuous species such as rats, sperm competition is a big factor in who actually reproduces. The emission of pseudo alarm calls would be a terrific evolutionary adaptation to help ensure that other male rats remain at a distance, facilitating the likelihood that his own sperm achieves the goal of fertilization. To anthropomorphize excessively, perhaps this is a kind of deception that "pulls the wool" over the eyes of other nearby males who would be all too ready to be the next to copulate with a sexually receptive female.

The Dark Side of Human Laughter

In human beings, the dark side of human laughter has long been known to occur in response to seeing others hurt, humiliated, or embarrassed (slapstick humor, so to speak). Dark laughter recognizes the ludicrousness of a victim's predicament coupled with the feeling that one has been psychologically luckier and perhaps even smarter than the unfortunates who have been at the brunt of some misfortune. This kind of scenario is regularly exploited in theatrical comedy. It is important to note that in competitive play encounters in human children, laughter is invariably exhibited more by apparent victors than losers, even though this effect may not have been scientifically documented yet. Likewise, the perpetrator of a practical joke is much more likely to laugh than is the recipient.

These patterns suggest that laughter is often recruited by competitive, perhaps even aggressive, urges. It can be used to inflict emotional pain on a rival. Perhaps such higher forms of twittering can only occur in sophisticated cognitive creatures such as primates, who can use their mental faculties for many purposes. This would be close to the classical Freudian interpretation of humor as an acceptable veil for otherwise unacceptable sexual and aggressive impulses (Freud, 1905a/1968). But there is no

evidence for such processes in other animals. Neuroscientific research suggests that laughter in the service of aggression is not an intrinsic aspect of the primary-process PLAY system. That is a much higher mental function.

THE NEUROCHEMISTRY OF PLAY

It is remarkably easy to inhibit play by using pharmacological manipulations. It is difficult to determine, however, whether a drug's inhibitory effects include specific changes to the PLAY system or merely generalized behavior disruptions brought on, for instance, by increased anxiety, cognitive disruptions, or sedation. Brain-imaging studies indicate that there is widespread release of opioids in the nervous system during play, particularly in the preoptic area (POA, which also governs sexual and maternal behaviors; see Chapters 7 and 8). These findings suggest that opioid release may play an active role in arousing the PLAY system (Panksepp & Bishop, 1981; Vanderschuren et al., 1995). Also, with the discovery of an endogenous cannabinoid system in the brain, and with the long-established cultural recognition that marijuana can bring on the giggles, it is not surprising that facilitation of "pot-like" activity in the brain does promote playfulness in rats (Trezza & Vanderschuren, 2008, 2009).

The role of opioid activity in PLAY has now been extensively studied in animal research. Very low doses of morphine actually promote playfulness and social dominance (Panksepp, Jalowiec et al., 1985; Vanderschuren, 2010) and also control play dominance. It is known that, when predicting "winners" in a play encounter between two animals, the animal's increased body weight gives a distinct advantage, just as in wrestling among boys. But brain neurochemical activities also exercise significant influence. It has been found that between two animals of equal physical strength, if one is given a small dose of an opiate receptor stimulant such as morphine while the other is given an equivalent dose of an opiate receptor antagonist such as naloxone, the animal receiving morphine always becomes the winner (Panksepp, Jalowiec et al., 1985). These results indicate that higher levels of brain opioids, sufficient to generate feelings of social confidence (i.e., reduced separation distress as discussed in Chapter 9), facilitate winning in playful competitions. Low levels of brain opioid activity, on the other hand, generate feelings of greater social need and hence insecurity. This puts

animals at an emotional disadvantage, making them more likely to lose. Of course, to facilitate play and confidence, *opiate doses must be low*. High doses sedate animals and reduce all social behaviors including play, with very high doses inducing catatonic immobility.

As in all findings in the behavioral and psychological sciences, there are alternative explanations for these results. For instance, opiate receptor antagonists such as naloxone may reduce playfulness but they may also simply diminish positive feelings that normally arise from all kinds of friendly social interactions. Another possibility is that opiates can reduce pain, so animals receiving the naloxone may experience some of the rougher activities during play as being more disagreeable than do animals receiving morphine. Regardless of the interpretation, the effects of opiate manipulations on play dominance are remarkably robust in animals that receive these agents at the outset of their mutual play experiences. If, however, patterns of dominance have already been established in the social relations of a pair of play partners prior to such pharmacological manipulations, the patterns of dominance do not shift as readily in response to these neurochemical shifts. This variation in outcome suggests that past social learning exerts a powerful force on play behavior.

Social deprivation is another factor that increases the desire to play, which suggests that it should be possible to artificially increase the desire to play. There may be highly specific play-promoting neurochemicals in the brain, perhaps neuropeptides. However, no such substances have yet been identified, although some candidates are emerging from genetic research (as discussed below). Part of the problem in searching for relevant evidence is that virtually all of the neuropeptides must be administered directly into the brain, and we really do not know enough about play circuitry to place the substances into the appropriate areas. However, we have evaluated the effects of a few neuropeptides, including oxytocin and CRF, both of which we found to reduce play; we also found that vasopressin does not as clearly affect play (Panksepp, Crepeau et al., 1987). We are still searching for the neurochemical system that will “turn on” playfulness in animals that are not psychologically ready to play. That effort has not been very successful so far. Perhaps what is needed is a symphony of neurochemical changes occurring all together in the right pattern. Only when we have fathomed the neurochemical tunes that are playing as animals partake in ludic activities will we begin to have a profound neural understanding of playfulness in the

mammalian brain. Other neurochemical systems will surely be discovered that have more specific effects on play.

Some progress is being made on this front. In studying brain gene-expression patterns that result from play, the discovery of elevations of insulin-like growth factor-1 (IGF-1) and a glutamate receptor subtype led to behavioral studies indicating that molecules that promote such increases do also facilitate playfulness (Burgdorf et al., 2010). Indeed, preliminary data shows that playfulness can reverse depressive symptoms, including strengthening brain areas that are often damaged by stress (Wöhr et al., 2009). And we are well on our way to identifying new antidepressant molecules using the above strategies, with a glycine site, glutamate receptor modulator that can both stimulate (at low doses) and block (at high doses) and thereby gently increase positive affect and diminish negative affect that is already in clinical trials (Burgdorf et al., 2011).

In sum, it is eminently clear that PLAY is a very rewarding process in the brain. As investigators have studied the neurochemistries that regulate play, they have also developed ideas about which brain chemistries are important in the generation of social rewards. The first chemicals that seemed important were the endogenous opioids, which are secreted during play (Panksepp & Bishop, 1981; Vanderschuren et al., 1995). Considering that SEEKING urges are likely very active during play and that dopamine seems to mediate euphoria in the brain, the likelihood is high that dopamine also participates in the euphoric aspects of play. In addition, it has become increasingly clear that the endogenous cannabinoids, which seem to promote other forms of positive affect in the brain as well, are a substantial part of the PLAY-reward package (Trezza & Vanderschuren, 2008, 2009). Finally, new gene-discovery approaches are beginning to yield other play reward-mediating molecular pathways in the brain (Burgdorf et al., 2010).

FUNCTIONS OF PLAY

Many investigators and theorists have considered what the functions of play might be. Suggestions have fallen into two broad categories: social and nonsocial. Among the possible social functions are the learning of various competitive and noncompetitive social skills. These range from behaviors that facilitate social bonding and social cooperation, to those that promote social rank and leadership, as well as the ability to communicate effectively.

Among the potential nonsocial functions of play are the learning or enhancement of such assets and abilities as physical fitness, cognitive functioning, the skillful use of tools, and the ability to innovate in the face of unexpected events (Spinka et al., 2001). Nonsocial functions can range from complex cognitive skills such as the ability to think creatively in a wide range of situations, to very specific aptitudes such as the skills acquired by young predators learning to hunt and by young prey learning to avoid predators. Unfortunately, there is no large and substantial scientific database for any of these ideas.

Surely, play increases reproductive fitness in various ways, but sexual-type behaviors are very infrequent during the course of rough-and-tumble play in rats, even though various other animals show quite a bit more mounting behavior during play. One might expect that animals that had no play during juvenile development would be disadvantaged when it came to adult sexuality. In fact, male rats that have been socially deprived during the entire juvenile period (21–45 days old) exhibit quite normal primary-process sexual behaviors when placed in the presence of a hormonally primed female. However, we have found in some of our unpublished research that in a competitive situation including two males and just one receptive female, the play-experienced animals are more effective in thwarting the advances of those who had little juvenile play. Thus, it does appear that juvenile play experiences may bestow an advantage in competition for access to reproductive opportunities.

Compared to animals that have had little history of play, those with a history of abundant play experiences spend more time with others suggesting a social-bonding (friendship) function of play. In fact, young animals like to spend more time with older animals that still chirp a lot (e.g., our high-chirpy lines of animals described above) than those that do not chirp much (see Figure 10.5). Such a function could be very useful for establishing future social alliances and cooperation and perhaps even empathy. On the cognitive side, some investigators have reported increased nonsocial problem solving in play-experienced rats. But we have encountered repeated difficulties replicating those results. Social effects, on the other hand, have been easier to document. Rats deprived of play are often more fearful and certainly more aggressive in various social situations (Potegal & Einon, 1989). Although more data are needed on such issues, it

does seem that animals that have played little tend to be more irritable and less socially creative.

AN INTERLUDE: PLAY AND DREAMING

It seems likely that PLAY is instrumental in honing a wide range of social and nonsocial skills—it is an experience-expectant process that prepares animals for future challenges. Data on how this happens are scarce. But let's be creative. Might PLAY be linked to the functions of dreaming? Both play and dreaming seem to be experience-expectant functions of the brain designed to evaluate past events as sources for creative and useful future behaviors. Perhaps play functions in ways that are complementary to dreaming. Both may help organize information in the brain in ways that promote higher-order affective responses to future life events. In other words, maybe both play and dreaming allow animals to test solutions to complex problems that they confront in real life. If so, we suspect that playfulness should have a bigger role in psychotherapy than it currently does. But let's first look at the phenomenon of sleep itself.

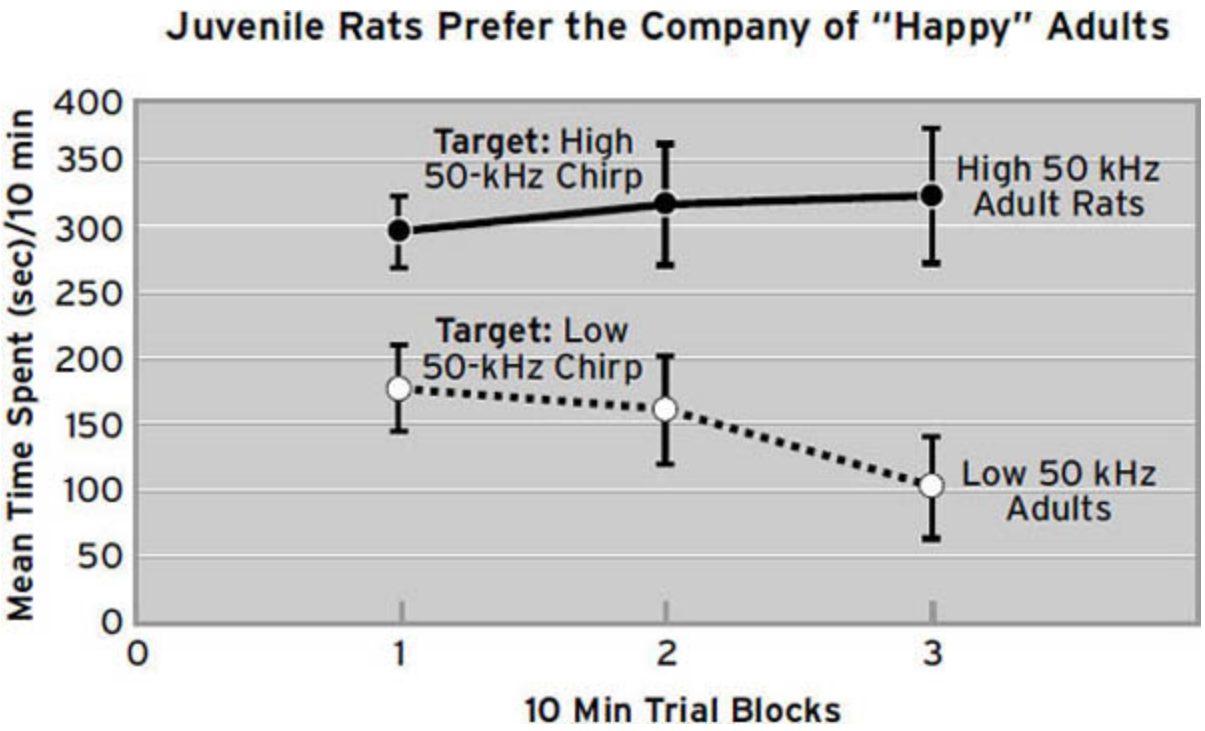

Figure 10.5. Juvenile rats were given a choice of going to two sides of a T-maze, each end of which contained an adult male rat, who differed in their social temperament. One side had an animal that exhibited abundant 50-kHz chirps, and the other contained an animal that exhibited low chirping. During a half-hour test session, young animals clearly preferred to be with the "happier" adult, and this effect became larger across the test session (data by Panksepp & Burgdorf, unpublished data).

Neuroscientists now know that mammalian brains contain endogenous daily rhythm generators—circadian clocks that engender periods of wakefulness and sleep throughout the day. The main circadian clock for sleep is situated in the neurons of the suprachiasmic nuclei (SCN), positioned at the base of the brain just above the optic chiasm, where the optic nerves cross over, providing both cerebral hemispheres with visual information from each eye. These SCN neurons are especially sensitive to the chemical melatonin and are also otherwise light sensitive. Melatonin is secreted from the pineal gland and powerfully influences the suprachiasmic nuclei: When light fades, this promotes sleep in people and in most animals (rats are nocturnal but have higher levels of melatonin after nightfall as well, indicating that darkness drives melatonin production).

The two primary sleep states in mammals are (1) *slow wave sleep* (SWS, also often called non-REM sleep, or NREM), which is typically dreamless, and (2) *rapid eye movement sleep* (REM), during which people and animals vividly dream. While REM sleep and dreaming do have distinct brain mechanisms, the two are usually well coordinated. A very specific site in the ventrolateral preoptic area has been identified as an important SWS generator, although it is clear that the neocortex also has intrinsic SWS generators—sleep is partly regional in higher parts of the brain (Krueger et al., 2008). REM sleep is generated by areas quite a bit lower in the brain stem just below the midbrain. During REM sleep, there is a sustained muscular relaxation, which typically prevents animals from acting out their dreams. Thus, the big antigravity muscles remain relaxed during REM, generating atonia throughout the body. But there are also storms of various phasic components during dreaming, as reflected in a variety of small muscular twitches, the most well-studied of which are the rapid eye movements for which this "paradoxical" phase of sleep is named. When

people and animals are in REM sleep, they also move their fingers, lips, noses, toes, various muscles of the middle ear, and so on. The point is that such peripheral twitches do not result in any coordinated whole-body behavior. The muscular twitching during REM sleep is reflected in typical EEG readings as enormous spikes of firing, especially within the visual system.

Key brain structures that generate SWS and REM sleep, as well as generators for waking life, lie quite deep in the brain stem. The SWS mechanisms reside higher in the brain stem, the basic waking mechanisms reside lower down in the brain-stem reticular formation, and the REM generators reside even farther down. Thus, mammals have an unusual brain arrangement, especially when one considers that higher regions of the brain generally evolved more recently than lower ones. The most influential mechanisms for generating SWS are situated in higher regions of the brain than the basic neural systems that allow us to be awake. The executive mechanisms for REM, are situated as the lowest, and perhaps most ancient, of the three. If we accept that structures located lower down in the central nervous system are generally more primitive than those found higher up, somehow we need to make sense of the fact that the major waking mechanisms of mammalian brains evolved more recently than the basic REM-dream generators. We must take this apparent inversion with a grain of salt, however, because evidence is accumulating that the experiences of dreaming arise from higher brain regions than REM sleep (Solms, 2000). In particular, evidence now suggests that dopamine-mediated SEEKING arousal may be of great importance for dream generation (Léger et al., 2010; Léna et al., 2005).

Despite these ambiguities, we still need to confront a seemingly upside-down state of affairs in our own brains: The REM arousal networks apparently are more ancient than our brain-stem waking systems. To make sense of this paradox, we might consider that a primitive form of emotional wakefulness may have evolved prior to the kind of wakefulness that we associate with neocortical function (with all of its sensory awareness and thought). In other words, in ancient evolutionary history, raw primary-process consciousness might have initially existed exclusively as a kind of dreaming-type wakefulness—one that was full of emotional arousals. This type of simple affective wakefulness may have been superseded by more cognitive frames of mind. But the emotional arousals may still prevail

during REM sleep. As a result, the dream contents of human and animal minds may shift as their prevailing emotional arousals shift. In order for higher cognitive brain regions to become optimally useful for learning and thought, it may be important to exercise emotion-related cognitive possibilities in the safety of dreaming sleep, thereby perhaps better helping integrate cognitive and affective issues. During sleep, such emotional arousals remain hidden behind the motor paralysis called REM-atonia, but they are not completely eliminated, being evident in the many twitchings of the extremities. Perhaps our emotionally rich dream-life is a residue of the progressive evolution of that kind of dual mentality; in the beginning such arousals may have been largely affective but, with brain expansion, they attained a balance between ancient affective and more modern cognitive processes. This may facilitate complex problem solving (Levin et al., 2008).

The idea that an ancient emotional form of consciousness prevailed early in brain evolution is supported by the fact that when the brain mechanisms for atonia are damaged selectively, animals still exhibit regular periods of dreaming-type sleep. Such animals act out their dreams because their large antigravity muscles no longer become so flaccid that they cannot move about. These strange "oneric" periods provide a window into their, and perhaps our own, ancient emotional minds. For instance, cats, whose inside eyelids (nictitating membranes) remain closed during these acted-out dreams (rendering them essentially blind), exhibit four major types of behavior patterns: predatory stalking, fearfulness, lashing out in apparent anger, and periodic bouts of grooming.

Our interpretation is that when a highly affective, nonreflective, dream-type consciousness first evolved, it was attended by abundant emotional activities whose free expressions were gradually inhibited and regulated in the course of evolution. This was because such simple-minded solutions to living were no longer as adaptive as they once were. The massive expansion of higher, more-cognitive brain regions in mammals may have required the evolution of new arousal mechanisms in the brain to help sustain waking in neocortical regions, thereby allowing higher, more cognitive forms of consciousness to emerge. Thus, the more ancient brain arousal mechanisms controlling simple-minded emotional arousals, which may have been all that reptiles ever needed, would gradually have been suppressed and remolded as ones that controlled the arousal of REM sleep. In this way, dreams may still be controlled primarily by ancient emotional arousal states

but by ones that in more modern animals allow cognitive information to be better integrated with emotionally stressful arousals. This could be a way to allow ancient frames of mind to still regulate higher information processing in more recently evolved animals.

In addition to its deep position in the brain stem, there is another reason to think that REM might be a primitive type of waking state that was brought under inhibition because it no longer sufficed to promote survival optimally: REM is found in mammals but not in reptiles or fish. Birds show only modest REM periods, for just a few seconds at a time. It seems quite unlikely that the brain mechanisms that generate REM sleep evolved uniquely in mammals instead of emerging from preexisting ancient brain functions. Surely at earlier stages of brain evolution, animals had simpler forms of consciousness, and with higher brain evolution, those ancient solutions had to be integrated with the more recently evolved brain functions. It is possible that, during waking, PLAY is the brain system that promotes such integration in a way that is functionally similar to dreaming during sleep.

In sum, the projection of the evolving mammalian BrainMind toward cognitive sophistication required a major evolutionary step: It needed the construction of new arousal systems to regulate the waking states of the thalamus and neocortex, as well as a new system to inhibit simple-minded emotionality (now expressed as REM activity). We know that both these systems exist in mammalian brains. The systems that arouse the cortex include biogenic amine (dopamine, serotonin, and norepinephrine) as well as acetylcholine-producing cell groups situated in the ascending reticular activating system (ARAS), located in the upper regions of the pons. In addition, various neuropeptide neurons, such as the orexin (also called hypocretin) ones concentrated in higher brain areas such as the lateral hypothalamus, are necessary for the smooth transition from slow-wave sleep into REM; without them animals and humans exhibit narcolepsy, the sudden collapse from waking into the REM state (McCarley, 2011; Zaharna, et al., 2010). The part of the brain that generates atonia during REM sleep is also different from those that generate the emotion-laden phasic activities of REM. It is found directly below the locus coeruleus—the largest norepinephrine cell group of the brain, which facilitates arousal throughout the cortex and with particular force during emotional states (see Figure 1.1).

Why are we considering these arcane issues in the context of a PLAY chapter? We envision a possible connection between PLAY and REM sleep: If we are correct in thinking that a key function of REM within the mammalian brain is to promote the integration of complex affective information, PLAY systems may perform a similar function during waking life. We suggest this possibility because, in play, many types of emotional behaviors are exhibited in the context of nonserious interactions. Supporting this view is the fact that both REM and PLAY are heavily under control of neurotransmitters such as acetylcholine, dopamine, norepinephrine and serotonin. Similar chemical mediators may be indicative of similar functions. PLAY may consolidate the diverse behavioral components of different emotions under the sway of a feeling of social joy, allowing children to gradually develop habitual creative and positive ways of responding to their physical and social environments. The PLAY urge may be of critical importance in the cultural and epigenetic construction of sophisticated social brains that can understand the emotional states and motives of others, opening the doors to sophisticated social cooperation and fellow feelings of camaraderie, compassion, empathy, and solidarity with and toward others. PLAY promotes social intelligence (Goleman, 2006).

REM dreams may exert very similar functions on the diverse primary-process affective components that invade and give meaning to every life. In other words, dreaming and play may have synergistic functions in the epigenetic creation of mental lives. As we have noted before, it is now clear that only a few higher mental functions are endowed by our evolutionary heritage within the higher cognitive regions of our BrainMinds. Most are learned, under strong cultural influences. The basic emotional systems we have focused on here all participate in constructing our cognitive strengths and weaknesses as well as consolidating each of us as unique personalities. The integration of our affective potentials with our cognitive abilities is created by the magic of each individual's unique *developmental landscape*.

EPIGENETIC EFFECTS OF PLAY ON HIGHER NEOCORTICAL FUNCTIONS

The principle of epigenesis (for discussion of this concept, see Chapter 6 and 9) is especially apt when considering the PLAY system. Even though

the neocortex is not an essential participant in the generation of PLAY, playfulness exerts an especially strong effect on the neocortex, leading to many changes in gene-expression profiles. When children play, their activity promotes epigenetic changes in this organ. Brain imaging of neuronal metabolism has revealed high levels of activity in the neocortex and many subcortical regions when animals are at play (see Panksepp, 1998a, Fig. 15.7; Gordon et al., 2002). Research indicates that social play also activates neural growth factors (such as brain-derived neurotrophic factor [BDNF]) in certain regions of the brain, most clearly in the frontal cortex and amygdala (Gordon et al., 2003). But BDNF is all over the brain, and hence the cerebral effects are very widespread, apparently promoting positive feelings in certain circuits and probably negative feelings in others.

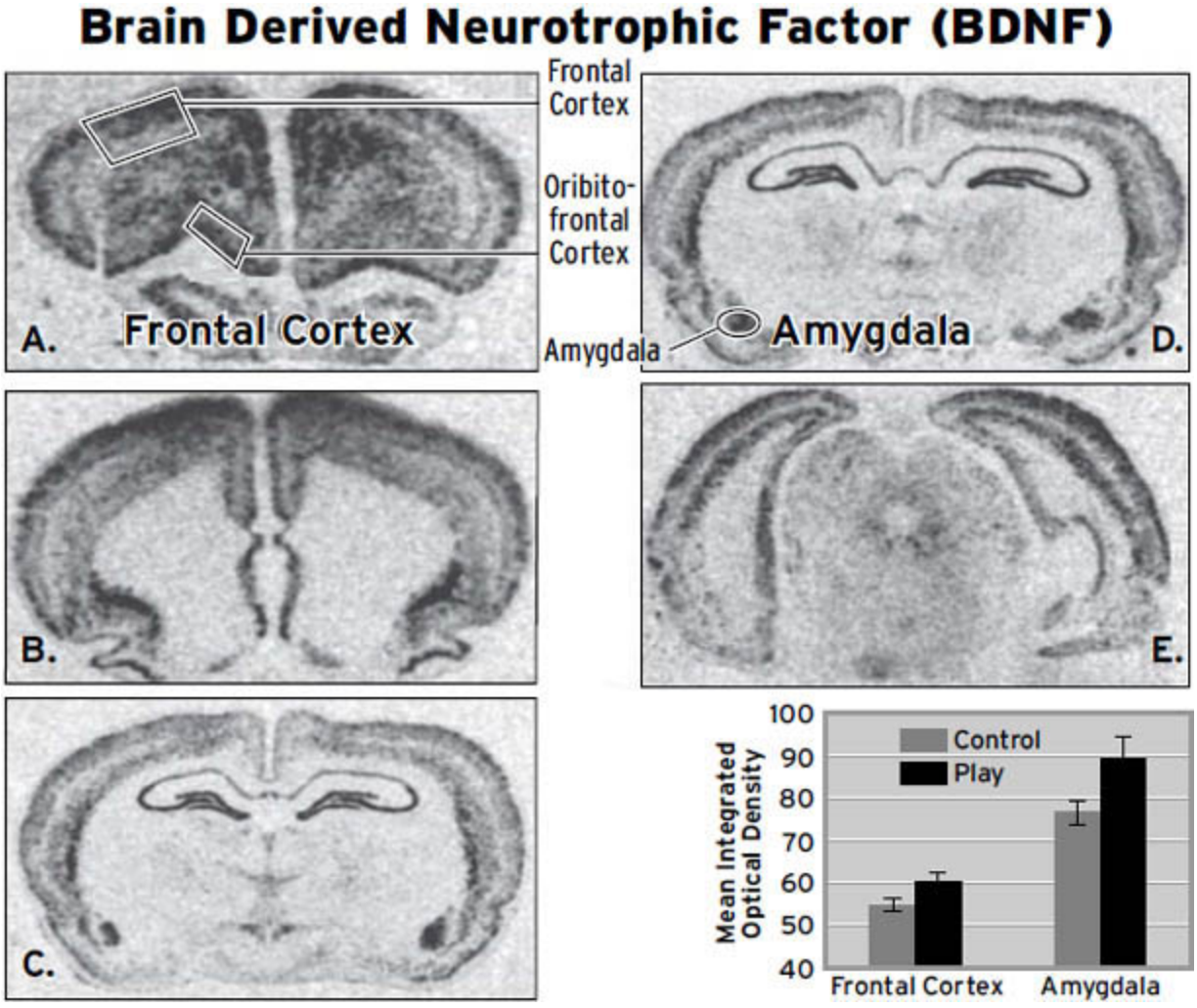

Figure 10.6. Pictures of BDNF gene expression (via in situ hybridization), of coronal rat brain slices (front to back; A–E), in animals with and without half an hour of social play. As shown in the histograms, this widespread neuronal growth factor was elevated in the frontal cortex and amygdala (data from Gordon et al., 2004).

A recent, more comprehensive, brain gene-expression analysis has indicated that activity of about a third of the 1,200 brain genes we evaluated in frontal cortical regions is rapidly modified by play (Burgdorf et al., 2010). It is reasonable to provisionally assume that the dynamic brain changes evoked by play facilitate brain growth and maturation, perhaps epigenetically creating prosocial circuits of the brain, perhaps partly by refining frontal-lobe executive functions (see Figure 10.7). We have recently identified molecular pathways that promote playfulness and positive affect, with the brain "fertilizer" IGF-1 (Insulin-like Growth Factor) being highly aroused during play. IGF-1 has proved to be a positive hedonic molecule in the brain (Burgdorf et al., 2010). Therefore, it is ever more likely that one of the effects of PLAY activity may be the creation of new prosocial neural pathways in the neocortex through epigenesis—the long-term modification of gene-expression patterns as a function of experience.

How might PLAY help program the neocortex? One of the dilemmas of play is that it takes children to the edges of their emotional knowledge. When that happens, there will inevitably be conflicting emotional feelings that deserve to be immediately worked through with the help of caring adults. Bad things will happen during free play and, without supervision, play could easily lead to one child bullying another to achieve social dominance. Under watchful adult eyes, however, every such moment of conflict becomes a wonderful opportunity for positive prosocial learning. Thus, in our era where young children's activities are more controlled than ever, we do need caring people to supervise playgrounds so that they can gently educate at those critical moments.

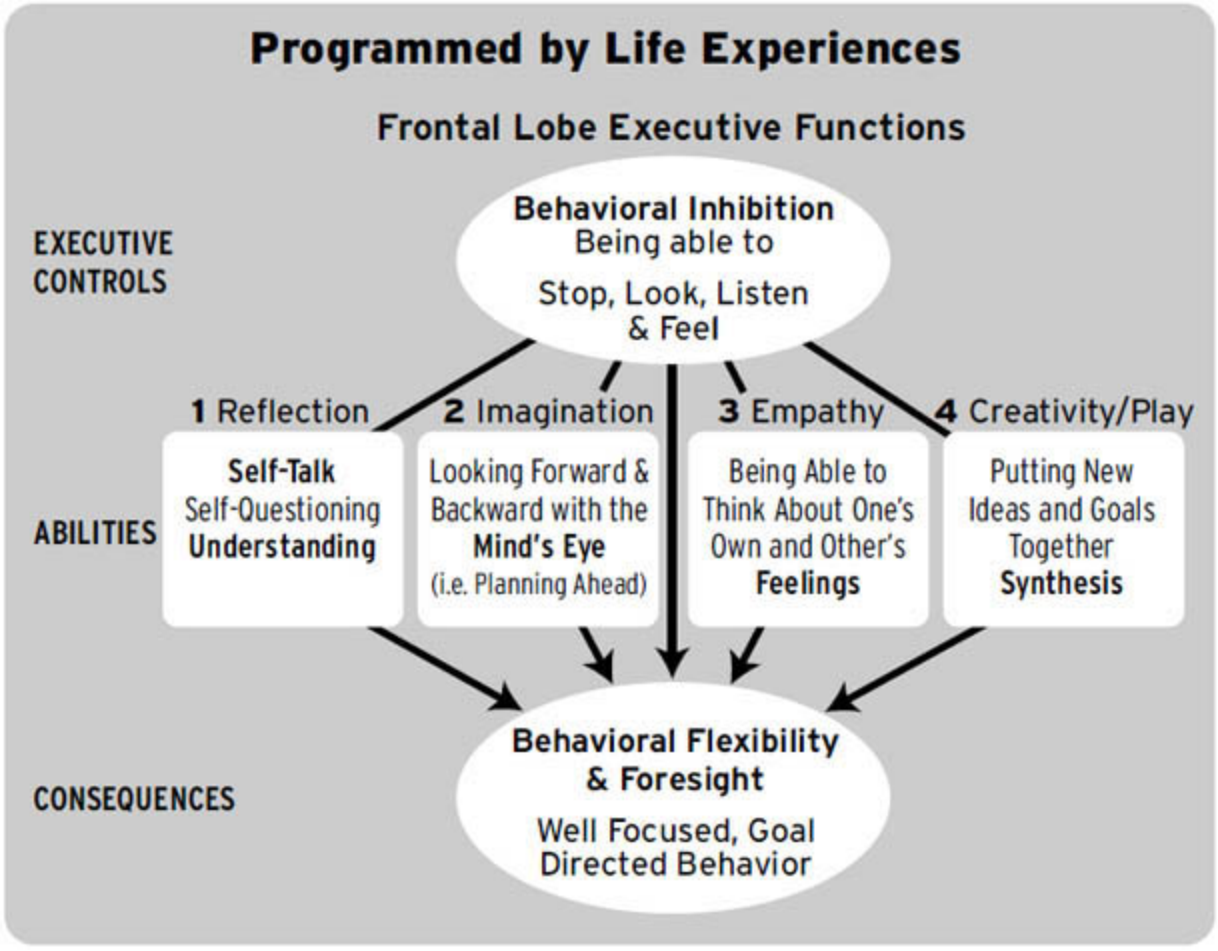

Figure 10.7. A synoptic overview of frontal lobe functions that may be slow to mature in children diagnosed with Attention-Deficit Hyperactivity Disorder (adapted from Panksepp, 2007).

Following our first well-controlled ethological analysis of human social play (Scott & Panksepp, 2003), we endeavored to evaluate this proposition informally in half-hour preschool play sessions. When prosocial expectations were gently but firmly conveyed and the reward was immediate continuation of play, young children understood and rapidly internalized the social rule of "do unto others" in order to continue having fun (Scott, 2001). These considerations highlight the importance of PLAY in the social development of the child.

The connections among PLAY arousal, mind development, and epigenetic changes in neural pathways, especially social ones, surely have implications for the practice of medicating young children with

psychotropic drugs. For example, as we saw above, neuroscientific research indicates that psychostimulants inhibit the PLAY system: Animals given these medications will clearly play less. If PLAY arousal is an important promoter of socially induced epigenesis and the gradual creation of prosocial brains, then the long-term administration of psychostimulants like methylphenidate (Ritalin) may have deleterious effects on the development of children's personalities. And we already know that PLAY arousal exerts a powerful effect on the neocortex. Thus, the administration of psychostimulants may also change the ways in which PLAY programs the neocortex.

PLAY DEPRIVATION: ADHD-TYPE IMPULSE CONTROL DISORDERS?

The fact that the administration of psychostimulants dramatically reduces both play and hyperactive symptoms in children indicates a possible connection between the PLAY system and Attention-Deficit Hyperactivity Disorder (ADHD). Parents of hyperkinetic children often complain that one of the undesirable side effects of psychostimulants is the reduced playfulness of their children. Perhaps ADHD in children is sometimes an indication of a play-starved or especially robust PLAY system, rather than a sign of psychopathology. Although we now know that ADHD children are anatomically (and hence functionally) a bit deficient (~5%) in their frontal-lobe executive functions (Castellanos & Tannock, 2002), this typically only becomes a social problem when kids on the low end of that frontal-size spectrum enter school. They are not as mature and cooperative as children who have better brain-mind regulatory functions.

Presently the treatment of choice for children with ADHD is methylphenidate and related psychostimulants, which chemically have brain effects similar to cocaine except for lower potency and speed of action. For a long time it was a paradox that psychostimulants—drugs that promote motor arousal—would often *calm* troublesome children. Now, an emerging rationale for the use of psychostimulants is the assumption that ADHD children have deficits in prefrontal cortical structures and activities; hence, this part of the brain needs to be stimulated so that it can promote attention and thereby better inhibit excessive emotionality. This should enhance children's ability to concentrate and to learn, but evidence for a

learning benefit is rather slim. Our controversial conclusion is that, although there are demonstrable brain problems with a small minority of ADHD children, most of those diagnosed with ADHD have no clinically relevant brain disorders (Panksepp, 2007b). Many of these children merely have problems with social compliance when their urges to play are thwarted.

If at least part of ADHD is a reflection of excessive desire (or hunger) for impulsive, playful activities, it becomes a profound societal issue whether it is ethical to put children on drugs because of such traits. Obviously, it is essential to maintain attention to academic matters in the classroom, but is it appropriate to induce compliance in children by giving them psychoactive drugs that reduce their playfulness? At the very least, more benign interventions should be attempted first, such as the provision of abundant rough-and-tumble activity early in the morning before class, under the watchful eye of playful young adults who are ready to promote social learning and to intervene gently when bad things happen.

Our past work with an animal model of ADHD has demonstrated that long-term abundant daily play helps reduce impulsive behaviors as juvenile rats mature (Panksepp et al., 2003). And early play can also make adult animals less aggressive and defensive (Potegal & Einon; 1989; Einon & Potegal, 1991). In addition to evidence from the animal kingdom, it has been noted that pathological aggression in human beings often follows from a childhood marked by a dearth of playfulness, even though other contributory factors are certainly required (Brown, 1998). Although some of the skills learned during play may eventually contribute to dominance behavior in adulthood, there is presently no clear evidence linking abundant rough-housing play with adult forms of aggression. It is quite clear that PLAY circuits are largely independent of aggression circuits and that play typically teaches people and animals how to better get along with each other.

Thus, we believe that if the power of PLAY is well recruited in our educational systems, especially at the preschool level, we will be able to reduce the all-too-frequent diagnosis of ADHD. Given the potentially deleterious long-term effects of psychostimulants like Ritalin, we suggest that children might better learn to control themselves in classrooms and assimilate academic material, if they start the day with half an hour of active play.

One thing is certain: During play, animals are especially prone to behave in flexible and creative ways. It is not surprising that play interventions have been used successfully in educational and therapeutic settings (i.e., play therapy) to facilitate the efficient acquisition of new information and behavioral modification (e.g., Power, 2000). However, since play is fun, it could also be used as a reward for desired behavioral change. To what extent would children be willing to discipline themselves with academic tasks if availability of extra rough-housing play were made contingent on good academic performance? The benefits, for both classroom discipline and educational progress, might be enhanced if the availability of physical play was used to systematically reward scholarly achievement. These considerations imply that we must view this ancient evolutionary brain function as a potentially desirable activity, rather than as a disruptive force whose energies need to be suppressed or dissipated on the playground after the earnest business of education has been completed.

PSYCHOSTIMULANTS AND DRUG ABUSE

There is also the worry that children's consumption of psychostimulants might induce an increased sensitivity to and craving for drugs of abuse, like cocaine or methamphetamines. This potential effect has never been measured in children, but it has been assessed in other animals. Preclinical research provides well-controlled data on the long-term consequences of psychostimulant exposure. Adult animals routinely become sensitized to periodic administration of all psychostimulants. In short, their nervous systems become chronically hyper-responsive to various drugs of abuse, and this increased sensitivity is reflected in increased drug desire (Berridge & Robinson, 1998) as well as an increased eagerness to pursue all types of hedonic rewards. In the vernacular, this increased intensity of motivation reflects a shift of normal desires: from "I want it," so to speak, to "I WANT IT, and I WANT IT NOW." Psychostimulant sensitization makes animals more urgently "consumerist" and more eager for all kinds of external rewards, from gustatory treats to sex (Nocjar & Panksepp, 2002). By contrast, if there is anything we should wish to sensitize in the brains of ADHD children, it is the urge for prosocial activities.

Although no study has yet attempted to evaluate the intensification of desire for drugs among medicated children versus nonmedicated ones, it is

long past time to evaluate whether psychostimulant-induced "sensitization" has transpired in kids medicated for ADHD. This could be done by contrasting the acute physiological effects of psychostimulants in children following their very first medications as compared to those that have been chronically medicated in the past. If it turns out that, in fact, these medications produce lasting changes, we should worry that such effects are not beneficial to children. Although we know that very young animals do not sensitize as readily as older ones (Solanto, 2000), we do know that they exhibit some sensitization (Laviola et al., 1999; Panksepp, Burgdorf et al., 2002). It is well known that such brain changes can promote elevated tendencies for drug seeking.

It is also worth considering that Tourette's syndrome, with its bizarre nervous impulses—which lead to tics and sudden verbal expletives, commonly including "forbidden" expressions such as curses and slurs (Chase & Friedhoff, 1982; Comings et al., 1991)—may represent aberrant play impulses, or components of play impulses, circulating without restraint through the nervous system. Pharmacological evidence provides some support for this hypothesis. Dopamine-blocking agents, which presently are most effective in bringing the symptoms of Tourette's syndrome under control, are also very effective in reducing playfulness in animals (Beatty et al., 1982, 1984; Panksepp, Normansell et al., 1987). Although these connections are highly speculative, if we keep our minds open to such possibilities, we may achieve a better understanding of the nature of play as well as some of the perplexing disorders of childhood.

OTHER CLINICAL CONSIDERATIONS

When children play together, they form friendships that give them a degree of emotional independence from their parents. PLAY helps develop the capacity to feel happy and self-determining. This allows children to feel grown up, self-reliant, and capable. It is an inestimable boon to their self-esteem and feelings of friendliness to others. Even pet animals, especially devoted and playful dogs, can help develop this potential if human companionship is scarce. Because PLAY is so important in cementing friendships, it is a central element in allowing children to mature. Well-honed play instincts, refined to be sensitive to the emotional needs and desires of others, allow children to function effectively outside of the family

arena. This may appear paradoxical to some. After all, PLAY is sometimes seen as a trivial pursuit in comparison to work. Nonetheless, this activity helps to produce satisfied and self-actualized adults because it promotes emotional growth and social sensitivity. Play helps prevent depressive disorders, and it promotes nerve growth in areas of the brain like the hippocampus, which can often show signs of stress-induced injury in depressed people (Wöhr et al., 2009).

When children experience deficits in their ability to play, they often appear depressed and envious of other children (Power, 2000; Powers et al., 2009; Ross et al., 2010). It is no wonder. If they have difficulty engaging in play, they can become resentful seeing others having fun together. One way or another, children, even friendless children, will normally find a way to play. Some invent imaginary friends. No doubt these fantasy (at times delusional) companions are also fabricated to reduce feelings of GRIEF, but knowing that the PLAY system exists and that all children have the urge to play allows us to understand why lonely children do their best to find or invent joyful companions.

The urge to PLAY with other children, if well nurtured, naturally leads to social competence and emotional independence from the nuclear family, and this paves the way toward successful maturation through adolescence. It may also solidify an affectively positive foundation for the mind, even down to the neurochemical level (Burgdorf et al., 2010), promoting children's smooth transition toward the accomplishment of satisfying prosocial goals throughout adulthood. If young children don't have a regular playmate, it would be wise for parents to make sure that a little rough-and-tumble activity is in the daily social diet of the child. Obviously, pets can often serve as beneficial play companions for children, once again highlighting that this is a MindBrain process that can be shared across different mammals.

SUMMARY

Until recently, neuroscientists and psychotherapists have tended to ignore the possibility that all young mammals, including our children, have a fundamental urge to PLAY—to engage in joyful competitive interaction. Perhaps play was seen as "childish" and therefore unimportant. On the contrary, a rigorous scientific approach suggests that a fundamental brain

system, common to all mammals, accounts for this universal inclination. Current research suggests that the PLAY system may be especially important in the epigenetic development and maturation of the neocortex. Further understanding of this system may hold a key to addressing certain problematic childhood emotional problems. The goal of early childhood education should be for kids to "thrive by five" (that is the current slogan for child development in the state of Washington). To achieve this goal, playfulness has to be part of the overall equation. The universal recognition of every child's need to play may help shape wise social and educational policies in the future.

Overall, a play-deprived child probably has a higher than normal probability of not only being diagnosed with ADHD but also of becoming reclusive and a potential menace to society as an adult. Of course, the development of human personality is a multifactorial process, and little in human adult life can be traced to a single cause. Poor rearing is commonly accompanied by many other participating factors ranging from poor nutrition to aggression in the home environment. A lack of secure infant bonding and a lack of early play are, however, certainly contributory factors promoting adult irritability and aggression (Brown, 1998).

It may be wise for society to help create the conditions under which all our children can *really* play throughout their childhood years. The difficulties that more and more children in modern societies encounter in being able to have a full measure of physical play may currently be impacting cultural qualities in yet unmeasured ways. In our judgment, many societies have become remote from the social-ecological needs of our hominid past, and in order to forestall declines in the interpersonal qualities of society, perhaps we need to establish more "*play sanctuaries*"—safe places for children to indulge themselves, prosocially, in playful activities that they themselves initiate.

This is neither a new nor novel idea. Long before scientists became aware of the functions of the brain and its genetic composition, Plato extolled the benefits of free childhood play in his treatise *The Laws* [VII, 794]:

> At the stage reached by the age of three, and after ages four, five, six, play will be necessary. These are games which nature herself suggests at that age; children readily invent these for themselves when left in one another's company. All children of the specified ages, that of three to six, should first be collected at the local *sanctuary*—all the children of each village being

> thus assembled at the same place. Further, the *nurses* are to have an eye to the decorum or indecorum of their behavior. [emphasis added]

Plato's basic message was that our children cannot become fully human without play. It is no different today. But today we do have a very active discussion about the role of play in promoting child welfare and mental health (e.g., Schaefer & Kaduson, 2006).

Abundant early PLAY opportunities may culturally and epigenetically benefit children's happy and empathic BrainMind development for a lifetime. It may also help mitigate the self-serving greed that has come to characterize so much of our business-as-usual economic environment. Social play may help open the gateways to better understanding of others and thereby to prosocial tendencies, ranging from heightened sociality to outright empathy. But these are just ideas, like many others, that remain to be empirically evaluated in rigorous ways, both by neuroscientists and clinicians. In Chapter 12 Panksepp will consider how PLAYful energies can promote rapid therapeutic changes through new affective-balance approaches in psychotherapeutic interactions.

CHAPTER 11

Toward a Neurobiology of the Soul

The Core SELF and the Genesis of Primary-Process Feelings

Go within again to that ancient source of knowing
To the depths of our creation beyond 4 billion years
To the mighty fires of Genesis that drew this Earth together
In the chaos and beginning of the ancients
Trace in quietude the unbroken thread of lives that made us who we are
Consider all those creatures gone before
We wear the face of every one that ever floated, wiggled, swam or ran
We walk the ancient paths that they began

—Sandy Hartman, "Go Again Within (the Biology of Spirit)" (2011)

THIS CHAPTER OFFERS A VISION of how the most troublesome issue in all of neuroscience might be addressed. As Robert Holt phrased it in 1989, "There is an impenetrable mystery in the fact that subjective experience exists in a physiochemical world." For us, this issue boils down to the question of how neural systems ever manage to produce subjective affective experiences. We have already provided evidence that primary-process feelings emerge substantially from the same subcortical circuits that engender coherent emotional actions. In other words, wherever one evokes coherent emotional actions by localized subcortical brain stimulation, those BrainMind states serve as "rewards" and "punishments" in the control of learning. A further understanding of the deep neural nature of affective experiences—a

fundamental form of phenomenal consciousness—may require an empirical clarification of the essential brain processes that some call "core-consciousness." Perhaps the primal nature of experience cannot be clarified without realistic "embodied" visions of what it means to have a "core SELF"—which, with a bit of poetic license, might even be referred to as our animalian "soul." Again, we capitalize our term for this brain function, because it is conceived to be a primary process of the mind—a "Simple Ego-type Life Form (SELF)"—a coherent center of gravity for internal organismic visceral-affective and external sensory-motor representations.

The problem of the human "soul"—reflecting our ineffable feeling that each of us is a unique "I"—has a torturous history. It has led to metaphysical dualism—the splitting of the mind from the body—along with endlessly varied ponderings on the nature of "the self" during the modern era (see Gallagher & Shear, 1999; Panksepp & Northoff, 2009). As Rene Descartes (1596–1650), who popularized dualism, put it: "I know that I exist; the question is, What is this 'I' that 'I' know?" His pondering eventually led to his personal "solution": "I think, therefore I am." Descartes and many other philosophers have recognized the importance of our personal memories, which are foundational for our thoughts, for our uniquely human conception of our*selves*. This aphorism has bred abundant variations. We prefer "I feel therefore I am" (Panksepp, 1998a, pp. 308, 420). This highlights the coherence of our foundational affective experiences.

Such alternative views of understanding the core SELF seek to recognize that raw affective forms of experience (primary-process or "core" consciousness)—characterized by basic feelings (the nonreflective, nonrational, affective ways of being in the world)—surely emerged on the face of the earth before higher forms of consciousness such as cognitive awareness. This has not been a mainstream idea in philosophy, but it is consistent with the views of some, for instance, David Hume (1711–1776), who grounded his philosophy on our capacity to have affective experiences, as in his *An Enquiry Concerning Human Understanding*. But Hume was also of the mind that our memories, like pearls on a string, pull together individual lives into a coherent "I'ness"—the self.

Immanuel Kant (1724–1804) admired the cogency of Hume's arguments, but in his *Critique of Pure Reason*, Kant continued to cultivate his "rationalist" argument that the mind has *a priori* cognitive powers that

provide intrinsic knowledge that precedes experience. Many other rationalist-cognitivist philosophers have found it difficult to bridge their visions of the higher conceptual mind to the ground of being that may be constituted by an affective core-SELF. Although this may not be an "either-or" matter, we will in this chapter explore the likelihood that an embodied core-SELF process—a primordial representation of the body, especially the visceral body, within the brain—may be foundational for affective "being" and the emergence of the higher mental apparatus. The core SELF is here envisioned as a label for those deep subcortical processes that engender organismic coherence—a unified presence of an active organism with a diversity of emotional feelings. (For an incisive historical-philosophical analysis of how such ideas can provide a universal substrate for nondenominational religious experiences, see Thandeka, 2005, 2009.)

The goal for this chapter is to explore the possibility that a neuroscientific understanding of the "embodied self"—a self that is grounded in the body and its neural representations—may clarify the dilemma of how experience first emerged in MindBrain evolution. This dilemma has no agreed-upon resolution yet and no unambiguous answer. But from a bottom-up, neuroevolutionary point of view, experimentally testable ideas are arising from affective neuroscience strategies to understand primary-process emotionality.

WHY DO WE NEED TO CONSIDER THE NEURAL NATURE OF "THE SELF"—THE ANIMALIAN "SOUL"?

How might raw affective experiences be created in the human brain? We have no final empirical answer to this question, but this whole book has made a case for the perspective that we do now know where to look—we should look among the ancestral, affect-generating instinctual mechanisms of the ancient medial subcortical networks of mammalian brains. If this is so, a detailed answer to the above question can only be obtained through causal brain research, work that is surely ethically impossible to do in human beings. Many would say this also applies to other animals. In any event, we have no effective strategy other than to study the corresponding processes in other animals. For this to work, animals must obviously have primary-process affective experiences. Otherwise, animal brain research on

such a topic would, by definition, be a fool's errand; we can be confident, based on the massive weight of evidence, that it is not.

This book has outlined evidence for ancient emotional feelings in other animals. Why the weight of evidence remains to be accepted by most neuroscientists is a cultural-historical issue, not a scientific one. One major historical reason is the fact that leaders in the field have long admonished students to recognize that since "subjective phenomena cannot be observed objectively in animals, it is idle to claim or deny their existence" (Tinbergen, 1951, p. 5). Indeed, agnostic and solipsistic world-views, not uncommon in the field of animal neuroscience, lead to the conclusion that the behavior of other animals, or even humans, can never provide definitive evidence that they experience anything.

However, a scientific approach to this problem is a matter of observable predictions and of the resulting convergence of evidence, rather than mere argumentation. The evidence is now overwhelming that all mammals have intense experiences when the ancient networks of their emotional brains are directly manipulated. Thus, brain networks that produce coherent emotional responses also generate feelings. But this still leaves a momentous scientific question, only gradually being discussed by neuroscientists. How does this "magic" transformation of brain activity to mental experiences actually happen? How does it come to pass that the material processes of the brain beget a mind, a "me"? No one knows the answer. At present, there can only be hypotheses . . . hopefully testable ones. Our goal for this chapter is to develop the possibility that a core-SELF concept will be invaluable for making empirical progress, although doubts will surely outweigh certainties for a long time to come. Still, abundant evidence indicates that affective feelings are very ancient in brain evolution, but we must now entertain new ideas about how they are actually constructed from neural activities. We can be reasonably confident that they arise from medial brain-stem regions (Panksepp, 1998a, 1998b), but neither the precise neural mechanisms nor the strategies to generate definitive understanding are crystal clear. The core SELF nevertheless seems clearly related to primary-process emotional and other affective processes of the BrainMind. Our main hope is that by placing some testable ideas on the table, we will inspire young scholars to undertake the research necessary to empirically clarify the underlying possibilities.

INTEGRATIONS BETWEEN COGNITIVE (HIGHER) AND AFFECTIVE (LOWER) FORMS OF CONSCIOUSNESS

We use the term "core SELF" with uppercase lettering for the same reasons that we did so to designate the seven basic emotional systems. Such brain systems are homologous across mammalian species and probably across other vertebrates as well. By providing a shared neural platform for diverse affective experiences, the core SELF can be considered to be a "*nomothetic*" (universal) brain function. As the core SELF, along with the many raw feelings it elaborates, interacts with higher cognitive tertiary processes, it promotes the emergence of various "*idiographic*" (individually unique, experientially refined) "extended" selves, during developmental brain maturation (Northoff & Panksepp, 2008; Panksepp & Northoff, 2009).

Of course, core-SELF structures vary in detail among different mammalian species. The dramatic variations in the bodies of different species would surely be reflected in natural variations in these foundational networks of mental existence across vertebrate species. However, since the brain is an evolutionarily *layered* organ, with the most ancient survival functions being most deeply conserved, we simply suggest that there are striking evolutionary similarities (homologies) that outweigh the differences. The affective evolutionary "tools for living" are quite similar in all mammals. In contrast to the universal (*nomothetic*) core SELF, the experience-dependent *idiographic* self is not homologous. This is because neocortical growth and the resulting cognitive capacities vary dramatically among species, leading to vast differences in reflective awareness. (We discuss such higher-order processes by using lowercase terms.) The idiographic self emerges during each life span through the unique experiential landscape of each person and each animal (Panksepp & Northoff, 2009).

In this chapter we share a working hypothesis concerning the primary-process nature of the SELF, based on a cross-species reading of the neuroscientific evidence. Converging evidence concerning primary-process affect generation in subcortical brain regions encourages us to speak about a nonreflective (anoetic) SELF and pure affective forms of consciousness. In this view, the core SELF and the various innate tools for living provided by the various primary-process emotional systems are the necessary ingredients for the concurrent generation of both organismic emotional-

behavioral coherence as well as the associated affective states. This view also proposes that the core SELF and the seven emotional systems interacting with higher brain functions, such as working memory, permit the emergence of higher levels of reflective "knowing" (*noetic* consciousness) as well as a multilayered existential self-awareness, which is a developmental, perhaps unique, quality of the human mind. The ineffable feeling of experiencing oneself as a specific and individual active agent amid the perceived events of the world surely reflects a recently emergent ability of the MindBrain, constituting a cognitive, even rational, form of consciousness.

As already noted, there is nothing unusual or unrealistic about postulating that a primordial form of consciousness is generated by subcortical structures. Indeed, during the middle of the last century, neuroscientists discovered that the *reticular formation*, a loosely knit conglomeration of cell bodies and neuronal fibers in the core brain stem, permits waking states in the cerebral cortex (Watt & Pincus, 2004). The neocortex cannot sustain consciousness on its own. Thus, the storehouse of our past memories, which can be drawn into active planning modes or "working memory" functions, is concentrated in dorsolateral frontal regions of the brain. Our evaluation of how all that relates to our personal concerns is concentrated in medial frontal regions (Northoff et al., 2006), and such deliberations are heavily influenced by subcortical primary-process emotional functions (Panksepp & Northoff, 2009). There may also be intermediate brain regions, such as the orbitofrontal cortex, that are devoted to affective working memory.

Psychologically, the core SELF is dominated by affective feelings that are accompanied by some rudimentary perceptions about the world and the internal homeostatic states of the body. Higher forms of self-consciousness are elaborated by the intermingling of these primary affective capacities with secondary/tertiary mental abilities that encode an animal's ecological, social, and cultural environments. The next chapter will draw attention to some possible links between the functioning of these subcortical affective systems and their effects on mental health or illness (and related psychotherapy issues).

NEUROEVOLUTIONARY PERSPECTIVES ON THE SELF: FROM EXPERIENCE TO AWARENESS

The issues we are considering here are so complex and difficult to understand that it may behoove us to consider some previously covered issues again, from a slightly different conceptual trajectory. Surely from a philosophical point of view, it is difficult to think about consciousness without postulating a self as the entity that contextualizes experiences. From a neurophysiological perspective, we must envision how the fundamental *coherence* of organisms—their internally felt unified presence in the world—is created by the ancient subcortical midline systems of the brain. What evolutionary reasons do we have to argue for the existence of a SELF within these ancient neural complexities? That is the question we will now explore.

It makes evolutionary sense to entertain the concept of a core SELF if one simply considers the fundamental difference between living and nonliving organisms. Living things carry out metabolic functions that keep them alive as discrete entities. For example, they utilize energy supplies and they eliminate waste products. Nonliving things do not perform metabolic functions. Nonliving things are coherent as units because of their chemical compositions. They have no clear and distinct biochemical processes to actively sustain themselves as individual entities. Thus, at a bodily level, *metabolism* distinguishes living from nonliving things. At a brain level, endogenously generated *motility* (spontaneous bodily activity) and, in higher animals, the capacity to predict future events distinguishes living from nonliving things. The fundamental capacity for metabolism and motility, although initially unconscious, provides an essential foundation for the emergence and evolution of consciousness.

We, along with other neuroscientific colleagues (Damasio, 2010), imagine that early in brain evolution, a primordial neural *map* of the body emerged in order to facilitate the overall coherence of many different functions, from action tendencies to the autonomic changes that accompany actions (Northoff & Panksepp, 2008; Panksepp, 1998b). We, along with Damasio (1999), call this body map a primitive "proto-self," which evolved with the emergence of primary-process emotional and motivational systems, into a more complex organ of mind, the core SELF. We postulate that this type of brain organization integrates primal experiences such as raw sensory, homeostatic, and emotional affects. However, this is not yet what is commonly meant by the term "awareness."

To make sense of core-consciousness we also need to envision how higher levels of mind emerged evolutionarily from the more fundamental forms. Thus, we must make a distinction between primitive phenomenal forms of consciousness, which provide the capacity for pure experience without yet having the capacity to reflect on the experience (namely to have self-conscious awareness, defined as the ability to envision oneself as an experiencing actor on the world stage). That is fairly complex stuff, much farther along than the mere capacity to *experience* oneself in the world.

We assume that raw phenomenal consciousness comes in two flavors. First, there is the ability to experience the various positive and negative affects—the diverse forms of "goodness" and "badness"—especially the raw emotional affects that have been the focus of this book. Second, there is the capacity to sense the world perceptually in experiential ways (i.e., as the "movie in the head"), which is the foundation for cognitive awareness. It is easy to claim that we do not really know which is more ancient in brain evolution, or how these types of phenomenal experiences are coupled. But if we had to make a choice, we would suggest that affective forms of subjective experience are older in MindBrain evolution than the cognitive forms, because they are elaborated in more medial and caudal (tailward), and hence more ancient, regions of the brain. An outstanding example of this is the location of the periaqueductal gray (PAG, also known as the "central gray"), at the very core of the midbrain. In contrast, the discrete sensory-perceptual functions are situated more laterally, which suggests a more recent origin.

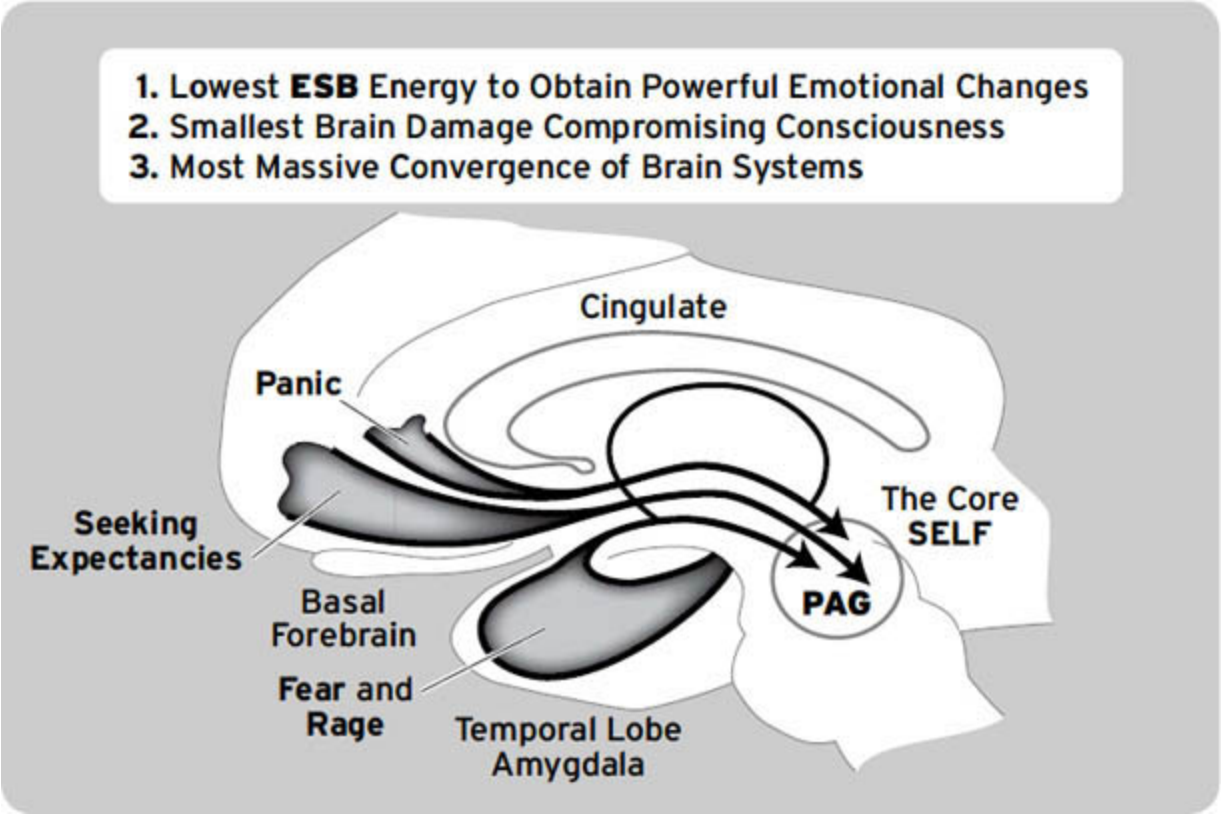

Figure 11.1. An overview of forebrain zones that are devoted to elaborating higher manifestations of basic emotional processes. Each of the emotional systems has higher spheres of influence, with FEAR and RAGE concentrated in the lateral and medial temporal lobes, SEEKING in the ventromedial frontal lobes, and various social emotional processes such as separation distress or PANIC/GRIEF in the anterior cingulate. All of these systems converge on the emotional and SELF representation zones of the midbrain. Three properties of the PAG are highlighted, which indicate its critical importance for core-emotional processing in the brain, compared to higher brain areas (from Panksepp, 1998a; republished with the permission of Oxford University Press).

Were primary affective and sensory-phenomenal experiences initially intimately linked during early BrainMind evolution, or were they two fundamentally distinct forms of primordial consciousness of the brain from the outset? We don't know. But new theoretical perspectives could be crafted from the supposition that the *experience* of conscious sight and sound were initially largely affective (Panksepp, 1998b). The immediacy

with which sudden visual or auditory stimuli can startle and frighten us, especially when such stimuli originate very close to our bodies, suggests a deep primal integration of these sensory systems with some of our most essential affective survival mechanisms. Consider also how we are prone to associate specific colors with feelings—red with passionate arousals, yellow with happiness, blue with cool or relaxed states, greens and browns with a secure love of the living land, black with death. Likewise, consider how easily sound arouses our emotional feelings, from the tone of someone's voice, and the songs of birds in nature, to the miracle of human-generated music (Malloch & Trevarthen, 2009; Panksepp & Trevarthen, 2009). The dynamics of music seem to have direct access to the affective structures of our core-consciousness (Blood & Zatorre, 2001).

In this context, it should be emphasized that the ancient affective processes concentrated in midline structures would not have been useful if they were not linked to sensory processes, which they surely were. However, these sensory inputs are not the ones that ascend toward the thalamus and then onward to the neocortex. They are ancient tributaries that directly enter midline reticular fields of SELF-related information processing, having linkages directly to emotional-affective processing. Again, this does not mean that the more recent streams of information to higher reaches of the brain cannot come to influence emotions. They surely can, but that mostly requires learning. Higher cognitive regulation of emotionality is not an intrinsically refined function of the brain; it emerges substantially through emotional education and the resulting social intelligence (Goleman, 2006; Keltner, 2009). This makes emotionally sensitive psychotherapy a useful aid in "growing up" (see Chapter 12). But there are also evolved dispositions for top-down, higher brain regulation of affective intensity. The neocortex can intrinsically inhibit primary-process emotionality, tending to keep it in the "subconscious" realm until needed to deal with major life-challenging situations. In psychopathologies such regulatory controls often fail, however, and individuals are flooded with affects that they can no longer control. Much of psychotherapy consists of promoting the cognitive regulation of one's emotional feelings.

In any event, it is quite clear that the primary-process mechanisms for emotional affects are situated in deeper midline regions of the brain, which suggests that emotional actions and the accompanying affects had some kind of priority in BrainMind evolution—that they evolved before

sophisticated sensory-perceptual neocortical capacities, like our ability for clear and refined seeing and hearing. However, subcortical structures can process sensory information in complex ways, so there is also some perceptual-phenomenal experience below the neocortex (see Chapter 6). For example, the superior colliculus can perceive visual information and the inferior colliculus can perceive auditory information at some level of psychological resolution (Merker, 2007). There are similar subcortical abilities to appreciate feelings of touch. These subcortical structures do not generate sharp conscious experiences of seeing, hearing, and body surface stimulation, but they surely give a sense of things in distinct ways. For example, these subcortical systems can ascertain where visual and auditory stimuli are in space, but more effectively at an experienced level during early childhood than in adulthood.

Obviously, the *cortical* capacities that we experience as the five major senses, usually with remarkable perceptual clarity, evolved much later than subcortical perceptual sensitivities. Thus, as individuals mature, the higher brain functions "take over" so markedly that most investigators believe the subcortical systems were *always* unconscious. (This explains classic neurological phenomena, such as "blindsight"—i.e., seeing without awareness—which is almost unconscious, but not quite so since people can correctly locate moving objects in space without having any clear visual perception of what they are seeing; they just have vague feelings that something, like pure movement, is happening at a specific location in space.) It is as likely that subcortical perceptual functions become subconscious developmentally, as higher experiential mechanisms gradually prevail in behavioral control and in their demands on conscious attentional resources (for a full discussion, see Merker, 2007).

It is especially germane to recognize that subcortical perceptual processing continues to interact with the affective networks of the core SELF. This helps explain the "low roads" to emotional arousals that we focused on in Chapters 5 and 6. Also, it may be worth considering that, in Mind-Brain evolution, organisms may not have needed refined perceptions before they had neural systems that could represent vital organismic survival needs, namely the primary process affects. Viewed in this way, we can understand that while struggling and competing for survival, across innumerable generations, there were great advantages for creatures with primordial affective systems to be guided ever more precisely by

sophisticated perceptual and cognitive-learning mechanisms. During early phases of MindBrain evolution, fairly simple sensory connections between the affective core-SELF networks and the external world may have sufficed (e.g., the "low road" of fear conditioning). However, the utility of evermore sophisticated distance receptors such as those for cortical hearing and vision, and the capacity to strategize with that information, paid off handsomely in later BrainMind evolution. All this leads us to appreciate why some kind of neurosymbolic matrix of the primordial body, reflecting a spontaneously active, affectively responsive organism, was laid down earlier in brain evolution than the emergence of the sophisticated distance receptors and their neocortical analyzers. This also allows us to appreciate why primary affective and higher cognitive mechanisms need to be distinguished.

To this day, the most ancient sensory systems such as olfaction, taste, and touch still have more affective immediacy for human beings than sight or sound. Perhaps primordial perceptual acuity was initially closely linked to such information channels that optimally served affective bodily and brain needs. Homeostatic affects remain especially intimately intertwined with the affective processes that represent bodily states. Thus, feelings of hunger still whet the appetite, and they also amplify the pleasures of taste and smell. We still attribute affective aspects to our perception of colors—from the passionate, stirring reds and joyous yellows, to the cool, relaxing blues, and soothing browns and greens. From an evolutionary perspective, therefore, we might be wise to leave open the possibility that affects may have guided the construction of many sensory-perceptual abilities (Panksepp, 1998b). If this scenario is on the right track, we will never fully understand higher forms of consciousness without first deciphering the more primal affective forms.

This may again help highlight why we call this poorly understood, barely studied, neural foundation for affective self-representation the core SELF. It took care of immediate bodily concerns—engendering (i) SEEKING, first to take care of homeostatic needs such as water, energy, and thermal balances (Denton, 2006), and then more subtle emotional needs; (ii) RAGE and FEAR to avoid bodily destruction and to compete effectively for many resources that are essential for (iii) primal LUST, which promotes species survival. These reptilian emotions were gradually supplemented with more subtle social principles. The next phase of mind evolution, presumably in

species existing before the divergence of birds and mammals, added the uniquely social-emotional systems of CARE, GRIEF, and PLAY, all built upon the preexisting reptilian emotions, especially SEEKING.

Let us put all of this in yet another (slightly different) way: The core SELF is both poorly understood and barely discussed in neuroscientific circles. We see it, however, as the neural foundation for the creation of all affective experience. It has the right ingredients, especially a vast field of neurosymbolic representations of bodily organs, from heart to guts, which are components of emotional experiences. What is affect, if it is not experienced by a subjective "me"? It first represented various homeostatic states as affective states of the MindBrain, experienced by changes transpiring in the neural representations of the body within the brain (neural networks that remained, of course, interlinked with what was actually happening in the peripheral body). For example, low body water levels and high blood solute levels are experienced as thirst, a rapid decline in blood sugar arouses hunger, and so on. The most important sensory experiences like the odor of foods, and various types of touch, are experienced as affectively pleasant or unpleasant. Often these associated affects may be learned, highlighting how we develop learned preferences and dislikes.

With the emergence of complex emotional networks, the core SELF could also anticipate a variety of environmental changes. For example, the smell of a predator would arouse the FEAR system, and the core SELF was shifted into a distinct neurodynamic that is still experienced as fearfulness. Similarly, the smell of a mother's breasts, especially when hunger signals are high, aroused infants' SEEKING urges, and they would approach and nuzzle closer to their mothers' bodies to feed and to concurrently be emotionally nourished through maternal CARE. From an evolutionary perspective it is noteworthy that the homeostatic, bodily need systems are concentrated in the midline of the hypothalamus, in more evolutionarily ancient positions than the slightly more laterally situated SEEKING system, which, as we have repeatedly emphasized, is the biggest and most pervasive emotional system of them all. The LUST and CARE systems are in more anterior positions than the bodily need systems, again clearly indicating a slightly later evolutionary origin.

This is an important point. All emotional systems do not have the same status in BrainMind evolution. One can assume that among the primitive "reptilian" emotions, the general-purpose SEEKING system was designed

quite early for acquiring a large number of resources, including safety from danger (e.g., flight). Thus, the SEEKING system may have served as the platform (the preadaptation) for the evolutionary emergence of the other social-emotional systems we have discussed—LUST, CARE, PLAY—while also facilitating the ancient FEAR and RAGE systems.[1]

This is one reason that SEEKING was the first emotional system to be discussed here. It is also the system that has the most evidence for mediating a very special kind of positive affect—not the pleasure of sensation, but the positive invigoration, euphoric excitement, of engaging productively with the world. The seemingly boundless enthusiasms of little children surely arise, in part, from this system, encouraging PLAY. As emphasized in Chapter 3, the SEEKING urge is not just "the reward system of the brain" but one that aspires for organismic well-being. The arousal of the SEEKING system is most certainly rewarding, but the rewards are not the typical pleasures of sensation but the eagerness to pursue all kinds of rewards, an idea that has also been emphasized in a more sensory-centric way ("incentive-salience"—a conditioned secondary process; see Berridge et al., 2009).

The core SELF establishes an explicit framework to account for the existence of affective consciousness and to escape from the infinite regress of "read-out" mechanisms (see Chapter 2). The raw, basic affects, in order to be experienced, do not need to be "read out" by higher MindBrain mechanisms, as so many contemporary emotion theories maintain. Of course, higher cortical functions may add other types of feeling, especially by allowing raw feelings to penetrate and intermingle with cognitions—higher brain functions may "listen" to the lower ones and add additional cognition-parsed affective coloring to experience. In this way, a variety of more subtle, higher-order feelings may be created by secondary and tertiary psycho-affective processes—such as courage, envy, guilt, jealousy, pride, shame, and social disgust/disdain, to name just a few (for a full discussion of jealousy, see Hart & Legerstee, 2010). The coherence of the core SELF may allow people and animals to have a fundamental sense of *owning* their affective experiences: The affects are an integral part of who they are, psychologically.

From a historical perspective, it is important to note that these are the BrainMind substrates that the behaviorists, through their doctrinaire avoidance of the psychological dimensions of brain activities, decided to

call the "rewards" and "punishments" that "reinforced" behavioral change. As a result of these conceptual choices, rewards and punishments could be defined as observable events in the world outside the animal's skin. Because scientists could use operational definitions focusing on objects in the world, as opposed to internal processes, discussions of affective experiences in animals were treated as moot. All the necessary concepts (except "reinforcement," which constituted some kind of "glue" in the system) were conveniently defined in visually evident environmental and learning terms, with no need to indulge in ambiguous neuropsychological concepts. "Rewards" and "punishments" were simply events in the world, and that was that. However, neuroscience soon opened up the possibility that they were, in fact, neural functions, or experienced processes of the brain. But such ideas were discouraged and even actively suppressed. When scientific conversations cease, then dogma rather than knowledge begins to rule the day. Our understanding of the true nature of MindBrain processes is proportionally diminished.

Current evidence indicates that at their most primitive level, raw affective experiences emanate from subcortical midline systems (SCMS) that are located in the upper brain stem (midbrain, hypothalamus, and thalamus) and that connect heavily with more rostral medial cingulate, insular and frontal, and orbitofrontal cortical zones. Our ability to contextualize universal, primary-process feelings within our unique idiographic selves requires higher cortical midline structures (CMS), as well as many other higher brain tissues (Northoff et al., 2006; Northoff & Panksepp, 2008; Panksepp & Northoff, 2009). This idiographic contextualization of experience, however, could not happen without the organismic coherence emerging from the lower reaches of this neural continuum, the SCMS that are especially rich in visceral body representations, here envisioned as the epicenter of the core SELF.

ANATOMY OF THE CORE SELF

Although we cannot yet be certain about the precise anatomical constituents of the core SELF, any viable brain candidate should fulfill the following criteria: (i) The infrastructure should be ancient, located in the ancestral medial regions of the brain; (ii) the key systems should be multimodal and capable of being represented at many levels of the neuroaxis; (iii) the

circuitry should have a characteristic, innate resting state that indicates a kind of set point for deviations from homeostasis; and (iv) the shared infrastructure for selfhood should become aroused, in distinct ways, during primary-process affective states. In sum, the SELF structures should be very richly connected to the rest of the brain.

The primary-process emotional networks of the SCMS/CMS continuum abundantly fulfill these criteria. These ancient deep midline systems "valuate" the states of the body and the world, and they engender emotional responses to prototypical life-challenging events. The raw feelings of the SCMS are then re-represented within CMS, and ultimately within related affective memory fields, as idiographic renditions of the self. Many investigators have now observed characteristic resting neural activity in these midline systems that are more intense when people are doing nothing (self-reflecting and/or ruminating) during brain-scanning sessions rather than when they are being bombarded by various cognitive tasks (Damoiseaux, et al., 2006; Fox and Raichle, 2007; Fransson, 2006; Raichle et al., 2001; Rilling et al., 2007). These systems are also abnormally active in individuals who are depressed and are ruminating about their lot in life (Alcaro et al., 2010; Grimm et al., 2008, 2009; Northoff et al., 2010). At least half a dozen such subcortical midline structures are particularly richly connected to other parts of the brain. These include, from the bottom upward, much of the (i) deep nuclei in the cerebellum and surrounding floor of the fourth ventricle (the parabrachial area and dorsal motor nucleus of the vagus); (ii) the PAG and immediately adjacent midbrain regions; (iii) the superior and inferior colliculi, especially the deeper motor components; (iv) the ventral tegmental area (VTA); (v) the hypothalamus; and (vi) a series of basal ganglia nuclei, most prominently the amygdala and nucleus accumbens. Which of these portions is the most important for core consciousness?

The removal of the whole cerebellum does not severely compromise consciousness. For this reason, we obviously rule it out as a substrate of the core SELF, even though it most certainly modulates and controls emotional behaviors. Large cerebellar lesions do dramatically compromise all kinds of muscular coordination, needed, of course, for all actions, especially complex emotional responses. Indeed this may be the reason why deep cerebellar systems routinely "light up" during human brain imaging of emotional arousals: All emotional actions require the complex coordination

of many body parts. Damage to the floor of the fourth ventricle is likely to kill animals, because so many autonomic functions are injured. At the top, all of the various basal ganglia nuclei can be destroyed, yielding many behavioral deficits, without impairing core-consciousness; they simply impair basic learning mechanisms, such as classical conditioning. This means that the deep motor layers of the superior colliculi and the PAG, along with the associated VTA and hypothalamic circuits, may be the most important structures that support the core SELF. We have already noted that the PAG is the most ancient, and most highly concentrated, emotional convergence zone within the brain; that is why we will focus on it here (for an excellent summary, see Watt, 2000). The PAG is much more important for the generation of raw emotional feelings than the amygdala, even though the amygdala, because it so routinely "lights up" during diverse emotional arousals (usually cognitive-affective tasks), continues to be excessively marketed in the popular press as the brain's most important emotional center. In fact, it is a cognition-emotion interface, much more so than being a generator of primary-process affective experiences. There are many other such interface nuclei between the cognitive aspects of the mind, which specialize in external information processing, and the affective mind functions, which inform us about the states of our brains and bodies.

The relationship of the PAG to other brain regions also suggests some central role for it in overall emotional life. The deep layers of the superior colliculi constitute a basic motor mapping system of the body that interacts with a series of sensory systems (touch, hearing, and vision) as one moves outward in the midbrain, and also with a host of emotional systems of the PAG as one moves inward to the midbrain core. Adjacent to the PAG is the mesencephalic locomotor region (MLR), which is capable of instigating neural patterns that would be essential for setting up various coherent emotional action tendencies, such as running toward or away from world events. Because emotional expression consists of such affective action tendencies, the MLR may also be a part of the core SELF. The VTA is a viable core-SELF candidate because dopaminergic fields of the VTA and other nearby regions, with their rich projections into the medial subcortical and cortical forebrain regions, elaborate the instinctual and learned components of SEEKING urges that promote self-related information processing throughout the higher midline regions of the brain.

Finally, although we have not emphasized it, the ascending reticular activating system (ARAS), which sustains waking activity in the cortex, lies just adjacent to the PAG and MLR. This system is constituted heavily of ascending acetylcholine, histamine, norepinephrine, and serotonin systems —all of which modulate attention and wakeful arousal (Pfaff, 2006). It interacts with the thalamic reticular nuclei to facilitate the processing of sensory stimuli throughout the higher perceptual regions of the brain—the neocortical areas that control vision, hearing, touch and all the interactions of these senses that allow us to resymbolize the world in language and thoughts. The ARAS was the brain system historically first implicated in the regulation of waking, and hence it is a major player in the control of consciousness, especially cognitive consciousness. It is not yet clear how these systems participate in specific forms of emotional arousal, but they surely have a role in the general arousal of many brain regions, especially neocortical areas during emotional states. For instance, brain norepinephrine facilitates incoming sensory signal processing so that signals are more intensely perceived (that is, they have more salience in the BrainMind). This system probably also increases the intensity of affective salience that may be largely experienced as general emotional arousal. In contrast, the more medial regions of the PAG appear to elaborate more specific emotional behaviors and the associated distinct feelings.

The complexities of these ancient midline circuits are being actively explored (Holstege & Saper, 2005). It will be easier to understand the functional organization of the descending "outputs" of the integrative systems within the PAG, including inputs into the ARAS, by monitoring specific motor nuclei of the brain stem for the observable actions of emotions and by monitoring the autonomic nuclei for the visceral components of emotions. The important point is that these emotional outputs are under coherent "orchestral" control from the PAG, which is the epicenter for emotional arousals but also critically important for the instantiation of the core SELF.

THE CORE SELF AND THE MECHANISMS OF AFFECTIVE CONSCIOUSNESS

The kind of consciousness experienced by the core SELF is fundamentally affective, without any propositional content. In our view, each emotional

system can generate a different global neural dynamic within the subcortical midline structures—within the core SELF—leading to the distinct primary-process emotions that humans and other mammals experience. To try to describe such brain mechanisms in word images is difficult, and one can only imagine the characteristic dynamics of each emotion. We suspect that sustained contemplation of these matters, across days or weeks, may facilitate comprehension. Also re-reading the same idea in different forms should facilitate integration (Austin, 1998; Panksepp, 1998b, 2009b).

We envision the following type of process. The arousal of each of the seven basic emotional systems may result in characteristic large-scale patterns of neural firing for each emotion; these patterns are characteristic oscillations that constitute neuromental signatures of the primary-process affects that dynamically resemble the emotional actions that are concurrently released (one dilemma for understanding, perhaps most especially among the smartest, is that humans can inhibit these expressive lower brain functions through high cortical willpower). In any event, the various primary-process affective dynamics may reflect the rates and patterns of neural firings within core-SELF structures, resulting in the distinct patterning of global oscillations within ancient viscero-somatic body maps of the brain. The evidence suggests that it is within these neural oscillations that the different affective arousals emerge, yielding a large variety of positive ("rewarding") and negative ("punishing") states of the nervous system. In other words, the neurodynamics of emotional affects (the large-scale neuronal activities) resemble the various primary-process instinctual emotional behaviors, which are the pounding force of RAGE, the frozen uptightness of FEAR, the caress of CARE, and so on. Thus, the biophysical similarity between affective network-firing dynamics in the core SELF and instinctual emotional actions allows us to study subjective emotional feeling in animals through a study of their objective, instinctual emotional expressions.

Such distinct global dynamics of brain emotional arousals have yet to be measured objectively *within* the brain. Indeed, brain scientists currently have no effective ways to study global neural network dynamics. This makes a psychoanalytic study of the experience of emotional dynamics in humans especially important. However, there are suggestive hints in the traditional neuroscience literature for certain types of relevant synchronous oscillation within the brain, such as the 4–7-Hz rhythms in the hippocampus

known as the *theta rhythm*, which helps animals to investigate the world (e.g., sniffing in rats) and thereby create memories in the hippocampus. The theta rhythm is the highly characteristic neural signature of the hippocampus as it is actively processing information. This rhythm is especially evident during artificial arousal of the SEEKING system in rats, a premier information-gathering emotional system, as animals sniff and investigate their surroundings (Vertes & Kocsis, 1997). In other words, the sniffing rhythm typically corresponds to the ongoing frequency of the hippocampal theta. It is important to recall (see Chapter 3) that this sniffing rhythm spontaneously conditions in an anticipatory way just by electrically activating the SEEKING system on a fixed-interval schedule (e.g., a pulse of rewarding brain stimulation given every 20 seconds). This may highlight how cognitive knowledge emerges from the patterned arousals of affective processes, perhaps allowing us to understand why Kant inferred that the brain has intrinsic knowledge. Such data support the view that emotional systems help create knowledge in higher regions of the brain. Likewise, as we discussed in Chapters 5 and 6, the unconditioned emotional response of FEAR may be critical for the generation of simple fear-conditioning in higher regions of the brain.

Thus, the quality of each affect may arise from the ways in which the signature oscillation of each emotional system promotes dynamic changes within the global neural space of the core SELF, in which all of the primary-process emotional networks are embedded. Arousal of the FEAR system, for example, might generate rapid oscillations that push the core SELF into an "uptight" shivery state of tension. RAGE may push global neurodynamics into invigorated forceful cycles that strike out at the world, and so on. The core SELF is massively connected to many other parts of the brain—to various sensory triggers and regulatory feedbacks, to motor functions, autonomic integrative responses, as well as to many higher cognitive processes, especially of cortical midline systems.

Here, concepts of nonlinear dynamics such as the "attractor landscapes" of chaos theory become important (Lewis, 2005). For instance, in humans the intense whole-body motor patterns of primary-process emotional responses, such as laugher and crying, can effectively promote affective changes of joy and sadness (Panksepp & Gordon, 2003). Also, because there are bound to be abundant evolutionary variations in the details of emotional substrates in different species, we can also begin to envision

variations in the feelings of different species depending on the variations and complexities of the large-scale instinctual attractor landscapes that are elaborated in the mindscapes of the brain.

To reiterate, we hypothesize that when an emotional system within the core SELF is aroused in a certain way, that emotional network and the core SELF are involved in a shared function, namely in the creation of both primal affects and adaptive behaviors. We propose that when the core SELF assumes the neural signature of the aroused emotional system (yet to be measured, but attempts have been made: Panksepp, 2000), the core SELF becomes the shared neural substrate of emotional instinctual behaviors and of affective consciousness across the different primary-process emotions. Furthermore, because the subcortical, medially situated core-SELF networks are intimately connected to *cortical* midline structures, the neural signature of each emotional system, when aroused, may reverberate upward throughout many cognitive areas of the brain, allowing the creation of various higher-order tertiary nomothetic (and in humans, also highly idiographic) emotional processes that are especially important in human affairs (producing feelings like shame, guilt, jealousy, compassion, empathy, and so on.). These higher-order socially derived feelings may reflect developmentally elaborated global brain dynamics, still strongly tethered to the basic emotions; it is unlikely they are just variations of "information processing" within working-memory fields of the neocortex, as many contemporary investigators seem to believe (see Chapter 2).

Again, we must emphasize that this is only a hypothetical description that allows us to envision the activity of global brain systems that remain to be studied in sufficient detail for us to understand how the underlying neural machinery actually works. Indeed, as noted, neuroscience currently has few tools for studying large-scale network functions. At present, without direct brain manipulations (e.g., localized brain stimulation) that can yield evaluative "reward" and "punishment" effects in animals, the emission of instinctual emotional patterns remains the best way to evoke and monitor emotional-affective processes in animal brains. This assertion is supported by abundant data, but it is not yet widely recognized in emotion studies (or psychology in general). And animals are commonly not granted any emotional feelings, just emotional behaviors, because supposedly higher brain regions somehow construct the intense feelings that make emotions so powerful and interesting for humans.

In any event, according to the weight of existing evidence, the primal affects are an instinctual/automatic rather than a cognitive/voluntary process. Healthy rats, for example, cannot help being afraid of cat scent, albeit there is abundant variability in this emotional response across animals. Furthermore, quite rapidly, the rats automatically learn to make associations to the cat scent, and through conditioning, they become fearful in the presence of these associated perceptions as well. As we have noted earlier, the rat will learn to fear the sound of the cat bell, the sight of the animal, its meow, the environments in which the cat smell is consistently present, and so on. These are all involuntary secondary-process reactions, based on automatic learning mechanisms of the brain. Furthermore, intelligent species like our own can use their prodigious higher brain regions to dampen or enhance the affective responses. For example, a frightened person might cheer himself up by imagining a happy scene. However, this will probably not eliminate the fearful affect. And if the primary-process emotional response, FEAR, is sufficiently strong, it will win the battle. It would be virtually impossible to feel calm in the hours following learning of the death of a child, for instance, regardless of attempts to think of happier times.

NEUROECOLOGICAL PERSPECTIVES ON AFFECTS AND THE SELF

We propose that as animals evolved and adapted to their various ecological conditions, they needed automatic gauges, more sophisticated than sensory affects and those arising from bodily homeostatic imbalances, to guide their survival predictions. Although lower animals might obtain nourishment simply by filtering their aqueous environments, evolution soon crafted animals that needed SEEKING systems to impel them to actively search for food and other resources. Other emotions emerged, as evolution—the effective sieve in the struggle for survival—promoted additional survival strategies for increasingly complex animals. Female reptiles, for instance, exhibit little in the way of maternal CARE. They lay hundreds of eggs (food for many other animals, perhaps even animals of the same species), and even though most of the offspring do not survive, the sheer number of hatchlings tends to ensure the survival of the species. One reason that salmon may die soon after spawning is that their voracious appetite may be

deleterious to the survival of the young, and hence the species; indeed, without a CARE instinct, they are liable to devour too many of their own small fry. However, when mammals evolved, their bodies produced far fewer offspring, and these offspring required nurturing care in order to survive. If mammalian brains had not evolved a CARE system that induced them to nurture their young, each species would have died out.

We have noted, however, that this CARE system can at times turn an indifferent eye to what we would call "cruelty." Spotted hyenas and black eagles typically give birth to more offspring than they can effectively rear. The brothers and sisters are born intolerant of each other, and they commonly fight until one dies or falls from the nest. The parents rarely intervene. This is not just survival of the fittest; it is as much the luck of the draw. Among the eagles, it is the first-hatched nestling that almost invariably "wins" the tournament. It is presumably no more physiologically "fit" than the second-born—it just has a maturational advantage—being a bit fatter and motorically more mature. Why do these creatures manufacture a "spare"—an expendable baby? Presumably because the probability is high that one would die anyway. The spare is simply "cheap insurance" if the firstborn dies. Of course this is just a theory, because science can rarely answer "why" questions in compelling and rigorous ways. It is much more convincing when it addresses "how" questions.

Still, the reason that affects emerged was probably because they were very effective in predicting and often protecting organisms against adverse future events. As mentioned in Chapter 2, hunger does not signal that one's body energy is dangerously low; it simply highlights that it is evolutionarily wise to "top up" on energy long before stores drop significantly. In this way, homeostatic affects (like hunger) anticipate the future, and they provide motivation above and beyond immediate metabolic considerations. Emotional affects provide a similar anticipatory function. For example, we become afraid in the face of possible danger and take precautionary measures before we are hurt or killed. The reason that emotional affects took the form of phenomenally experienced states of mind—valenced subjective states—is presumably because this was an effective way to motivate and guide animals in relatively precise ways in unpredictable environments. Affects proved ideal for guiding ("reinforcing" and "punishing"?) behavior, thus facilitating learning. We tend to repeat those

behaviors that are followed by affective delights, and we avoid behaviors that are followed by affective discomfort.

We propose that as the seven basic emotional systems evolved, the unconscious proto-self also evolved into a rudimentary core SELF that was capable of a primordial form of conscious experience. By "primordial" we do not mean that it was experientially minimal. In fact, we think these feelings were "enormous" in their psychological salience and later probably diminished with the development of the cortical-cognitive abilities that allow precise behavioral regulation, in part by inhibiting lower brain functions. Cognitive decision making, although typically guided by affects (Damasio, 1999), is also disrupted if affects are too intense. In other words, we think that it is the core SELF, interwoven with various basic emotional and motivational systems, that generated robustly experienced affects, which continued to provide critically important survival information as additional brain complexities—most especially the neocortex—evolved, allowing for the emergence of higher cognitive functions. As noted above, perhaps the most compelling evidence in this regard is that stimulation of these ancient neural substrates still generates intense affective experiences in humans (Panksepp, 1985). Extensive damage to brain regions where all emotional systems converge, such as the PAG of the midbrain, compromises all forms of human consciousness (Schiff, 2007).

We are inclined to this view also for the reason that recent brain neuroimaging experiments show that when people engage in activities that focus on themselves, in *self-related processing* of diverse forms of information, the midline brain systems are aroused (Northoff et al., 2006). Indeed, midline brain systems involved in self-related information processing form a continuum from the medial brain stem to the medial frontal and cingulate cortex, within the more ancient regions of the cerebral mantle. The subcortical portions of this midline continuum, the SCMS, are remarkably homologous across all mammalian species, and they are also found in the brains of other vertebrates, including reptiles and birds (Northoff & Panksepp, 2008). The cortical parts of the midline system, the CMS, include some of the more affectively relevant regions of the neocortex, including insular, medial frontal, and orbitofrontal cortex. Cortical midline systems are not as unambiguously homologous across species as the subcortical ones, because the size and complexity of the neocortex differs so radically between species. This again suggests that the

subcortical reaches of this self-related processing continuum constitute a nomothetic affective core SELF, and the higher reaches contribute to increasingly idiographic cognitive selves.

FUNCTIONAL EVIDENCE FOR A CORE SELF

Although it is useful to distinguish between SCMS and CMS in terms of homology, they are functionally connected, and the CMS depend on the SCMS for their integrity and existence. Lesions to these brain regions can dramatically compromise consciousness, and the deficits get more severe the lower one inflicts damage in this neural continuum for self-related processing (Merker, 2007; Panksepp & Trevarthen, 2008; Watt & Pincus, 2004). For example, the complete destruction of the PAG, which lies at the heart of SCMS, results in the destruction of all self-related processing of environmental events. With total damage to the PAG, all world-directed activities are compromised. Animals are marginally awake, but they do not appear to be conscious of things in any meaningful way (Bailey & Davis, 1942, 1943). By comparison, damage to higher cortical midline systems does not inflict the same degree of impairment in consciousness (Watt & Pincus, 2004).

There is also a gradient of severity when damage extends from medial to lateral sectors of the brain. If someone has suffered lateral brain damage resulting in the loss of sight, hearing, or the ability to use language, the person still experiences himself as the same human being he has always been. His fundamental sense of self and affective experience is not impaired, even though he has lost some precious cognitive abilities. In contrast, damage to medial sectors of the brain, especially the frontal regions, can more severely compromise self-experience (Northoff, 2004; Northoff & Bermpohl, 2004).

There are also relevant findings from patients who have undergone “split-brain” surgery. This is a procedure used mainly to treat severe epilepsy, whereby the corpus callosum is severed. The corpus callosum is a massive set of neural axon fibers that provide most of the communication between the two halves of the neocortex. It is essentially the conduit that contains the vast majority of the wiring between the two halves of the higher brain. Each half of the neocortex processes different types of information. The left half is generally more verbal, social, and happy while the right half is less

verbal and more emotional and unhappy (Davidson et al., 2003; Tucker & Williamson, 1984). This has abundant implications for the way we humans have structured societies across the ages (McGilchrist, 2009). The two hemispheres function differently, but usually they are in some type of coordination. One would expect that after split-brain surgery a lack of communication between the two might lead to incoherent functioning. However, nothing of the sort occurs. For instance, when a split-brain individual dives into a swimming pool, there are no behavioral signs of incoherence, such as one side of the body flailing, suggesting that half of the brain has been taken by surprise.

There are, though, various experimental situations, and occasional situations in real life, where those separated hemispheres behave at odds with each other. The right hand that is controlled by the verbal left hemisphere may pick up a paper, while the left hand, controlled by the right hemisphere, folds the paper up and puts it away. It is well accepted that the right hemisphere is generally more emotional (feeling complex issues), while the speaking left hemisphere is more cognitively propositional (pontificating on complex issues). At a cognitive level, the two hemispheres may have different goals and intentions. However, the general picture of split-brain patients suggests that they continue to exhibit coherent emotional, motor and global, whole body, intentional behavior, indicating that there is a coherence of self and consciousness, which must therefore be rooted in the subcortical systems that are still connected to each other in the normal way (Panksepp, 1998b).

Other relevant observations are evident in the behaviors of animals that have been decorticated early in life: As we have emphasized repeatedly, they sustain a remarkably strong level of behavioral coherence and spontaneity. They even retain the ability to compete effectively with normal animals in rough-and-tumble play. Thus, it appears that subcortical structures, which obviously evolved first, provide bedrock neural structures for a foundation, on which more variable cognitive renditions of the self, generated by developmentally emergent higher brain functions depend. Because the subcortical midline functions emerged so much earlier in the ancestral cauldron of brain evolution, we can be confident that those structures have priority in sustaining the coherence of the mind. This intrinsic coherence demands some kind of neural explanation, and the core-

SELF concept merely highlights where and how we might seek a solution to this unsolved problem.

HIGHER BRAIN REGIONS AND AFFECTIVE STATES

We acknowledge that our hypothesis is controversial. Many cognitive and behavioral neuroscientists might not accept that subcortical midline regions are more important in generating affect than the higher cortical regions that help mediate human thoughts and other cognitions (e.g., such as Damasio in *Descartes' Error* but perhaps not in his more recent works: Damasio et al., 2000; Damasio, 2010). LeDoux, (e.g., as he first enunciated in 1996) still seems to oppose the view that "lower" animals experience their emotions as affects. Indeed, most discussions of human consciousness tend to focus on the more recent evolutionary endowments of the BrainMind as being most critical for the genesis of consciousness. However, let us reiterate once more why we must conclude (as also emphasized by others) that the lower reaches of the brain are, in fact, more critical for affective states (Denton, 2006), as well as for primitive perceptual states (Merker, 2007), than the higher regions.

In essence, three robust lines of evidence point to the critical importance of deep subcortical regions for affect generation. First, when one electrically stimulates specific medial regions of the brain and evokes affective states associated with most of the primary-process emotions, one always needs lower current levels when stimulating the lowest reaches of the network. For example, all emotional systems converge on the PAG, and one can obtain the most robust emotional response from this structure with the smallest current levels. In other words, the circuitry is simply more sensitive or more concentrated in lower than in higher brain regions. Second, when one electrically stimulates localized regions of the hypothalamus and gets affective states associated with RAGE and SEEKING, for instance, it is typically the case that *both the behaviors and affective states are diminished more by lesions in the lower reaches of these circuits than in higher projection areas* (Valenstein, 1966), although higher basal ganglia, like the extended amygdala, are important in communicating reward values to yet higher brain regions (Waraczynski, 2006). Third, recent evidence from brain imaging indicates that *subcortical signals of neuronal arousal are positively related to the degree of experienced affect,*

while higher brain regions are more typically negatively correlated with cognitive emotional experiences (Liotti & Panksepp, 2004a; Northoff et al., 2009). This strongly suggests that the lower regions actively generate primal affective states while the higher regions may be regulating, reprocessing and dampening them. Likewise, such interactions help explain how lower brain arousals may disrupt cognitive processing.

Most of the relevant causal evidence for a subcortical locus of control for primary-process affects comes from animal research. How well does this apply to humans? Certainly, brain-stimulation studies in humans are quite consistent with our thesis (Heath, 1996). An especially neat piece of correlative evidence comes from human-brain imaging in a fearful predator-rich simulated environment (Mobbs et al., 2007): The higher FEAR regions (e.g., the amygdala) are aroused when a predator is at a distance, while the lower regions (the PAG) become more aroused when the predator is about to bite. (An electric shock to the finger served to simulate the predator.) In other words, while you are still relatively safe—only mildly anxious as the predator is stalking from a distance—only higher FEAR circuits are clearly engaged. After all, you are merely *thinking* about the predator, and you still have a good chance of escaping. However, as the electronic "predator" closes in, the PAG exhibits ever more arousal. And it is known from causal brain-stimulation studies that it is the dorsal PAG that is capable of elaborating the most intensely fearful feeling of which mind-flesh is capable. Meanwhile, it is clear that this brain region is not as important for the generation of positively affective states of mind.

Thus, if we were to ask one deeply neuroexistential question from a knowledgeable brain scientist, it would be this: In your natural desire to avoid intense negative emotions, which brain area is the one you would never allow to be artificially activated with localized brain stimulation? If our neuroscientific colleague knew the cross-species evidence in this field, along with enough human data, he or she would surely never select regions of the neocortex or even the amygdala. Instead, the scientist would choose the dorsal parts of the PAG where the circuits for FEAR, RAGE, and the psychic pain of GRIEF are concentrated. There are no more emotionally aversive sites known in the brain. For instance, a patient stimulated in those areas may suddenly exclaim, "I am scared to death!" (Nashold et al., 1969). In contrast, the ventral regions are not as negative and are often positive, including prominently LUST and SEEKING urges. The PAG, however, is

not the only subcortical midline structure that participates in affect generation. But it is just one of the most important and probably the most potent (Figure 11.1).

Although higher brain regions are of critical importance in emotion regulation, a large and complex topic we will not dwell on in this book (but see, e.g., Harmon-Jones & Winkielman, 2007), it seems clear that raw emotional feelings are closely associated with the arousal of the same subcortical emotional systems that generate the instinctual emotional behaviors that we can see directly, as well as the related bodily physiological changes, such as autonomic and hormonal arousals, when monitored with complex technologies. If we are ever going to scientifically understand the causal mechanisms that generate intense emotional feelings, we have no option but to engage empirically with certain lower midline regions of the brain.

In sum, there are two overriding reasons to believe that phenomenally conscious core-SELF experiences may be dominated by affective consciousness. First, if one focuses on the anatomy of subcortical midline structures, one sees that the PAG lies at its foundational epicenter. The PAG is a structure into which all emotional systems converge to some extent, but especially negative affective ones (Panksepp, 1998a, 1998b; Watt, 2000). For this reason, we can view it as a structure that provides a massive assembly point of the neural systems that generate emotionality. If we are correct in hypothesizing that the subcortical midline structures are the substrate of the core SELF, the fact that the PAG is part of this system indicates that emotions may play an important role in the functioning of the core SELF. Second, the arousal of subcortical emotional systems produces valenced varieties of good and bad affective experiences. These experiences have been monitored by a host of evaluative tasks ranging from the seeking or avoidance of brain stimulation to conditioned place preferences/avoidances. If affects are elaborated by the SCMS and if the SCMS is the substrate of the core SELF, this indicates that the core SELF is the seat of affective experience.

SELF-RELATED PROCESSING IS GROUNDED IN LOWER BRAIN MOTOR FUNCTIONS

What are the neuroevolutionary foundations of the core SELF? There is an evolutionary reason to suppose that the core SELF may be supported by the motor apparatus and that primal core affective consciousness could not exist without this action-related scaffolding for the core SELF. First, sophisticated motor capacities may have existed long before animals developed the complex sensory-perceptual apparatus that allows us all to see and hear so well. Second, at the end of the day, it is physical behavior that determines survival; the purpose of sensory information and internal affective changes is to guide motor systems. If there is no capacity for internally generated actions, then sensory information would have no purpose—e.g., seeing allows a hermit crab to pull into its shell earlier and more effectively than trying to do that once it has been bitten. If the core-SELF substrate is supported by such instinctual motor-action systems, and the instinctual action systems contain affective properties, one can also begin to envision how certain types of sensory information could easily generate affective responses.

We propose that self-related information processing is integrally linked to the instinctual action tendencies of the brain. This, of course, is not to suggest that sensory functions are not involved but rather that the core SELF is laid out, if you will, in action coordinates. This provides a stable neural matrix for SELF-representation. As one proceeds upward within the brain to more idiographic selves, the sensory-perceptual apparatus becomes ever more influential. We noted above that most people find the idea counterintuitive that primordial consciousness is rooted in action systems. Almost everyone assumes that consciousness is a sensory function, because the contents of consciousness tend to be sensory impressions of one sort or another. (Our capacities for sight, smell, touch, taste, and hearing are sensory functions that generate much of what we think about.) However, just because the contents of consciousness are largely sensory does not mean that the ancestral foundations of consciousness are necessarily closely aligned with the sensory-perceptual apparatus. The “sense” that matters most, after all, is interoceptive and therefore self-related.

Perhaps most sensory-perceptual capacities should be envisioned as influences on the higher order “awareness” functions of consciousness rather than as consciousness itself. Obviously, we do not become less conscious in any meaningful way if we go blind. The overall quality of consciousness is modified much more by frontal-lobe damage, where motor

functions are concentrated. The frontal motor-action areas of the neocortex are heavily invested in executive functions, such as attentional focus, motor planning, imagination, and higher social emotions such as guilt, shame, and empathy. Obviously, these executive regions of the brain need to have perceptual inputs in order to know how to plan. In this way, one can see that the superficial contents of consciousness may be sensory, while the basic capacity to be conscious may rely definitively on the action-generating apparatus of the brain.

Most investigators may not have even considered the idea that primary-process consciousness is integrally linked to emotional-instinctual action coordinates because they assume that motor responses are mere "outputs" of the brain, and hence the motor apparatus just governs nonconscious motor reflexes like the knee jerk (this way of thinking has been especially prevalent in areas such as fear-learning, as noted in Chapter 6). How could such "mere" outputs of the brain constitute central integrative principles? Perhaps this relationship of instinctual actions being a foundation for consciousness may be easier to picture if we consider well-studied visual functions.

It has long been known that visual perception tends to degrade if it is not anchored in exploratory eye movements (Yarbus, 1967), In other words, the capacity to see deteriorates if one does not move the eye muscles. Also, as we will show, the stability of low-level visual perceptions in the superior colliculi (SC) is dependent on stable eye-movement coordinates. Therefore, eye-movement maps determine where, and presumably how, the SC processes the incoming visual sensory information (Sparks, 1988). The SC, in addition to their role in vision (i.e., the most superficial layer of the SC), are integrated with hearing and touch in successive neural layers down below—a most interesting evolutionary progression. Touch preceded hearing, and hearing preceded vision. This area is a candidate for being a core structure of the self (Strehler, 1991). In any event, an action system required for visual orientation lies at the very "basement" layer of the SC, which sits right over the PAG. In other words, the lowest neuronal layers of the superior colliculi constitute a basic motor orientation system that stabilizes, and perhaps permits, visual perception in self-referential coordinates, which may also interact with primary-process emotional systems just below in the PAG.

Thus, there are good neurophysiological reasons to conclude that self-related visual processing relies solidly on one's inbuilt visual motor functions rather than merely on the visual sensory-perceptual apparatus. Again, just bordering on the PAG in the midbrain, the last neuronal layer in the superior colliculi controls motor capacities that govern exploratory eye movements. These motor regions are more stable in how they respond to the world than the overlying sensory regions. For instance, the neural networks on the surface of the tectum (the outer layers of the superior colliculi) behave quite flexibly as they harvest information about the location of visual stimuli. However, exactly how one visually orients to stimuli determines which regions of the superior colliculi process the incoming information. In other words, the visual scene can "slide" across the visual-sensory SC depending on where the eyes are oriented. In contrast, the underlying motor systems, which generate appropriate visual orienting and searching movements, use a very stable set of action coordinates that remain in the same location regardless of what type of visual scene is being processed (Sparks, 1988).

This bears repeating. When one records from the visual surface of the SC as animals make orienting eye and head movements toward visual stimuli, the neural registrations of the stimuli on the SC change position; they "float around," so to speak, depending on how the animal has just oriented itself. In other words, the same stimulus in visual space changes locations on the surface of the SC depending on where the animal has moved its eyes and hence its focal attention. By comparison, the underlying motor map always remains stable and predictable, behaving exactly the same when animals make specific orienting movements. There are good reasons to consider that the functions of the emotional action networks were laid down by evolution in comparably stable ways. Such findings encourage us to suggest that the emotional affective functions of the core SELF are fundamentally based on the instinctual emotional action systems that we can visually observe in animal behaviors.

And to all outward appearances, all mammals have this type of stable "grounding" of raw emotional feelings intrinsically in their ancient emotional action systems, no doubt supported by feedbacks from the body. One thing we can be confident about is that further down, in the most ancient regions of the midbrain, right smack in the very middle of the upper brain stem, we find the PAG where all the emotional action systems,

especially negative affective ones, along with their powerful affective charge, converge. This certainly speaks loudly for the primacy of emotional action systems in the evolution of the mind.

If we pull all of the above data together, it would seem that coherent emotional action systems have primacy in the genesis of consciousness. Obviously, the brain needs stable mechanisms for generating psychobehavioral coherence. If our concept of a core SELF is on the right track, then it is reasonable to postulate that all the diverse "idiographic" selves depend on the integrity of the core SELF that is so deeply affective. Therefore, an affective core SELF may provide a solid neuroevolutionary scaffolding for all higher self-functions.

THE EMERGENCE OF IDIOGRAPHIC HIGHER SELVES

The deep motor areas in the superior colliculi and the underlying zones in the PAG with which they interact are more richly connected with motor areas in the frontal cortex than with posterior cortical sensory areas. Indeed, the frontal motor areas in the neocortex are where plans and intentions are generated, indicating the link of those psychological features of the brain with motor functions. In yet other human experiments that have studied brain activity changes, the feeling of "owning" the activity was linked to increased activation in a variety of frontal cortical brain regions (Ehrsson et al., 2004). In addition, the magnitude of activation was commensurate with the degree of subjective ownership.

The frontal motor regions of the neocortex are brain regions where idiographic selves begin to "incubate" as a function of lived lives, as those higher networks help to establish behavioral priorities in time, on the basis of sensory information. It has long been known that when there is damage to the frontal/motor regions, more powerful changes in personality result than when damage occurs to the posterior sensory regions of the neocortex (Elsinger et al., 1992; Passingham, 1993; Perecman, 1987). Thus, the construction of idiographic selves is elaborated by the higher motor action-oriented executive apparatus of the frontal cortex. Higher-order emotional action capacities may be especially important for the genesis of the affectively tinged, idiographic self-functions that we call personality.

An intriguing question remains: To what extent do higher cortical regions actively participate in the generation of affective states? Clearly there are a

large number of top-down cognitive functions that can instigate and regulate emotions (Gross, 2008). As every wise person knows, it is easier to reach a reasonable cognitive decision on affectively loaded matters when emotional arousal is comparatively low. Conversely, subcortical emotional arousal tends to energize and sustain thinking in obsessive self-serving grooves. Thus, it is not surprising that the medial emotional regions and the lateral, more cognitive, regions of the frontal lobes are in a see-saw balance (Liotti & Panksepp, 2004b). When one views an event in the world affectively, the medial frontal areas are more aroused than are the lateral (cognitive) areas of the frontal lobes. If one looks at the same stimuli more cognitively and less emotionally, the balance is reversed, with lateral areas becoming more activated compared to the more emotional, medial regions (Northoff et al., 2009). Clearly, strong emotions and rationality do not go together. At the same time, it is also clear that feelings are very important in making up one's mind when there are choices to be made (Damasio, 1994).

We must note that more recently some complex emotional action responses, such as fearful grimacing, have been instigated by microstimulation of certain areas in parietal somatosensory cortical regions (Step-niewska, et al., 2005). We do not yet know whether these stimulations can mediate affective *rewards* or *punishments* in learning tasks. Until such issues are resolved, it is reasonable to believe that they are learned substrates of emotional actions that emerged in those brain regions through life experiences rather than evolutionary heritage.

DUAL-ASPECT MONISM AND THE ANCESTRAL MIND

Taken together, the core-SELF system, constituted from a complex reticulum of instinctual circuits, generates not only emotional behaviors and associated bodily changes but also raw affects. For these reasons, we espouse a theory of *dual-aspect monism. Monism*, as opposed to dualism, proposes that everything that happens in the mind is ultimately rooted in a single substance—in this case, the physical brain, apparently most fundamentally in the subcortical and cortical midline systems. *Dual aspect* refers to the idea that these midline systems concurrently generate two seemingly distinct aspects of emotions—both coherent emotional action tendencies and associated primary-process psychological states (affects). The fact that both are reflections of the self-same integrative process in the

brain allows us to use events that we can measure objectively (instinctual emotional behaviors) as proxies for subjectively experienced processes that we must infer indirectly (i.e., instinctual emotional feelings). Thus, instinctual RAGE behaviors in an animal reflect an internal RAGEful affect. This allows us to directly read affective issues in animals based on their external behaviors. We know that the circuits that generate RAGE do not feel good; given the chance, animals will try to escape such stimulation (Panksepp, 1991).

We are not suggesting that these systems are not sensitive to the many states of the body, activities in the rest of the brain, and the events of the world. They obviously are, but the lion's share of the mystery of affective experience is to be found in the dynamics of the outlined medial brain systems. At least now we know where to focus our efforts if we want to understand raw emotional feelings in greater detail. We regret that there is comparatively little detailed work being conducted on these neural systems from the emotional-feeling perspective, which should have important impacts on understanding many mental health issues and the nature of psychiatric disorders (see the next two chapters for discussions).

One can also see that the core SELF is the part of the brain that generates rudimentary learning-cognitions, probably largely unconscious, in that it combines emotional affects and action tendencies with rudimentary sensory impressions. As we have discussed, these sensory impressions (sight, sound, and so on) need not emanate from the neocortex. They can control emotional learning completely through subcortical sensory systems (Chapters 6 and 7). The core SELF also receives information from the body's interior. For example, the interoceptive neurons can detect water, energy, thermal, and other imbalances as being affectively represented in the core SELF as thirst, hunger, or cold. The emotional systems are also directly regulated by autonomic information, such as blood pressure and various hormonal states of the body. Some external stimuli, such as the smell of a predator and pain, can arouse the FEAR system directly—resulting in the generation of affect and action tendencies in conjunction with simple perceptions. But most of the linkages to world events are ultimately conditioned through learning.

The process of relating external stimuli to the core emotional SELF and the gradual emergence of various idiographic selves are surely not isolated cognitive processes. As far as the emergence of idiographic selves is

concerned, complex cognitive processes—our capacity for imagination, planning, and an appreciation of cause and effect—are surely involved. But they are always attended by an affective valuation of the world, which allows higher brain systems, during psychological maturation, to stay in touch with the utility (valuation) of various perceptions, in order to facilitate the evaluation of future courses of action that can enhance the survival of each individual and, in the long run, of the species.

THE NEURAL SELF AND PSYCHOLOGICAL WELL-BEING

In this section we will raise two points that may impact the field of mental health. First, as human beings we all have highly developed idiographic selves, so it is difficult to understand what it might feel like to experience self-related processing in its most basic form. However, we imagine that it might cause people and animals to feel that they somehow *own* (Blakemore et al., 2000; Blakemore, 2003; Ehrsson et al., 2004; Jeannerod, 2003) the world in which they live, that they are active agents in causing things to happen (Gallagher, 2000; Gallagher & Frith, 2003). They might obtain a sense of *mineness* about their experiences (van Gulick, 2004) and they may project their emotional values onto objects in the world. Conversely, they also introject valued objects into their internal lives. This sense of ownership might allow people and animals to feel that they somehow belong to their worlds—that they are affectively *embedded* in the context of their lives (Izdebski, 2008; Northoff, 2004; Schore, 1994; Zinken et al., 2008).

As we have already noted, severe attachment disorders early in life have been linked to an attenuation of neural lines of communication between the neocortex and emotional networks of the brain (Schore, 1994). This attenuation may restrict the ability of the neocortex to appropriately inhibit and regulate emotional expression. We know that one of the big functions of the neocortex is to suppress emotionality, because such intense affective arousals may disrupt subtle forms of higher cognitive processing. Early attachment disorders often result in personality disorders later in life, and people with personality disorders typically have difficulty in modulating their emotions. For these reasons, many have presumed that the dearth of neural connections (which would allow one region of the brain to effectively modulate the activities of other regions) may be responsible for

personality disorders. In simple terms, the higher midline regions regulate the lower midline regions; the medial and lateral regions of the frontal cortex also work in a see-saw fashion. Perhaps the frontal executive and posterior perceptual regions of the cortex have certain mutual interdependencies that are needed for a healthy and happy upper-mind development.

Although we are persuaded by this argument, we offer an auxiliary way to understand personality disorders and related mental conditions. We have noted that significant damage to subcortical midline systems, such as *the complete* destruction of the PAG, results in a severe diminution of self-related information processing. People and animals that have sustained this kind of damage are not meaningfully conscious. However, more minimal or subtle damage to subcortical midline systems, to the core SELF, might exert a deleterious effect on a person or animal's sense of emotional orientation. The sense of being embedded in one's world might be impaired, diminishing the capacity to be an active agent who can claim a sense of *mineness/ownership* about important personal experiences. These are psychological features about which people with personality disorders complain. Therefore, in addition to the failure of cortical inhibition, neural imbalances within the subcortical midline systems may contribute to personality disorders as well as many other psychiatric problems (Koenigsberg, 2010; Stein, 2009).

The second point relating to mental health focuses on the fact that cortical and probably also subcortical midline systems have highly active *resting* states, or a highly active *default mode network*, as it is often called (Damoiseaux et al., 2006; Fox and Raichle, 2007; Fransson, 2006; Raichle et al., 2001). This high resting activity, which probably reflects internal ruminations, diminishes as one focuses on external demands. Because these midline systems of the brain mediate self-related processing, we hypothesize that high arousal of the default mode network should not be increased further by *external* cognitive stimuli but only by internally generated emotional materials. Indeed, this has been found in depressed individuals who are prone to ruminating upon their problems (Northoff, 2007). Such findings help explain why the brain may not readily integrate incoming information during high levels of emotional arousal and why cognitive processing in particular can be compromised at such times. Psychologically, this is not surprising. We all know from personal

experience that we cannot think efficiently when we are in a state of affective arousal. However, the above findings offer an established brain mechanism for that, allowing more detailed analysis of the underlying brain processes.

What is of further interest is that the high resting state indicates that self-related processing may be an ongoing process/activity. Such ongoing self-related processing might allow us to maintain a continuous and temporally extended *sense of relatedness*. In this way, the normal high resting-state activity can be seen as a "physiological baseline" that is indicative of mental health (Northoff and Bermpohl, 2004; Northoff et al., 2006). Thus, it will be most interesting to study more intensively how this resting state is modulated by various types of strong internal emotional arousal, and whether different neurodynamic patterns emerge in these CMS structures as one becomes emotionally aroused in distinct ways. As noted, it is already evident that such brain functions are hyperaroused in depression, which may highlight the fact that depressed people have an extreme, negatively affective self-focus, perhaps commonly provoked by ongoing overarousal of the GRIEF system that amplifies psychological pain. We would anticipate that this heightened neural activity would also be evident in subcortical midline regions during emotional arousal.

It will be very important to develop more direct neural measures of such MindBrain changes. Because fMRI cannot monitor neural activity directly, it will also be desirable to have direct electrical measures of brain arousal as opposed to secondary effects such as changes in blood flow. The only applicable technique is still the first type of brain imaging ever discovered: electroencephalography and, more recently, its fancy cousin, magneto-encephalography. When studies like that are done, with *well-positioned electrodes* recording from the CMS and the SCMS, we anticipate they will prove to be the optimal brain regions from which one might obtain affect-specific neuronal signatures from the human brain (Bekkedal et al., 2011; Northoff et al., 2009).

SUMMARY

Philosophers who are interested in consciousness have not been able to escape considering the nature of the self. For example, although Descartes did not explicitly speak about the self when he explored the phenomenon of

consciousness, he nevertheless assumed that it existed. His declaration of "I think, therefore I am" only entitled him to confirm the existence of thinking, which is a disembodied form of higher consciousness that generates awareness of one's experiences and thereby ponders world events and one's place in the flow of life. But he assumed that something, namely his *self*, was doing the thinking (Copleston, 1962b). Thus, Descartes implicitly accepted that the existence of consciousness, along with a coherent and stable set of autobiographical memories, implied the existence of a self. Of course, he did not envision it as a strictly neurobiological process but as one that was related more to nonmaterial aspects of a religiously conceptualized soul. Thus, he chose to make a sharp demarcation between the physical and mental realms, reserving the mental (spiritual) realm to human beings alone. This choice has impaired the progress of BrainMind science ever since.

In this chapter, we have presented an alternative hypothesis about the nature of the self, which may help to explain how affects are created in mammalian brains. Central to our hypothesis is the assertion that midline systems in the brain, which give all mammals a universal (nomothetic) core SELF, can support various renditions of the self (idiographic forms) in other regions of the brain related to higher information processing. It is becoming ever more evident that midline systems mediate self-related information processing of all kinds (Northoff et al., 2006). We envisioned that the self initially evolved as a homologous nomothetic core SELF, which helps the rest of the brain elaborate more idiographic forms of selfhood, especially the vast diversity that is evident in highly cerebrated species like humans but not absent in lower mammals (although the diversity is greatly diminished if animals are genetic clones). Additional anatomical and experimental data allowed us to hypothesize that the primary-process emotional systems play a pivotal role in the functioning of the core SELF as well as learned valuations of the world that eventually, with higher cortical involvements, lead to a diversity of idiographic selves. On this basis, we proposed that affects are created when midline systems assume distinct types of neuronal firing patterns when the various emotional networks are aroused.

Affects provide an ongoing evaluation of the external and the internal world. Further, because of their evolutionary design characteristics, primary-process affects always evaluate the internal and external world in

relation to the survival of the individual and the species. Thus, the midline systems that generate and regulate emotionality are continuously involved in self-related ("What's in it for me?"-type) processing of external information. In this way we can again envision how all mammals are "active" information-seeking creatures rather than just "passive" information-integrating ones. This is one reason it is much wiser to conceptualize the mesolimbic dopamine network as one that participates in elaborating SEEKING action urges rather than *just* producing higher-order processes such as "wanting" and "incentive salience" or being "the brain reward or reinforcement system." The SEEKING concept can explain the fact that sensory stimuli become more capable of attracting both approach behaviors and focused attention in a large variety of emotional contexts. It also helps explain why artificial activation of the SEEKING system is "rewarding," but not in the typical sense of promoting pleasurable sensations. It promotes enthusiastic engagements *with* and appetitive eagerness *toward* the world, focused especially on those stimuli in the world that predict either euphoric excitements or satisfactions and pleasures.

We have reviewed a range of data indicating that these midline systems, which support self-related emotional processing, are fundamentally based on intrinsic action-motor processes rather than just sensory inputs into some kind of cognitive analyzer (as is assumed by all read-out theories). This implies that emotional arousal concurrently results in the generation of affects and in the generation of action tendencies: alterations in muscle tone, autonomic responses, and relatively stereotyped emotional action urges such as approaching or running away, engaging socially in various ways, or withdrawing. For this reason, we espouse a theory of *dual-aspect monism*, which states that subcortical midline emotional systems concurrently generate various behavioral, physiological, and affective emotional manifestations through a coherent integrated system for SELF representation (Panksepp, 2005b, 2009b).

Finally, we noted that midline systems operate at a high resting state when nothing else is happening to an awake animal. This allowed us to consider the possibility that emotionally healthy animals and humans typically engage in continual self-related processing of information, which may become extreme—ruminations filled with negative feelings and self-loathing—in disorders like depression. Through the existence of emotional

systems in the brain, most especially the ancient SEEKING system (the granddaddy of all the positive social-emotional systems) that participates in so many other emotional responses (e.g., "the seeking of safety" promoted by the FEAR system), animals become active agents in their worlds rather than passive zombies. And with higher MindBrain evolution, this means seeing the world with an affective mind's eye, with ears listening for emotional nuances, and having the largest diversity of feelings, most intensely, when one is in various forms of social-emotional contact with one's own kind as well as evolutionarily related animals that have similar emotional systems. The human-animal bond, easily formed with companion animals, is strongly based on the fact that we share evolutionarily related social-emotional systems.

The existence of subjectivity in the brain also means that we cannot simply approach the study of brain functions with the traditional third-person tools of science. The mind is not a neutral thing like a stone in a field. It has a point of view, an I-ness. And every viewpoint that really matters is strongly tinged with affect. To see the BrainMind for what it really is, we must deal with subjectivity ever more effectively in neuroscientific terms. When we do an accurate archaeology of the mind, we find affective experience at the mind's foundation. And, surprisingly, affect remains one of the most poorly understood, understudied, and under-discussed functions of the brain. Affect is the very heart of what the brain does when it processes external "rewards" and "punishments." Affective change may be the way "reinforcements" work in the nervous system to create learning and memories. As the first author will discuss in the following chapter, our failure to view the BrainMind in this way has had profound negative consequences for the maturation of modern biological psychiatry and for a better and more coherent understanding of how psychotherapy can help restore emotional equilibrium.

CHAPTER 12

Brain Emotional Systems and Affective Qualities of Mental Life

From Animal Affects to Human Psychotherapeutics[1]

The further we go out into the outside universe, the closer we come to our origins, reaching back to the beginning of time. The deeper we go into the dark matter of the unconscious, the more we understand our origins and our present . . . the further we can predict future problems, whether of anxiety, drug addiction, or sexual impotence.

—Arthur Janov (2007, p. 17)

THE GOAL OF THIS PENULTIMATE chapter is to try to integrate the diverse lines of thought covered so far in order to draw out some psychotherapeutic implications of our emerging appreciation of basic emotional systems. At this level, one needs to deal with a new understanding of the BrainMind that has not yet been integrated with clinical thought. The goal here is not to give advice or make definitive claims. It is to explore the multifaceted dimensions that our new understanding of the emotional foundations of mind offers for our therapeutic endeavors—whether in the interpersonal dynamics of the consulting room (Siegel & Solomon, 2012) or on the traumatic field of life itself (Belenky, et al., 1996).

My viewpoint is that substantial therapeutic effects can be achieved in affective disorders by direct manipulation of primary-process emotional

circuits, through psychological, somatic and physiological approaches. The more traditional view is that lasting change can only be achieved by working on emotional dynamics through the gateway of language—by dealing with individual life events through the mediation of tertiary cognitive processes. This view is, of course, accepted by all psychotherapists, but there are some who believe that, for many emotional ailments, especially those arising from early developmental problems, one also has to address, more directly, the underlying emotional dynamics, and sometimes even work at nonverbal primary-process levels (e.g., Janov, 2007). While all agree that psychotherapy, as traditionally conceptualized, has to operate through the linguistic gateways of the human mind, there are reasons to believe that the next revolution in psychotherapeutics will emerge from new neuropsychoanalytic perspectives and more direct manipulations of the affective MindBrain functions, using multimodal approaches. To conclude this narrative, the final "Coda" chapter will frame the goals of this book in a historically relevant philosophical perspective.

A premise of this book is that the farther we go into the depth of our affective foundations—our inside universe—the closer we come to our mental origins. At some point, as we descend into ever more ancient recesses of our brain, there may be nothing but unconscious neural networks creating pure behavior—organisms going hither and thither with no feelings—perhaps like undulating jellyfish riding the tides with the dimmest forms of preconsciousness. Maybe the neural networks in our spinal cords are like that: deeply unconscious. We just don't know and have no good way to find out. But we can finally fathom the nature of primal emotional feelings—they arise from the same brain regions as unconditioned emotional actions and reactions. Understanding these processes, provides a solid foundation for additional progress. The understanding of how affective dynamics are created in mammalian brains may be the single most important scientific question for psychiatry and consciousness studies, as well as for psychotherapists who try to restore emotional balance. To put it mildly, the history of this field has been chaotic, with prominent blind alleys, such as the James-Lange neocortical readout theory of emotions, which is still at the forefront of understanding for many psychologists, although it has no sustained critical lines of research to support it. At best, it currently deserves to be a diminutive leitmotif behind the major within-brain causes discussed in this book. Many

others believe emotions, indeed affective *feelings*, can be dynamically unconscious. Perhaps, but that may only occur if feelings are denied or repressed by excessive cognitive activities, a common disposition of the human mind, which can surely inhibit subcortical emotional turmoil to some extent. But those pressures of mind will seep out in unexpected ways and create chaos in people's lives.

It may come as a surprise to some psychotherapists that, according to the present analysis of the ancestry of mind, the traditional construct of the unconscious (introduced by Sigmund Freud) is not completely "unconscious"—it is not totally bereft of experiences. What is deeply unconscious are the automatic learning and memory processes of the brain. The Freudian dynamic unconscious, or preconscious (he used these terms ambiguously) is supposedly partly constituted of the emotional states described in this book. But these states, when sufficiently intense, are experienced affectively, albeit not reflectively (cognitively), not only by humans, but surely by many other animals. We can now be confident that other mammals do *experience* their emotional arousals—although most, like newborn human infants, may not be reflectively *aware* that they are having such experiences. That is what the evidence now indicates, and it may be worth remembering that Freud also often claimed that *the affects* are never unconscious. It feels like something to be in a primal emotional state. They are raw affective experiences—special phenomenal states of mind, a unique category of *qualia*, that arises from the very foundation of the conscious mind.

DEVELOPMENT OF AFFECTIVE STATES AND THE EVOLUTIONARY LEVELS OF BRAIN AND MIND

Although intense primary-process emotional arousals, as they occur in mammals, are probably never un-experienced—are never unconscious—the secondary-process mechanisms of learning, the next level of control, are deeply unconscious. This bears repeating. As far as we know, learning and memory reflect neural mechanisms grinding away in deterministic ways, connecting our primal affects to world events. As a result, complex forms of consciousness emerge in higher tertiary-process brain regions—the neocortical mantle, with very different casts of mind (more purely cognitive representations of self and the world)—than those found in the ancestral,

primal attentional, emotional, and motivational terrain of subneocortical regions, where affective states prevail. They are evolutionary solutions to anticipate the future intrinsically, without forethought, which arises from their profound influence on neocortical programming.

Our higher mental activities are profoundly cognitive, as neocortical brain regions (always in conjunction with lower BrainMind functions) construct images of the world from the diverse sensory portals that allow us to remain in contact with external events. A neuroscientifically and genetically defensible position is that the neocortex is fundamentally *tabula rasa* at birth—a random-access-memory type of blank slate—with most of the highly predictable functional specializations that come with maturation being a consequence of subcortical specializations weaving predictable types of cortical "modularization" through the developmental magic of epigenesis, along with a great deal of culturally guided learning. And thereby, our autobiographical storehouses of knowledge and memories emerge, much of it under the instructive and motivational influence of the SEEKING system.

Without clear visions of how the lower affective mind and the higher cognitive mind are interfaced (very heavily in the medial cortical and subcortical basal ganglia regions: see Figures 1.1, 1.2, and 3.1), we cannot have a clear discussion about what it means to have psychiatrically significant problems of the mental apparatus and how new MindBrain therapies can be developed. It is likely that most behavioral and cognitive therapies work primarily because they come to regulate affects and better coordinate cognitive views with positive affects. Only recently has a rich conversation emerged about the potential utility of more direct dynamic affective therapies, where an individual's emotional life is situated front and center.

In the future we have to recognize the overwhelming evidence for the subcortical localization of affective processes, so dramatically demonstrated by localized brain stimulation work already summarized, along with remarkable brain-imaging work such as that of Damasio and colleagues (2000; see Figure 12.1). Of all the many brain changes seen in humans during emotional arousals, induced from one's own storehouse of autobiographical memories, the overwhelming proportions of arousals were subcortical as humans experienced what we would call RAGE, FEAR, GRIEF and joyfulness (PLAY?) (see Figure 12.2). If anything, cortical

regions tended to shut down during emotional arousals. Clearly, to make sense of the affective brain, we simply must understand the evolutionary layering and integration of neural developments as well as, of course, the vast inter-digitations among levels of control (the nested hierarchies which I discussed in Chapter 2, see Figure 2.1).

This evolved, multitiered vision of the BrainMind has implications for psychiatric disorders and their therapies, both neurochemical and neuropsychological. Here, I will briefly explore some implications of this knowledge for understanding human emotional problems and for the development of new clinical interventions aimed at helping reestablish emotional homeostasis when the vicissitudes of life, and the affective imbalances of the MindBrain, have become exceedingly troublesome to people.

In doing this it is important to remember that animal research has told us close to nothing about the transitory flow of memories and thoughts that accompany our emotional arousals. But at the same time affective neuroscientific approaches to the mammalian mind have told us most of what we know, at a *causal* level, about how brains actually generate emotional feelings and about how the deeply unconscious learning and memory processes of mammalian brains actually operate. However, access to higher mental *experiences* in other animals remains a scientifically unmanageable problem. The vast multitiered BrainMind interactions create abundant layers of complexity in our attempts to understand mental disorder—to construct clear word-images, conceptual symbolic descriptions of psychiatric disorders and the influences of therapies used to ameliorate the destructiveness of unregulated emotions in people's lives.

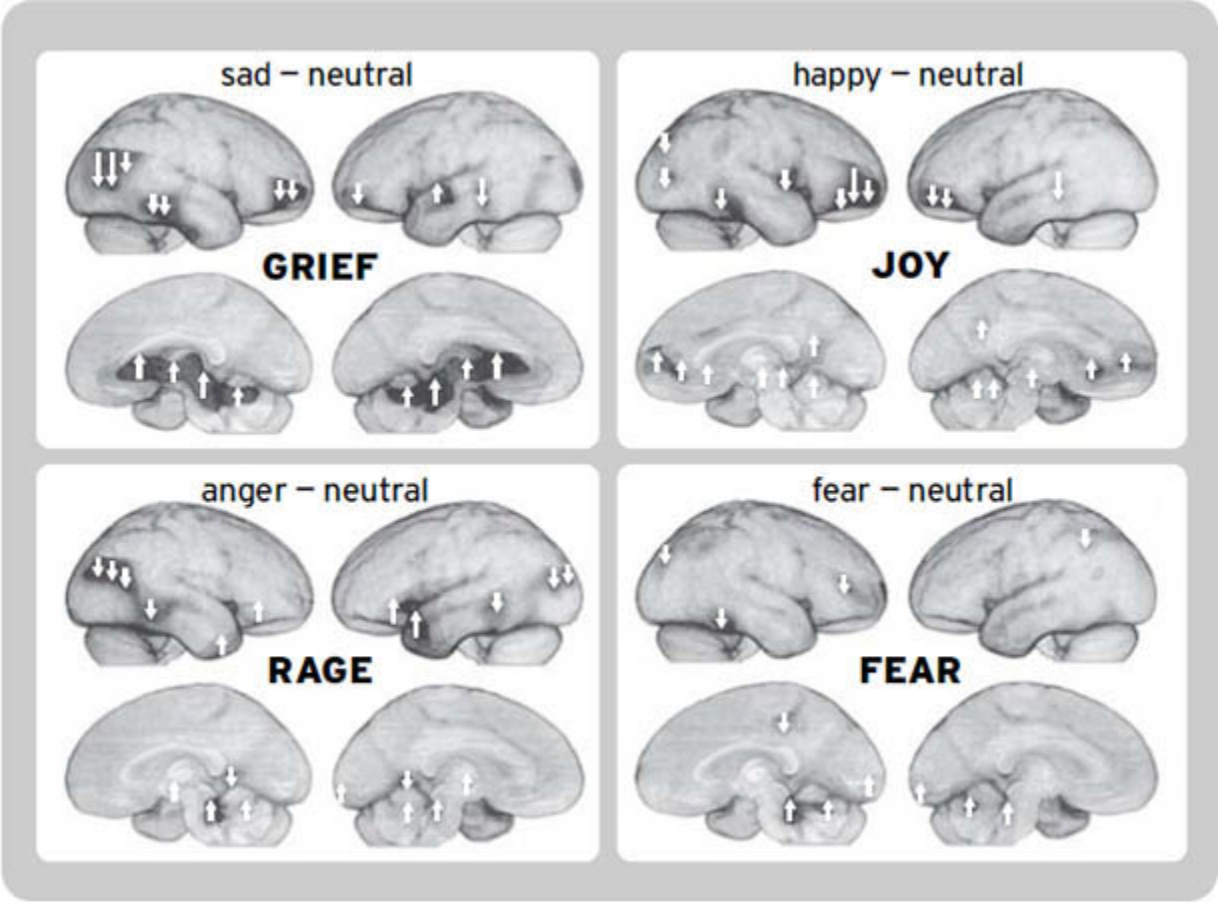

Figure 12.1. An overview of brain arousals and inhibitions when humans are experiencing four basic emotions: sadness, happiness, rage and fear, during PET scanning (based on Damasio et al., 2000). Distinct lower, subcortical brain regions exhibit arousals that are evident during each of these emotions, while cortical inhibitions (reduced blood flow) are present in various cortical areas (the quantitative data in Figure 12.2). Because the color-coded changes are difficult to see on these black and white renditions, upward arrows indicate increased subcortical arousals, and downward arrows indicate reduced regional neocortical arousals (data graciously shared by Antonio Damasio; for color figure, see Panksepp, 2011b).

The limited goal of this chapter is to explore the relevance of affective neuroscientific knowledge to selected psychotherapeutic issues, as well as at times to interject synoptic fragments of the affective views of the emotional mind elaborated so far. I also wish to emphasize developmental perspectives that may protect against as well as promote future emotional

problems—namely how positive affects can successfully counteract negative affects. It is becoming ever clearer that emotional resilience can be advanced through childrearing practices, sustained positive interpersonal regard, as well as by diverse time-tested (e.g., psychoanalytic) and newly emerging clinical interventions throughout the life span. Of course early experiences have long been recognized as being of definitive importance in long-term mental health issues, and now the neural mechanisms are being illuminated, most dramatically through preclinical (animal) research.

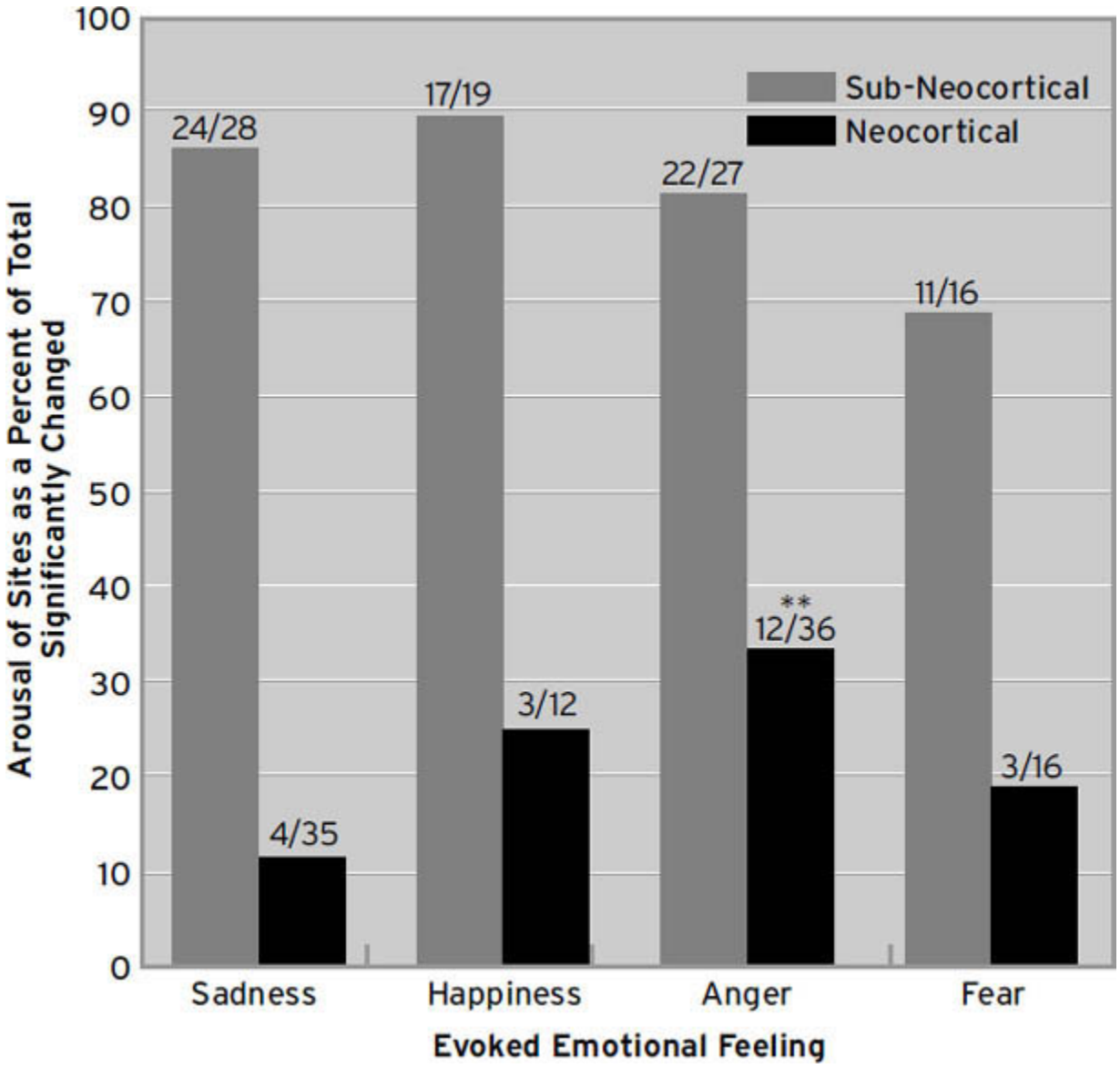

Figure 12.2. An overall summary of the data provided in Figure 12.1. Clearly, subcortical arousals prevailed over areas that exhibited inhibitions when humans were experiencing basic emotions. Anger showed the greatest overall percentage of neocortical arousals, but still the general asymmetrical pattern held for each emotion (data abstracted from Damasio et al., 2000).

Thus, affective neuroscience may be of considerable use for all psychotherapists and parents, especially those who are concerned with healthy child development. People should understand that children are born with certain affective capacities that are central for the quality of their lives (see Sunderland, 2006). Such knowledge will promote better childrearing practices, where (i) the affective life of children becomes a central issue for helping parents to know what their infants need to flourish (Narvaez et al., 2012; Worthman et al., 2010); (ii) positive attachment dynamics in families become the key to helping children thrive (Code, 2009; Hughes, 2007); and (iii) realistic visions of our emotional lives, with fulfilled potentials for mindfulness, promote positive personal transformations (Siegel, 2007, 2010).

In the previous 11 chapters we outlined key scientific issues arising from affective neuroscience, with some clinical reflections. I will now discuss (i) how an understanding of primary-process emotions must be incorporated into evolutionarily informed animal models of psychiatric disorders, (ii) how an understanding of primary affective processes provides a new foundation for psychiatric and psychological science, and (iii) how these new lines of understanding provide the basis for novel approaches for the development of biological and psychotherapeutic interventions that target the affects more directly than ever before.

Key questions I will address here include: How do raw affective experiences created within the brain relate to emotional disorders? What are the implications of this knowledge for achieving emotional homeostasis, greater feelings of well-being, and more positive outlooks on life? And of course, how can affective neuroscience research on other animals give us better knowledge about the emotional lives of human beings?

There are many novel strategies waiting to be evaluated, both clinically and preclinically (i.e., in animal models). How can we counter disorders characterized by negative affects (e.g., depression) with our increasing

knowledge of positive affective systems? For instance, we can envision many new ways to use the positive affects of SEEKING and PLAY to counteract the negative affects of depression and anxiety disorders. With the discovery that we can monitor animals' emotional feelings rather directly through their affective vocalizations and their instinctual emotional tendencies, the intellectual commerce between animal research and psychiatric practice, both with regard to development of new medicinal and psychotherapeutic interventions, can be enriched. However, we should not underestimate the challenges that remain for interdisciplinary integration. Before proceeding to clinical issues, let us consider the difficulties that have prevented a fuller interpenetration of clinical and basic science issues concerning the nature of our primal emotional minds. In a phrase, the relevant neurosciences (from behavioral to molecular) currently need, but do not have, clear vision of the primary-process affective infrastructure of the BrainMind. Indeed, for silly historical reasons, the topic of animalian emotional feelings and implications for psychiatry is rarely discussed. In psychology, most of the discourse is at the tertiary-process level, where cognitions and emotions are inextricably conflated, leading to very difficult, at times muddled, discourse in which few of the concepts are neuroscientifically anchored. More clarity can be had if we respect the hierarchical circular causal influences, first bottom-up and then top-down, that control the BrainMind (see Figure 2.3).

In many past writings, I have made the case that the primal affective foundations of the mind are profoundly biological and subcortical. The guiding principle has been that raw affects arise from the dynamics of large-scale neural networks that generate instinctual emotional behaviors rather than from the higher, self-related perceptual brain regions that mediate cognitive awareness. Primal emotional feelings go hand in hand with emotional action dynamics, constituting distinct varieties of mental experience. The resulting affective dynamics also have characteristic whole-body feels to them: They are the primary-process phenomenal-experiential states that depend heavily on visceral bodily representation within the brain (the core SELf) that engenders primal "affective consciousness" and secondarily on autonomic arousals that can be indirectly experienced by the higher mental apparatus. Because these subcortical dynamics—large-scale analog network functions—are the primordial wellsprings of emotional life, psychotherapists need to clearly

envision the nature of these psychic energies in order to more directly, and thereby more effectively deal with complex human emotional problems in psychotherapy.

Sharing and discussing our emerging knowledge of these systems, which exist in all human brains, may, in and of itself, be a valuable therapeutic insight for those in emotional distress, who are, at times, beyond wits' end as to what is happening to them. Just telling distressed individuals that their seemingly free-floating emotional distress is "real," even when done with empathy, may simply come across as vague and insubstantial reassurance. Explaining that everyone has a discrete set of emotional feeling systems, which are distinct brain processes shared by all, and that these systems exist for good and important reasons in all mammalian brains is solid knowledge. And, as a general rule, many people would prefer being constructively informed in addition to being empathetically reassured. In the higher MindBrain, affects and cognitions can work productively hand in hand. They can also wage an all-out war. They are two very distinct aspects of our consciousness. Ultimately all psychiatric disorders are manifested at both levels. If one modifies affects, cognitions often will follow, especially with good counsel. Changing cognitions can also work, but not if the affect doesn't follow suit. The reciprocal interactions of cognitions and affects makes this difficult (see Figure 12.3).

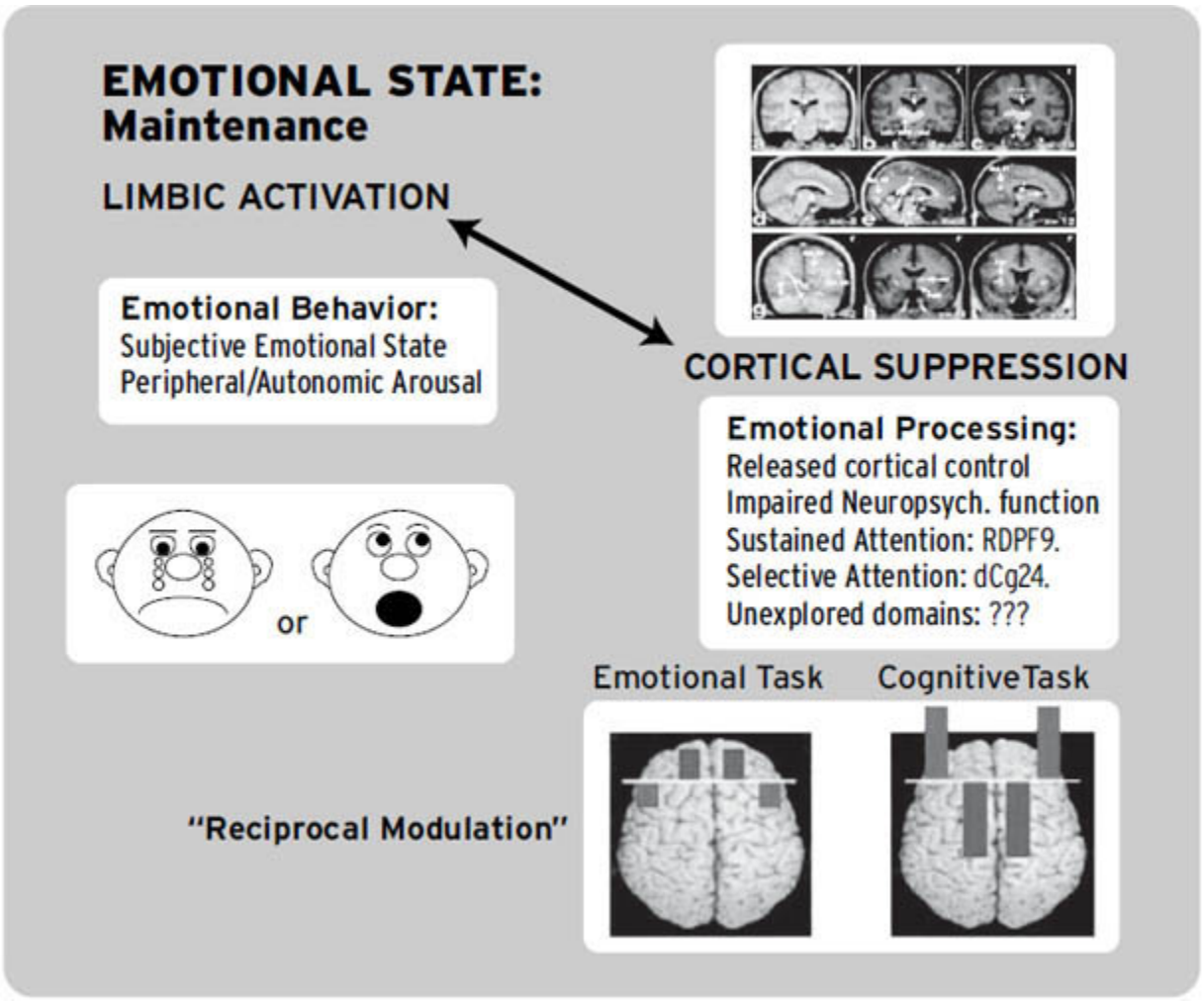

Figure 12.3. A summary of the general patterns of neocortical and subcortical arousal changes in human brains as a function of emotional state activation. Overall, there is consistency of subcortical limbic arousals whenever emotions are aroused, with decreased neocortical arousal (summary diagram derived from Liotti & Panksepp, 2004). The upper right insert highlights an example of brain arousals exhibited by a male brain during orgasm. Practically all the arousals are in subcortical areas that are known to regulate basic male sexual behavior, as summarized in Figure 7.2. (The PET scan is provided graciously by Janniko Georgiadis and colleagues.) The lower right insert is a summary of fMRI data when people have been viewing the same emotional picture with an emotional-feeling set of mind (left) and a cognitive-analytic set of mind (right); during affective viewing there are midline frontal arousals, and lateral working-memory inhibitions, while the pattern is dramatically reversed during a more

cognitive-analytic viewing of the same materials (the fMRI brain scan summaries are graciously provided by Georg Northoff).

AFFECTIVE NEUROSCIENCE AND THE DYNAMICS OF EMOTIONAL MINDS

With apologies for repeating the point, knowledge of the primary-process states generated by the seven basic emotional systems described in this book is essential to a neuroscientific and evolutionary grounding for clinical thinking about emotional disorders. Although most of the details of these systems remain to be worked out, an affective neuroscientific infrastructure has been established that permits basic emotions, and their powerful affects, to guide clinical thinking. Here, I will focus on the implications of that knowledge for affect-specific therapeutic issues, especially from developmental perspectives.

New affective balance therapies (ABTs) can now be devised for rebalancing the "heart" rather than just the "head," to put it metaphorically —an abiding goal of new ABTs should be to aim for more direct and more precise beneficial interventions within the primal affective lives of individuals. This should obviously include cognitive restructuring, but often that, by itself, is not sufficient for optimal reestablishment of affective homeostasis. To put it simply, a psychotherapist wishes to alleviate, even "cure," unpleasant problems in psychological life that promote suffering. If what has gone wrong is within recent and specific sets of life problems that have clear cognitive precipitants, then cognitive-behavioral counseling is an ideal way to proceed. On the other hand, most severe emotional problems do not simply arise from recent events. Their etiology can be traced to sustained past stressors and traumatic vicissitudes, often extending back early in life, so far back, that few explicit memory traces remain—just imbalanced emotional states and associated cognitive biases.

These early imprints can be remarkably long lasting because very stressful life experiences have left emotional systems sensitized or desensitized, with permanent, epigenetically induced high-stress reactivity and excessive primary-process negativistic feeling. We must also appreciate that events that are overwhelmingly stressful, and wonderful, to young minds may not seem so to mature adults. Similar stressors at different ages

have different effects depending on critical maturational issues, as well as the genetic "susceptibility background"—the primal temperament—of each individual. The classic temperaments—choleric, sanguine, melancholic and phlegmatic—were not that far off the mark, even though our new Affective Neuroscience Personality Scales provides more objective estimates of such emotional strengths and vulnerabilities (Davis, et al., 2003; Davis & Panksepp, 2011). Similar tests need to be developed for adolescents and younger children, for they may be very helpful for teachers and guidance counselors. For instance, the profound desire of youngsters to have abundant real play in their lives is commonly underestimated. Compelling angers and unrealistic fears need to be known. They need to be acted out. They need to be talked about, with care.

Below, I will focus on how an appreciation of primary-process cross-mammalian emotional systems may guide understanding of the role of affective experiences in the genesis of psychiatric disorders, and also guide development of new therapies to alleviate human suffering. But such issues are also of foremost concern in optimizing therapeutic relationships. Across many studies it has been found that the emotional quality of the client-therapist relationship, rather than the specific therapeutic approach taken, is perhaps the most important overall variable in the outcome of psychotherapy (Lambert & Barley, 2001). This is substantially due to the critically important need for emotionally attuned therapists to share genuinely positive social feelings with those clients who are in psychic distress. A better understanding of our intrinsic prosocial emotions may also help illuminate how affective dimensions of the psychotherapeutic processes operate. Psychotherapy may be CARE writ large. The key to an effective "therapeutic alliance" may reside in higher-order empathic resonances, the foundation of which may be the CARE circuitry of our brains, which is well attuned to the nuances of GRIEF and PLAY.

This leads to interesting questions such as the mental and bodily health consequences of many of the positive emotions. Does a therapist need to find those rare but precious moments when pain can be turned into joy? Can one fight off negative affects in the BrainMind by simply amplifying opportunities for positive emotions? So far, this strategy does work in animals (Burgdorf et al., 2011), and some of our own best ideas are now in clinical testing, with drugs that have emerged from taking the emotions of animals seriously (Moskal, et al., 2011). As we better understand these

brain systems, we may be better able to devise more effective clinical interventions that allow various positive social emotions to strengthen psychological resources and help reframe troublesome memories—to reconsolidate psychic pain within the balm of positive affects.

Diverse new therapeutic insights may arise from understanding the emotional tools that evolution has given us to promote survival. Emotional disorders are invariably tied up with one or more of the basic emotional systems. Clearly, the negative emotions (RAGE, FEAR, GRIEF, and depleted SEEKING resources) are commonly encountered in psychiatric problems. Excessive positive emotions, which can also lead to problems, are equally important in framing new therapeutic approaches. Although current evidence for relationships of emotional primes to psychiatric problems is inferential, here are thumbnail sketches of some likely clinical implications, stated baldly, without extensive analysis, for each of the systems described in this book. It is impossible to sustain any crystal clarity in this narrative, since there is no generally accepted scientific nomenclature for key issues, and all systems interact with each other and higher BrainMind functions. Thus, so much about the underlying functional details remains to be scientifically documented. Indeed, this whole chapter is offered as food for thought:

1. SEEKING urges, which participate in all emotional arousals, invigorate and guide our search for resources. Along with higher brain functions, this system energizes dimensions of life-span development that are devoted to the human search for self-identity and meaning in life. Excessive and imbalanced arousals of this system can promote a variety of addictive behaviors and lead to delusional thinking and the paranoid tendencies—a "too muchness" that often characterizes mania. When aroused, in yet poorly understood ways, it can promote creativity, and when excessive, florid schizophrenia and megalomania. When this system is drained of resources, anhedonia and depression ensue. Recruitment and positive utilization of this mental energy can help alleviate depression, for a while. Thus, this system figures heavily in many positive and negative psychological outcomes. On the positive side, it promotes growth-enhancing engagements with the world, and on the negative side it promotes addictions, obsessive-compulsive disorders, schizophrenia, mania when overactive, and an empty, fatigued depression when the system becomes chronically dysregulated or underactive. For instance, part of the psychological sluggishness of depression is due to excessive dynorphin-induced dysphoria that arises from suppression of SEEKING drive. In contrast mild stimulation by opiates, or endogenous opioids, can gently stimulate this system, produce pleasurable and satisfying feelings, and inhibit various negative affects. Hence, new drugs that can antagonize dynorphin while promoting mu opioid activity, as can be achieved with buprenorphine, should be highly effective antidepressants, especially when psychic pain is prominent (Watt & Panksepp, 2009; Panksepp & Watt, 2011; and see Chapter 9).

2. RAGE looms large in societal issues when groups or individuals attempt to restrain the wishes and aspirations of others. Sustained arousal of this system leads to chronic irritability and explosive aggressive disorders. Anger is also part of everyday frustrations in human interactions when conflicting beliefs and goals clash between individuals and the larger community, even to the point of promoting schizophrenic delusions. RAGE, like all other emotions, requires the SEEKING system to be engaged. Psychotic rage is one of the hardest emotional problems to treat. As with all forms of destructive anger, males wreak most of the havoc in all mammalian species, except perhaps hyenas (because females often have more testosterone). Among humans, practically all mass murders have been perpetrated by men. Because Substance P is a clear facilitator of defensive RAGE in animal models, the pharmacological blockade of this system could prove to be a powerful antianger agent in humans, especially if it is accompanied by a reduction in RAGE-inducing frustrations in the patient's social environment.

3. FEAR (and the resulting diverse anxieties that it generates) is the perennial companion of many. This system promotes specific phobias as a function of learning, and generalized anxiety disorders when underlying brain substrates have become chronically sensitized. This system was designed to counter predation—a defense against injury and premature death (perhaps like RAGE, thus leading to the historical conflation called the "fight or flight" response). A progressive aspect of civilization has been the construction of safety nets against such events (from police forces and armies to healthcare systems). Now, however, with the push for individuality and libertarian independence in some nations, we are faced with increasing levels of novel, nonlethal cognitive predatory practices that rend the social fabric and erode the secure bases that support people's lives. Hence FEAR is once again on the rise, as are the ranks of those seeking to profit from it. Clinical anxiety abounds not only among those in the population who face the insecurities of increasingly challenging daily lives, but also among those who have experienced the most intense traumas that human life holds in store. Treatment of Post-Traumatic Stress Disorder (PTSD) in our veterans' medical centers has, lamentably, been a growth industry for many years now. Our understanding of the neural mechanisms of learning and memory is now opening the path to direct pharmacological intervention in these processes, potentially speeding the course of psychotherapy. Currently, effects of psychotherapy can be promoted by cognitive facilitators such as d-cycloserine, which actually speeds therapeutic relearning, as in exposure therapy. Successful human trials that have used d-cycloserine to promote psychotherapeutic treatment of anxiety disorders such as PTSD have been conducted within the past decade (Ganasen et al., 2010; Heresco-Levy et al., 2002; Hofmann et al., 2006). Work with animal models has also shown that it is possible to counteract fearful or traumatic memories pharmacologically to make them less psychologically troublesome (Adamec & Young, 2000; Adamec et al., 2005).

4. LUST is a common affective "companion" of young adults and eager adolescents, and it can be both a positive and negative force in human relations. Unregulated arousal of this system may participate in various anti-social behaviors, from unwanted sexual advances to predatory pursuit and stalking of objects of desire. The persistent SEEKING action urges that accompany LUST should also be recognized as vitally important for dominance relationships and determining whose will prevails in social interactions. Significantly, the quality of adolescent and adult socio-sexual behavior may be molded by the quality of early social-play relationships. The challenge is to manage these energies in ways that optimize health and well-being outcomes, rather than allowing them to be a major source of life frustrations. As this chapter was being written, it was reported in the news that a middle-aged computer programmer, extremely lonely and seething with lustful rage over his socio-sexual failures, had

murdered three women and shot nine others before committing suicide at a health club in suburban Pittsburgh. In a well-planned and hence intentional act of violence against women, he acted out his frustrations at not being able to attract female sexual companionship over a period of two decades. Perhaps this lost soul had very little social play as a boy and did not learn how to approach other humans in interesting, friendly, and non-threatening ways. That the evolution of LUST and positive SEEKING could provide a primary-process platform for the evolution of CARE and PLAY is hard to believe from such inhumane actions. However, each primary emotion helps engender idiographic higher-order personality structures that are not intrinsic to the emotional primes, highlighting the importance of learning and culture in how each is manifested.

5. CARE is the great gift of Mother Nature that helps promote lifetime resilience, and it increases the likelihood of lifelong happiness. Without CARE, humans cannot thrive interpersonally. Sociopathic and psychopathic tendencies may thrive when this motivation is deficient. And psychotherapy is virtually doomed to failure without it. The roles of endogenous opioids and oxytocin loom large in all social emotions, and it is possible that the optimal therapeutic environment needs to recruit such neurochemistries. Facilitation of opioid activity in the brain can alleviate many of the most severe forms of depression rapidly (Bodkin et al., 1995). It is possible that a substantial amount of depression is caused by diminished pleasure chemistries in the brain (Watt & Panksepp, 2009). CARE surely figures heavily in the emergence of empathy as higher BrainMind regions are molded by prosocial concerns and perspectives.

6. GRIEF signals social need. Those who do not get enough care will be subject to elevated GRIEF and psychic pain both in the present as well as later—their lives are more likely to be full of separation distress, resulting in chronic feelings of insecurity, sadness, and the inability to experience pleasure. The chronic insecurity of borderline personality disorders and social anxiety disorders may arise from this all too common negative affect. Excessive activity of the GRIEF system can promote a host of other psychiatric problems ranging from depression to social phobias and panic attacks; chronic under-activity of this system may promote maladaptive attachment styles as well as autistic and psychopathic aloofness. But above all, this system promotes feelings of sadness and grief, which can become chronic psychic pain (Panksepp, 2011a). Many common forms of depression are precipitated by neurochemical cascades initiated by such feelings and by the ensuing despair within the BrainMind (Watt & Panksepp, 2009). As noted several times already, safe opioids such as buprenorphine are much underutilized in the treatment of depressions that do not respond well to conventional antidepressants. But there are good reasons to believe that these drugs will have optimal long-term therapeutic effects only if they are accompanied by improved social attachments, including temporary attachments to psychotherapists (whose tasks include the facilitation of new real-world perspectives on life-affirming social possibilities).

7. PLAY networks give us perhaps the most evolutionarily recent primary-process emotional urge: the urge to engage creatively and joyously in the mental world of others, to establish friendships and to learn through eager friendly competition (with repeated, adequately balanced give-and-take—winning and losing, in a manner of speaking). This process, when done with full acceptance, is often accompanied by euphoric positive affect, whereby one feels a sense of secure belonging within the social order. It is sad that this natural rambunctious behavior of young children is too often viewed as a problem by parents and other adults, leading perhaps, in situations where childhood impulsivity is most disturbing, to diagnoses such as ADHD at one phase of life and mania at another. But PLAY is also a blessing for the development of social skills. Like all the basic emotions, PLAY is an especially rich experience-expectant

> process that energizes a great deal of learning, ultimately serving as the driving force, along with the social emotions of GRIEF and CARE and LUST, of much of the world's artistic production. At its best, PLAY is permeated by one of the greatest joys of life: the capacity to laugh, one of nature's finest emotional gifts. And this blessing is not unique to humans. Even rats exhibit a happy laughter-type sound when they play with each other or are tickled by the playful hands of human beings, yielding measurable brain benefits (Burgdorf et al., 2010; Wöhr et al., 2009; Yamamuro et al., 2010). Primary-process social joy can be molded, with a keen sense of fun and humor, into distinct therapeutic interventions that can probably counteract chronic negative affects such as repressed RAGE, and with remarkable clinical skill, perhaps even quell the demoralizing effects of profound FEAR and GRIEF. Perhaps PLAYful joy is much underutilized in psychotherapies, especially among the young. Indeed, there are solid reasons to believe that it can be used as a daily psychic tonic for young children to help reduce the ever-growing diagnosis/incidence of ADHD in our culture. Our increasing understanding of this emotional system should figure more heavily in the discussion of diverse emotional problems, including depression, since facilitation of PLAY urges should be a key target for future therapeutic interventions.

In sum, these systems need to be considered in diverse mental health issues, only touched upon here, and their detailed neuroscientific understanding may be of foremost importance for the development of new psychiatric systematics and ever more specific BrainMind medicines. They are all brought together into a coherent symphony of bodily actions and affective possibilities through an ancestral core-SELF structure, shared homologously in all mammals. In higher brain regions, this unity can be lost as diverse idiographic tertiary process selves are engendered, providing the neural soil for dissociative identity (or multiple personality) disorders.

We must remember that at the foundation of mind, all these systems are enactive, action networks. Emotionally troubled people should be allowed to enact their energies at appropriate moments, and therapists should be trained to recognize when emotions are real, and how one can, with the understanding of the affective depths of humanity, help transform a negative affect into a positive one.

While psychotherapy must obviously continue to deal realistically with the *holistic*, multitiered appreciation of individual human lives, we are finally entering an era when an understanding of the nomothetic (universal) *parts* of the MindBrain—evidence-based primary-process affective views of the mammalian brain—can provide an understanding of the universal affective-emotional *foundations* for the MindBrain shared by all humans. In turn, this knowledge should provide a scientific grounding for new affectively oriented therapies that concurrently consider the interactive dynamics of body, mind, and brain. Future generations of psychotherapists

will be well served by developing skills and understandings at all of these levels.

The rest of this chapter will delve into implications of such knowledge, not only for our understanding of the foundations of a positive emotional life, but for promoting psychotherapeutic healing and life-affirming cultural initiatives. An understanding of positive emotions lies at the heart of what it means to live a "good life" (Sheldon et al., 2011). As we understand more about the neurochemical coding of brain affective processes, new ideas are bound to emerge for the treatment of excessive anger, anxieties, depressions, phobias and traumas, and perhaps even psychotic delusions (Panksepp & Harro, 2004; Watt & Panksepp, 2009). I do not claim psychotherapeutic expertise but feel it is incumbent on me to entertain various possibilities that thoughtful neuroscience can bring to the therapeutic table. I encourage clinicians to develop these ideas further.

One of the most striking and highly replicable rediscoveries of recent decades has been the remarkably powerful influences of early childhood experiences on future mental health (Heim, et al., 2010). There are findings too numerous to summarize here, but they range from physical risk factors such as premature births and drug exposures (e.g., Johnson et al., 2010; Stone et al., 2010) to the quality of mother-infant and child relationships (Fearon et al., 2010; Lahey et al., 2008). Children who are abused may develop chronic anger and diverse psychosomatic symptoms. But just as in rats, abundant loving maternal attention promotes resilience and better-regulated stress responses as children grow to adulthood (Lester et al., 2007; Propper et al., 2008).

It has long been a matter of debate whether one can "cure" such patients simply by dealing with their current life circumstances. Some feel it is essential to deal more directly with the emotional traumas of the past. For instance, Art Janov's (2007) position is that there are ways to guide people with early emotional traumas back to the sources of their implicit traumatic memories. He argues that special reliving techniques, within appropriate supportive and understanding therapeutic contexts, can permanently ameliorate the impact of those long-lasting brain changes. This would be an example of a novel ABT, but it remains to be fully documented in standardized therapeutic trials.

There is an enormous potential for the development of other novel strategies, both psychological-behavioral as well as combined somatic and

neuropharmacological interventions. Development of these approaches can be guided, in part, by what we are learning about our ancient primary-process emotional systems. I will now consider how such knowledge might have useful implications for the development of new therapies but also for the evolution of psychiatric taxonomy, such as the codification of psychiatric syndromes, based directly on underlying endophenotypic emotion issues (Panksepp, 2006a).

EMOTIONAL ENDOPHENOTYPES VERSUS SYNDROMAL THINKING IN PSYCHIATRY

Research on the animal brain allows us to focus on primary-process or core affective issues in *neuroscientific detail*—something that is quite impossible in human research, even with modern brain imaging (which is better suited for the higher cognitive regions that interest most psychologists). Animal research allows us to go to the affective core. Cross-species preclinical research allows us to arrive at ever more coherent understandings of the nature of emotional illnesses and the affective requisites of mental health. Currently we rely heavily on descriptions that try to categorize mental illness on the basis of general symptoms (i.e., the *DSM*-type "syndromes"). Let us briefly consider these existing diagnostic categories as they are encoded in the major handbook of psychiatry—the various successive versions of the *Diagnostic and Statistical Manual* put out by the American Psychiatric Association over the past half-century. Current psychiatric diagnostics are based on concepts that are passed down to us by pioneers such as Eugen Bleuler, Sigmund Freud, and Emil Kraepelin.

These conceptual psychiatric syndromes were never based on an understanding of the brain or its emotional systems. This is one reason why outdated concepts have caused increasing problems from *DSM-I* to *DSM-IV*. Many fear that the current construction of *DSM-V* will not be immune to those flaws (Hyman, 2007). Such problems might be alleviated if we could replace (or at least supplement) the old conceptual structures with better visions of the real psychiatry-relevant entities of the mind (e.g., the various *endophenotypes* of the emotional brain).

Endophenotypes are natural aspects of brain functions that can be studied at the neuroscientific level; for instance, researchers can examine responses as simple as eye-blinks, pupillary dilation or constriction, and startle

reflexes (Gottesman & Gould, 2003). We know that loud noises invariably startle people and animals. However, when exposed to the same noise a second time, the startle response is less pronounced. This phenomenon, when studied with mild auditory stimuli that allow animals to prepare for much louder sounds, is known as "pre-pulse inhibition", and is often attenuated or absent in schizophrenic individuals. We can be confident that these preparatory, regulatory responses are controlled by coherent, analyzable circuits within the brain (especially basal ganglia), although we can have no such confidence for psychiatric *syndromes*, because those are concepts created by human insight and ingenuity.

Through affective neuroscience, endophenotypic thinking (acknowledged almost universally as an important new way of approaching psychiatric science) can include the domain of primary-process emotions. The natural emotional networks of the brain may provide the most relevant endophenotypes of all, because they go to the affective core of psychiatric matters. Further developments along these lines may help us cut through the Gordian knot that has arisen from the once-revolutionary syndromal thinking of previous eras (Panksepp, 2006a).

The current problem is highlighted best by the most prevalent psychiatric problems, including various types of depression and other mood disorders. As noted in the introduction to the *Textbook of Biological Psychiatry* (Panksepp, 2004, p. 18): "*DSM-II* had only 8 types" (of mood disorders), "but by *DSM-III* (Revised) there were 97, and according to Paul McHugh (2001), if you consider all the subcategories and specifiers in *DSM-IV*, one could categorize 2665 subtypes." Such complexities arise from the higher levels of MindBrain organization that can vary enormously among individuals. Magnification of diagnostic minutiae does not offer any clear linkages to solid brain research or therapeutic practice, and many have started to hunger for a different approach to categorizing and dealing with psychiatric problems. The possibility of building a diagnostic taxonomy around emotional endophenotypes is just one, but currently the most robust, scientifically based vision for the future of psychiatry. Among other popular approaches right now are those based on genetic underpinnings, but those linkages are not yet yielding much clarity—only susceptibility factors, and many "linkages" of unknown significance. In any event, practitioners should clearly envision the natural primal emotional systems of the

mammalian brain, and they should conceptualize human emotional problems, at least partly, in those terms.

This can lead to novel types of psychiatric systematics along with new ideas for experimental psychotherapies (a few have been noted, and more will be noted later). But to achieve such progress, we also have to develop new approaches to preclinical (animal) research that are of clear psychiatric relevance, focusing on the diversity of primal affects that lend themselves to empirical evaluation. Cross-species evolutionary perspectives will be invaluable in such pursuits. For instance, one integrative idea that has permeated this book is that imbalances in the GRIEF and SEEKING systems may be major influences in the genesis of depression (Watt & Panksepp, 2009; Panksepp & Watt, 2011). The GRIEF system promotes psychic pain that characterizes depression. When SEEKING energies diminish, as they seem to do during all sustained negative emotional states, a chronic dysphoria and deep psychic fatigue and emptiness sets in, which may reflect the active inhibition of SEEKING urges, or simply the depletion of those energetic-euphoric resources.

These lines of thinking are presented because they allow totally new approaches to preclinical modeling of psychiatric disorders. For instance, in modeling depression, we may no longer need to impose massive negative stressors on animals, such as the commonly used persistent stress of social defeat, continuous variable stress or repeated unpredictable stressors. Rather we currently know enough to try to induce imbalances of specific underlying emotional networks (Wright & Panksepp, 2011). Likewise, instead of using very general and nonspecific measures of depressive affect, such as measuring despair (giving up) in forced swimming tasks, or diminished struggling of mice suspended by their tails, and so on, we can invest more wisely in direct measures of internal affective states by monitoring affective vocal responses to systematically applied stimuli (such as tickling procedures) that probe the status of relevant positive affective systems (e.g., happy 50-kHz ultrasonics in rats), while also monitoring relevant negative affective responses (e.g., distressed 22-kHz “complaints”) that can be gently induced by the application of a puff of air to the back of a rat’s neck. It is truly remarkable that currently we have abundant animal models of psychiatric disorders with no serious conversation or evaluation of the affective processes within their brains. This is surely slowing progress on understanding many basic issues directly relevant to human

psychiatric concerns. We can do better by taking primary-process affective circuits more seriously.

ANIMAL MODELS, PSYCHIATRIC SCIENCE, AND THE FUTURE OF DIAGNOSTICS

The coherent blending of basic affective neuroscience, psychiatric diagnostics, and clinical practice has barely begun. Although there is abundant enthusiasm for a new synthesis, solid bridges among the various possibilities remain to be built. For historical reasons, traditional preclinical research is still wedded to a behavioral model in which measurable actions and body chemistries count, but emotional feelings do not.

A behavioral approach to psychiatric research sees visually explicit symptoms (e.g., sustained immobility in the Forced Swimming Test) as the end point, the prime indicators of mental illness. Steven Hyman (2007), the former director of the National Institute of Mental Health, was especially critical of existing inadequacies of animal models used to simulate human psychiatric syndromes. But he failed to note that they may be "unsatisfactory" largely because investigators of basic neuroscience models rarely use affective concepts to guide their thinking. Because disturbing and imbalanced affective experiences serve as the triggers for many mental illnesses, the more explicit recognition of mammalian brain systems that generate affects should be helpful. If we devote more effort to studying affective changes, then our animal models may provide far better insights into what may be happening in brain systems that are prime regulators and sources of mental illness.

For better models, our primary goal should be to characterize the brain anatomies, physiologies, and neurochemistries of the unconditional primary-process emotionality of other animals by using neuroethological approaches—the neurology of natural emotional behaviors—and then to utilize that knowledge to understand the emotional imbalances of human and animal minds. By directly manipulating specific emotional circuits, one has the possibility of dissecting the most important influences on long-term mental health outcomes. One reason such furrows of understanding have not been cultivated more vigorously is that the emotional feelings of animals have been marginalized by the same scientists who are best positioned to do substantive work on such critically important topics.

Perhaps consideration of the relevant truth diagram once more (Figure 1.5) may help clarify where we should currently be in our thinking about raw affective feelings in other mammals. The evidence is overwhelming that all other mammals feel primary-process affective states intensely, but at present, relatively little work is being conducted to characterize these affective systems in preclinical models.

TOWARD AN INTEGRATION OF LEVELS OF CONTROL: AFFECTIVE THERAPEUTIC PERSPECTIVES

In humans, it is not unusual for affectively charged thoughts that accompany emotional arousals to be rendered unconscious soon after the emotional arousals subside. In other words, the reflective *ideas* that are churned up in higher regions of the mind while one is intensely emotional can rapidly become *cognitively* unconsciousness once the affective storms have passed, often remaining dormant, until primary affects are once again re-aroused. It is difficult to bring the flow of ideas and images associated with emotional arousals explicitly back into phenomenal experience once passions have subsided. This is why reestablishment of primal affects in therapeutic environments can be a rapid pathway to change. Because affects energize and guide the cognitive apparatus, clinicians can deal directly with the associated ideas and ruminations that spill forth readily, allowing therapists to directly understand maladaptive emotional patterns in action, which provide ideal moments to remold their power over each patient's mental apparatus.

At workable moments like that, new therapeutic interventions may be applied that allow clinicians to "soften" the painful edges of past experiences through our emerging understanding of memory "reconsolidation" (see Chapter 6). When memories are retrieved, they can be modified, reprocessed, and hopefully stored away again in less disturbing forms. Indeed, there are reasons to believe that if countervailing positive emotions can be aroused at moments of emotional crisis, the long-term influences of troublesome memories may be diminished.

Before proceeding, however, let us briefly acknowledge just some of the strands of history in this field that have followed similar paths. There is the primal therapy of Arthur Janov already noted (*not* the "primal scream therapy" of popular, sitcom myth) and also the more conservative but

highly effective and emotionally oriented process-experiential approaches of Leslie Greenberg and colleagues (see Elliott et al., 2004), as well as the short-term experiential dynamic therapies of Habib Davanloo (2005) and David Malan (1979, 1999) and others, where a main goal has been to get people to experience their "true feelings" intensely enough to allow sustained modifications.

It is becoming clear that dynamic emotion-focused approaches are generally highly effective in promoting lasting therapeutic change (Abbass et al., 2006), often yielding more lasting results than cognitive-behavioral reinterpretations and restructurings of higher mental functions. Affective experiential approaches are commonly based on the perspective that troublesome emotions can rapidly energize relevant thoughts more than cognitions can instigate relevant feelings, which can be beneficially used, at least in supportive therapeutic environments. The intense re-experiencing of emotional episodes opens up new treatment possibilities because it provides therapists an emotional "closeness," especially within a secure therapeutic alliance, that is optimal for therapeutic change. The impact of affectively troublesome memories can be reduced by being reframed with affectively positive perspectives.

Emotion-oriented therapies appear to work remarkably effectively because they address with immediacy the relevant primal affects—thereby bringing forth the most relevant associated cognitive materials from the higher regions of the mind. It is not unusual for clinicians to hear, "My mother has always demanded too much of me, and treated me like I was never good enough"; "I was smaller than the other children, and I still feel demeaned and insecure today"; "I have been fighting this all my life, and I still cannot forgive myself." The personas that might emerge from such clinical storylines are readily recognizable: the resentfully obliged and dutiful child; the harried, self-doubting overachiever; the sad, frustrated, self-sabotaging ne'er-do-well. Such archetypal storylines are, of course, virtually as familiar as one's own life story. The affects they reflect are often counterproductive for long-term emotional well-being, for they reach to the imbalanced affective foundations of people's mental lives. But this is exactly where the work of therapy needs to be applied, hopefully modifying the sustained and affectively powerful learning patterns that have made negative states of being habitual.

AFFECTIVE BALANCE THERAPIES

Fortunately, affective issues are currently at the forefront of scientifically informed therapeutic thinking. At present, some of the most interesting discussions in psychotherapy are emerging from new interdisciplinary frontiers: (i) developmental social neuroscience (Schore, 2003a, 2003b; Siegel, 2010; Stern, 2004); (ii) an emerging neuropsychoanalysis (Solms & Turnbull, 2002); (iii) a human and cross-species affective neuroscience (Davidson, 2004; Panksepp, 1998a); and (iv) visionary perspectives on regulatory processes of the autonomic nervous system (Porges, 2009a). These approaches are finally grappling with the emotional nature of the human mind and also, at times, with the deep affective nature of the mammalian brain.

Dan Siegel said it well in the foreword to Louis Cozolino's (2002) synthesis of clinical and neuroscientific approaches to the human mind: Clinicians, he said, immerse themselves "in the stories of individuals who come for help in feeling better. . . . Whatever the approach, lasting change in therapy occurs as a result of changes in the human mind . . . which involve changes in the functions of the brain. Exactly how the mind changes during the therapeutic process is the fundamental puzzle that the synthesis of neuroscience and psychotherapy seeks to solve." Perhaps the most critically important neuroscientific piece of the puzzle is how emotional feelings emerge from the brain and how chronic emotional feelings can change. Providing an introduction to this knowledge has been the aim of this book.

There is ever-increasing interest among psychotherapists about the neural nature of affects, their embodiment in actions, and how they interact with cognitive processes. There is intense engagement with the topic of how emotional affective states can be better used to remold the affective well-being of people in distress (e.g., Fosha et al., 2009a). Although psychotherapy has traditionally sought to deal more with the cognitive aspects of the emotional labyrinths of individual lives, a few revolutionaries are moving the discussion toward key affective issues (e.g., Fosha, 2000; Greenberg, 2002; Greenberg & Watson, 2005; Hughes, 2006, 2007; Ogden et al., 2006; Schore, 1994, 2003a, 2003b; Siegel, 2007, 2010; Stern, 2004).

The role of insecure early social attachments is an especially prominent vector in derailed affective lives (Heim, et al., 2010). As a result, some

revolutionary therapists are aspiring to retrieve the early implicit affective "memories" that have been engraved in developing neural matrices, which control primordial mind states in infants, and work with them directly by using nonverbal forms of "primal healing" to mend the residual psychological "wounds" that carry through to adulthood (Janov, 2007). Such attempts to deal directly with the earliest childhood traumas—reflected in both sensitized and desensitized emotional systems—are seeking to expunge the implicit residues of intensely experienced emotions from the memory banks of the brain. Many patients report remarkable benefits when encouraged to relive these early traumas. We need to consider how such therapeutic models might be integrated with our emerging understanding of the ancient, universal emotional principles of the mammalian brain. There are no clear answers here, but again the idea of a "reconsolidation" of past traumatic experiences in the context of therapeutic CARE, perhaps even PLAY, comes to mind.

Because we can now grasp the neurodynamics of primary-process emotions, we can envision new variants of ABTs that currently lie on the horizon. We should perhaps even consider long neglected ideas such as simply attempting to fight negative affects with the healing power of positive ones. Possible examples include affectively oriented therapies that not only aim to get at the emotional lives of individuals directly and rapidly, but also to utilize various somatic therapies that use the qualities of the body-to-brain-to-body continuum to rapidly shift moods toward positive affect. Every emotion has such linkages. When a patient's emotional action apparatus has become rigidly "frozen" into a negative affective state, might it not be wise for therapists to initially encourage the types of movements and body repositioning that allow the mind, brain and body to shift into different emotional states? To shift, hopefully toward more flexible positive feelings where different affective perspectives can be considered. For instance, might playful interactions, along with direct bodywork, yield more rapid progress at times than remaining just at the cognitive level of interaction (Ogden et al., 2006)? Do we need to consider all the levels of BrainMind organization, from primary to tertiary, for optimal therapeutic progress? How else could it be, if the evolutionarily more ancient affective processes guide how the higher mental apparatus operates (Figure 2.3)? We need to concurrently think about both body and brain, body and mind, to deal most effectively with emotional problems.

Nowadays, researchers and mental health professionals are increasingly interested in understanding the neuro-affective imbalances and disorders that underpin psychopathology, along with the MindBrain changes wrought by psychotherapy; these processes can be envisioned with contemporary brain-imaging and other neuroscience technologies. Since those issues are well covered elsewhere (e.g., Cozolino, 2002, 2010; Doidge, 2007), my main goal in this chapter is to focus on our emerging understanding of how affective feelings are generated and memories are consolidated within the brain to promote further advances in clinical interventions.

Although we are finally in an era where most thoughtful investigators are in agreement with Freud's belief in the biological and affective foundations of the psyche (Freud, 1937/1968, p. 357), the kinds of fairly straightforward affective BrainMind dynamics envisioned in this book remain to be widely integrated into therapeutic thinking. This is partly because of historical barriers, some which have already been discussed. But there are many others. Indeed, Freud's own psychoanalytic metapsychology, perhaps "enriched" by too much conceptual baggage (e.g., Oedipus complex, penis envy), could be used as an example. It was creatively constructed from limited, culture-bound clinical observations, leading to a less than favorable historical trajectory. Across the years, many problems have arisen from an excess of theoretical creativity, accompanied by too little solid understanding of the evolutionary layers of brain and mind.

The resulting Gordian knot cannot be completely untangled, but we can entertain how affective neuroscientific knowledge can serve as a new, and hopefully solid, foundation for future clinical thinking. The main lesson is that emotions across individuals are most similar at the primary-process level; they get diversified individually through learning and memory. And at tertiary process levels they will vary most of all. Well-targeted pharmacotherapy may be most useful at the primary level, especially since that can also have strong influences on all higher levels. Behavioral therapy approaches will work optimally at secondary-process levels, and cognitive approaches may be very effective at levels of thoughts and ruminations, with effective procedures having beneficial regulatory effects all the way down. New dynamic affective balance approaches can tackle the whole package effectively.

CONSCIOUS AND UNCONSCIOUS PROCESSES IN THE BRAIN AND PSYCHOTHERAPY: PUTTING THINGS IN PERSPECTIVE

Although arousals of the primary-process emotional networks of mammalian brains are intensely experienced by humans and other animals, it is especially important to recognize that the secondary processes of the BrainMind, the basic forms of learning, memory and habit formation, are among the most unconscious "mental" processes of them all. Once we understand this, then many of the bizarre and faulty views from psychology's past may be rectified. For instance, "free will" is not a figment of our imagination as too many scientists are ready to claim these days. Free will is a higher tertiary-level neurocognitive function that we use on a regular basis (and quite effectively when we are not too emotionally aroused) for planning future actions. This is brought out beautifully in the concept of "autonomy" and "self-determination" as developed by Ryan and Deci (2006). However, we cannot readily will ourselves out of underlying emotional turmoil that has been created through the consolidation of maladaptive affective patterns at primary and secondary levels of MindBrain organization. At primary-process levels of emotional processing there is no free will, there are no "controlled cognitions." Neither do the automatic secondary-process learning and memory functions, that are molded by our wild animal passions developmentally, exhibit free will. That can only emerge from well-sculpted, deeply self-reflective, cognitive attitudes.

Our primal emotional needs and bodily motivations shape who we become before we know—before we become "aware of"—what is happening cognitively, often yielding end results without our "personal" consent. Thus, it is important to recognize that our raw, affective phenomenal experience of emotions and our cognitive reflective awareness of our emotions are very different types of mental processes. For rapid therapeutic change, perhaps it is often the affective experience itself that needs to be the starting point. But affective experience has been one of the greatest problems of neuroscience, little talked about, and hence resistant to empirical understanding . . . indeed, even to attempts at cogent scientific analyses. One of the main goals of this book has been to provide an introduction to how that can be changed.

The Tortuous Path to Understanding Basic Emotions: Our Inherited Tools for Living

Why has a detailed neuroscientific understanding of the mechanisms of affect generation been so slow to emerge? Partly because it could *only* arise from the kind of detailed brain research that is quite impossible to conduct in humans but increasingly feasible in animal models. Progress has been delayed further by traditional conservative biases against the use of primary-process mental constructs in the scientific analysis of neural controls in the analysis of both animal and human behavior. Accordingly, basic emotional networks and the affective feelings they generate have simply not received the attention they deserve. And these feelings commonly continue to be neglected by those best situated to reveal their neural infrastructure (i.e., by behavioral neuroscientists), and hence they are not as clearly evident in modern biological psychiatry discussions as they need to be.

In a sense it is tragic that most investigators interested in learning and memory who so effectively use classical fear-conditioning as their main methodology (see Chapter 5) do not yet explicitly acknowledge the existence of an unconditional FEAR system in the brains of the animals they study (Panksepp et al., 2011). The amygdala generates emotional behaviors and associated autonomic responses, but they are typically portrayed as mere unconscious motor "outputs" in animals (Davis et al., 1995, 2010; LeDoux, 1996), as opposed to affect-generating emotional systems. This shortsightedness has prevented those interested in fear-learning from envisioning that the "unconditioned fear responses"—namely arousal of the FEAR system—may be critically important for the genesis of fear-learning (see Chapter 6). As soon as we realize that this circuitry is also the locus of control for anxious feelings, we can envision how memories become fearful. Even more, we may begin to actively consider how various positive affective circuits may counteract such psychological negativity, hopefully yielding ways for troublesome memories to be reconsolidated in more acceptable affective frames of mind.

For new therapeutic advances, we need to understand how sustained arousal of the unconditioned FEAR system critically contributes to the genesis of chronic anxiety disorders (Panksepp, 1990b; Panksepp et al., 2011). New treatments for anxiety should aim to dampen the psychic

influence of this system, whether by pharmacological desensitization of the FEAR system or by psychotherapeutically defusing fearsome memories. This can currently be achieved by the direct pharmacological reduction of the arousability of the FEAR system, as with benzodiazepines and to a lesser extent arousal (e.g., brain norepinephrine) inhibitors—"beta blockers" such as propranolol. Such agents can reduce the impact of troubling memories that normally arouse this system, and may allow psychotherapy to strengthen the "muscles" of counteracting positive affect systems. In considering such options, it is always important to keep in mind the levels of control in BrainMind evolution: The FEAR system promotes anxiety-laden memories, resulting in troubling thoughts, that are unique from individual to individual. However, the learned anxieties that arise from the primal FEAR system are bound to be quite similar across humans and other mammals.

Because of advances in genetics and neuroscience, it is now clear that animal models can promote an accurate archaeology of many of those ancient affective principles that still control human lives. Thus, a cross-species affective neuroscience has helped elucidate the many subjectively experienced primal emotional feelings that are among the evolutionarily provided whips and carrots—the affective tethers and guides—for our endlessly complex cognitive abilities. Ultimately, much of animal and human learning is closely linked to how certain courses of action make organisms feel. Thus, the negative affective tethers that come to impair mental health need to be countered with the affectively positive guides that can promote happiness: These can range from fantastically imaginative and creative thinking (promoted by our SEEKING system) to ethical and moral decision-making (promoted by all our prosocial emotions—CARE, GRIEF, and PLAY). And if we understand the neurobiological nature of these feelings, and how they control learning, we may have the beginning of a solid neuroscience of what it means for the human mind to experience positive emotions, and hence better ways to counter emotional disorders, through explicit, affectively beneficial clinical interventions. Modern brain imaging will help in these endeavors (e.g., for overview, see Cozolino, 2010; Northoff, 2011). But at the same time, we should be realistic about the anatomical and functional limits of such techniques.

AFFECTIVE NEUROSCIENCE, BIOLOGICAL PSYCHIATRY, AND PSYCHOTHERAPY

In our current era of brain imaging, the ancient regions of the emotional brain have received less attention than neocortical functions, partly because of the greater sensitivity of the techniques for large, highly firing neural systems. The result has been a focus on the cognitive regulation of emotional processes. Relatively small, slowly firing neuronal brain regions (where chemistries released are more important than the frequencies of action potentials) are not as readily visualized by these techniques. However, these neurophysiologically "sluggish" lower brain regions are of decisive importance for our emotional lives.

As I have previously noted, the subcortical localization of basic emotional systems has been dramatically confirmed by the fact that investigators can surgically eliminate all of the neocortex at birth in various "simple" experimental animals, and the subjects grow up to be seemingly normal creatures as far as their basic sets of emotional energies are concerned. They exhibit exploratory urges and seeking behaviors, fear, anger, lust, maternal care, and playfulness. The last is especially surprising, because physical play is such a dynamically flexible behavior. Similar patterns have been observed in human children born without a neocortex (Shewmon et al., 1999; see Figure 13.2).

When adults have similar brain damage, functional impairments are much greater, perhaps largely because once primal urges are cognitively rerepresented within maturing neocortical areas, both humans and other animals come to rely ever more heavily upon those higher, developmentally programmed "software" functions. Once one has started to rely on those fine new cortico-cognitive tools for higher forms of consciousness, one cannot effectively return to simpler ways of being. Whether the neocortex has *any* evolutionarily based affective functions, as opposed to learning-dependent development, is currently unresolved. It can surely engender a host of emotional thoughts and behaviors. Still, it seems, the epicenters for emotional affects remain subcortical, even though ancient cortical areas such as insula can generate various specific sensory affective feelings such as disgust and pain (Craig, 2002, 2009), but surely not without participation of subcortical circuits. Orbitofrontal areas participate in many negative and

positive feelings related to taste, temperature and other sensory rewards and punishments.

When the subcortical emotional powers of the human brain become tempestuous (or dysregulated beyond understanding), overwhelming and often lasting emotional problems can emerge. In humans, these are always accompanied by cognitive changes, such as emotionally entangled attributions, ruminations, all sorts of plans and worries, as well as cognitive "propositional attitudes" about how the world is organized. This fact helps to explain why, every time emotions occur in an intact MindBrain, there *always* seem to be precipitating cognitive reasons in the environment and cognitive consequences for the way we think and perceive the world. But affective change is foundational in most psychiatric disorders.

This view suggests that psychotherapies need to deal not only with the cognitive precipitants of emotional turmoil, but also, ever more directly, with the concurrent affective issues. Affective neuroscience suggests that some people become hyperemotional without precipitating events, because of internal brain irritations. An example is when individuals have "limbic seizures" that are caused by sensitized emotional networks (Lewis & Pincus, 1989). Severe affective imbalances can occur for purely neurobiological reasons. Such problems can be alleviated by directly manipulating the brain. Cognitive interventions would be unnecessary, although wise counseling is always useful, especially with regard to the process of readjustment that Freud called "working through."

There are also childhood traumas that leave their imprint largely on the reactivity of emotional systems, with no explicit cognitive residues (Janov, 2007). Subcortical circuits can sensitize and desensitize through experience. These may also be helped with well-targeted pharmacological interventions, perhaps without extensive time spent talking about one's life, even though focusing on how the resulting personality traits have affected one's life should provide useful insights. It is important to consider that therapists who have established a strong therapeutic alliance are in a position to steer patients into different and more positive emotional states by engaging with them at more primal levels. For instance when negative emotions are allowed to be expressed, but positive emotions can also be evoked by skilled therapists, it may be possible to explicitly guide patients gradually toward more positive emotional states, allowing lasting therapeutic changes to take root.

Obviously, chronic emotional pressures change the way that individuals respond and fit into the world. With the advent of numerous new drugs for various ailments, the psychopharmacology revolution has provided many examples where simply manipulating brain chemicals can have enormous therapeutic effects for those in relatively modest, but psychiatrically significant distress (Kramer, 2005). Indeed, most psychopharmaceuticals *are* ABTs, because, when they work well, they shift primary-process emotional responsiveness and mood in desirable directions strictly at a non-cognitive neurochemical level. But such reductions in undesired feelings, often allow cognitive perspective-taking to become more effective.

Obviously, most human emotional problems are caused by life events. Having a caring person simply listen to the full impact of emotional events, directly from the battlefield, so to speak (Belenky, et al., 1996), is therapeutic. It is known that the subjective intensity of emotions diminishes when one puts feelings into words (Lieberman, et al., 2011). Even the chronic affective problems that have arisen from explicit non-cognitive traumas are rapidly embedded in complex cognitive narratives that need to be fully communicated and explored in therapeutic conversations. As we have seen, basic affects and cognitions always form a two-way street: Emotional arousal modifies the way we think, and the way we think can modify our feelings. Much of the "everyday madness" that characterizes human relationships and tragedies needs to be dealt with on both emotional and cognitive levels. However, the sharp edges of cognitively promoted emotional dilemmas can be softened by the opportunity for "existential testimony" in the context of social support that can promote mindfulness, which is the capacity to focus on one's daily life with an equanimity that transcends one's troubles (Siegel, 2007, 2010). And playfulness, applied judiciously at just the right moment, should also help.

Indeed, emotions and cognitions work so closely in the intact human MindBrain that most psychologists are loath to distinguish between the two, a viewpoint that does not withstand close bottom-up neuroscientific scrutiny. Primary-process emotions become cognitivized—enmeshed with specific conscious representations of internal and external events—through learning. Thus, in most human psychological problems, cognitions become embroiled with primary emotions to the point where they cannot be readily distinguished. Still, affective neuroscience highlights how primary-process, pre-propositional emotional energetic states have minds of their own as

ancient forms of affective mentation that preceded language and thought by vast spans of evolutionary time. A clear recognition of such emotional energies, and their role in mental life, allows one to concurrently pursue therapeutic work at more fully integrated affective and cognitive levels.

An understanding of how primary-process emotion can derail human lives should be of value for a scientific understanding of all types of psychotherapy as well as the establishment of a new basic neuroscience infrastructure to serve as a foundation for future developments in biological psychiatry (Panksepp, 2006a, 2006b; Panksepp & Harro, 2004). Thus, affective neuroscience suggests new psychotherapeutic perspectives that may complement well-established behavioral, cognitive, humanistic, interpersonal, and mindful therapeutic traditions.

EMOTIONAL DYNAMICS AND AFFECTIVE BALANCE THERAPIES

Psychotherapy can benefit from our increasing understanding of the emotional-instinctual action dynamics of the brain. Simply enabling people to understand such universal, shared systems in their brains may be therapeutic in and of itself. In this way, emotionally troubled individuals may confront the world with more confidence and a better understanding of the universal principles that underlie emotional state generation and regulation. Cross-species affective neuroscience not only provides a coherent structure for thinking about basic human problems, but also a concrete vision of how affect emerges from the brain. Unconditioned emotional dynamics provide a scientific way to understand how primary-process emotional feelings are actually generated in the brain.

ABTs may provide novel evidence-based ways to modify emotional feelings directly, allowing clinicians to use new psychotherapeutically facilitated affective attitudes as a foundation for restructuring cognitive distortions and ameliorating the resulting intrapsychic stressors. Indeed, such affect-based interactions may enhance the effectiveness of the classic pharmacotherapies that revolutionized psychiatry in the middle of the last century, followed more recently by a variety of direct brain stimulation procedures, ranging from electroconvulsive shock and other forms of brain stimulation such as transcranial magnetic stimulation and deep brain stimulation (Panksepp, 2004). Some of these methods are able to directly

modify the affective tone of the nervous system because they act upon the primary-process emotional networks within the subcortical regions of the brain (Coenen, et al., 2011).

Although primary-process emotional dynamics emerge from subcortical brain networks that we share with other animals, they could probably be recruited more effectively in psychotherapeutic environments than is typical in current practice. For instance, it has long been known in psychological science that one can induce emotional feelings by simulating emotional actions (Stepper & Strack, 1993). Indeed, one can rapidly get emotion-typical affective changes such as joy and sadness merely by simulating the action dynamics of laughter and crying; this can even be achieved with mental action imagery (Panksepp & Gordon, 2003). Likewise, music is a powerful way to induce emotions in ways that can be harnessed for therapeutic ends (Bernatzky, et al., 2011).

How such voluntary control over our emotional expressions, and hence affective states, can be harnessed in psychotherapeutic situations remains to be systematically studied. It seems fairly straightforward to bring these affect-specific energies to bear on all varieties of experiential psychotherapy. The incorporation of highly focused emotional exercises could contribute greatly to psychotherapeutic approaches to human problems (Ogden et al., 2006), while also providing opportunities to educate people about the primary-process aspects of their emotional lives.

To gradually master one's own emotional dynamics in this way may help pave the path toward emotional intelligence and thereby homeostasis in a variety of situations. Pursued on a daily basis, positive emotional exercises may strengthen one's "emotional muscles" in ways that can counteract the effects of past traumas and inoculate the emotional circuits against future adversities. For instance, when negative emotions are aroused in therapeutic environments, they could be followed on a regular basis by various positive affects—from emotionally powerful musical excerpts to bodily expressive movements, rich in positive affect. A great deal of basic science needs to be done to evaluate the efficacy of such novel techniques, both in the context of existing body therapies (Ogden et al., 2006), as well as part of an emotional education program that may help provide prophylaxis for emotional extremes that might otherwise cascade into major psychological problems. Understanding the dynamics of one's own emotions, as part of a

comprehensive therapeutic program, may help reduce the incidence of stress-induced psychological problems.

AFFECTIVE BALANCE THERAPIES IN CONTRAST TO TRADITIONAL PSYCHOTHERAPIES

Assimilation of the complete spectrum of affective principles into psychotherapeutic practice may help recontextualize the legacy of behaviorism within psychotherapy. Behaviorism offered one precious gem to psychotherapy: *behavior modification* based on the rearrangement of external reinforcement contingencies. For instance, one can readily reduce undesirable behaviors by paying people to avoid their bad habits, a procedure that is currently commonly used in treating addictive urges. However, that externalist view continues to skew cognitive thinking in the field of psychology, leading to continued misconceptions of organisms as passive information-processing machines rather than emotionally proactive creatures. In contrast, a coherent vision of the affective mechanics of the mammalian mind provides a clear picture of the BrainMind infrastructure—the active, emotionally tuned interpersonal mental apparatus—which is needed as a guide to therapeutic thinking.

Still, at present, cognitive conceptions of psychopathology remain more prominent than explicit affective conceptions in clinical thinking. Although emotion regulation problems are surely connected to dysregulations in both cognitive and affective aspects of mind, perhaps our current zeitgeist encourages psychotherapists to seek a more comprehensive scientific grasp of our cognitive, rather than our affective, nature. Perhaps this is because of the more massive institutional investments that are being made in the cognitive rather than the affective neurosciences. Whatever the reason, we do not really understand much more about how the higher brain functions can weave together our cognitions than we do about how the lower brain generates emotions. We do know that the personality characteristics of therapists—no doubt especially their capacities for affective attunement—are typically more important than the specific procedures they use. It is well known that when one is feeling bad, the attention of caring others can rapidly reduce negative affect. Twelve-step programs are probably so remarkably effective, because they provide the social concern and affirmations that are needed to become reconnected to one's potential for

positive feelings. The social-affective power of other minds can help people deal effectively with negative affects, and thereby the affective terrain of the brain may provide a clearer description of the psychological forces that lie at the heart of most human psychological problems, and the intra- and inter-personal mental dynamics that need to be recruited for optimal therapeutic effects.

Indeed, perhaps the cognitive issues that are relevant to psychotherapy remain more scientifically slippery, and harder to understand, than the underlying emotional ones. Certainly, pure cognitive benefits tend to slip away more readily than affective benefits. People can easily reach moments of apparent clarity in the midst of psychotherapy sessions only to have all that progress dissipate as they regress to their old affective habits between appointments. This may be because each of the primary-process emotions has "enslaved" large cognitive territories for self-serving purposes. If so, it is possible that attempts to achieve emotional homeostasis more directly might allow simpler and more effective routes to facilitating desired cognitive reorientations than working more strictly at the cognitive level.

The stranglehold that self-centered emotional systems (for an expansion of this concept, see Northoff & Panksepp, 2008; Panksepp, 1998a; Panksepp & Northoff, 2009) exert on cognitive processes can be overwhelmingly robust. For instance, PTSD can reflect highly aversive affects that are stirred up by simple secondary-process memories (e.g., classical conditioning as described in Chapter 6), which are often unconsciously triggered (by unattended stimuli). To the surprise of many therapists, it has recently been found that rather simple cognitive-type interventions—such as eye-movement therapy to be described at the end of this chapter—which do not aspire for any deep cognitive "insights," may help dampen the power of traumatic memories as effectively as other treatment modalities. Further, perhaps the affective storms of PTSD could be substantially diminished by therapists who know how to help patients reframe their traumatic feelings in affectively positive experiences that can be triggered easily by non-cognitive approaches. To reiterate, it is now widely recognized that memories are not as stable as most people used to think they were. As discussed in Chapter 6, every time memories are retrieved, there are opportunities to help them "reconsolidate" in less troublesome forms. Currently, this phenomenon holds out the promise that emotionally troubling memories can be reconfigured in affectively positive

frames of mind with the assistance of fairly simple somatic maneuvers, and perhaps even systematic presentation of positive affective experiences such as listening to soothing, comforting, happy music.

Since all psychotherapies have to begin cognitively, and most are designed to restructure the way people think about their problems, perhaps more direct affective approaches have not been as widely considered as they should be. It is quite understandable that in species such as humans, where language mediates practically every interaction, cognitive approaches will remain preeminent in psychotherapeutic enterprises. But do we know that it is within the higher cognitive dynamics of the Brain-Mind that the major therapeutic effects are actually generated, even as there is abundant evidence from brain imaging for higher brain changes? No one really knows, but perhaps many of the beneficial transformations actually occur implicitly within all the nested, hierarchical levels of affective-cognitive interactions in the brain (Figure 2.3). Indeed, perhaps the most lasting effects occur if the therapeutic path has been paved by changing the primary-process affective tone. If so, the work of clinical practitioners may be facilitated by more fully assimilating and utilizing the available evidence about brain emotional systems arising from affective neuroscience, and aiming to more fully utilize the most direct affective maneuvers available. Although people love to talk about the endless episodic memories that constitute their explicit minds, and psychoanalysis serves personal growth well in this way, it is by no means clear whether the cognitive or affective aspects of such interpersonal experiences are more important in providing long-term psychological relief. No doubt both are important, but I expect that without sustained affective change, the cognitive restructuring might not be as effective.

In any event, it is clear that psychotherapy is in the midst of an emotion revolution. The primal affective aspects of mind are no longer marginalized, but, rather, are recognized as the very engines of the psyche (Fosha et al., 2009a, 2009b). For instance, Greenberg and colleagues' work in process-experiential therapy has emphasized the necessity of experiencing and expressing clearly differentiated primary emotional feelings, such as anger and fear, in the context of therapy (Elliot et al., 2004; Greenberg, 2002). The work of Foa and colleagues (1998) has demonstrated that the actual experience of fear during therapeutic sessions is essential to the success of exposure treatment for anxiety disorders. In contrast, in dealing with war-

traumas, practitioners in the field find that the immediate communication and discussion of what has happened, opens the doors to immediate and palpable benefits (Belenky, et al., 1996). But the bottom line is that further progress with such novel approaches must be grounded in understanding the nature of the underlying pathogenic factors. There is insufficient space here to discuss all the major psychiatric disorders in great detail, so I have selected depression as an exemplar of how affective neuroscience thinking may be useful for further progress.

PSYCHOPATHOLOGY AND THE BRAIN WITH A FOCUS ON DEPRESSION

So what are the sources of depression within the brain? This is a hot topic of neuroscience inquiry right now, and surely lasting answers for depression must come, in part, from a much better understanding of the affective storms experienced by people as they move through the ups and downs of life. Depression may arise when certain primary process emotional systems become chronically imbalanced. If so, we need to better understand neuroscientifically the primary-process emotional systems—the raw affective endophenotypes that exist in the brains of all mammals (Panksepp, 2006a). Each of the fundamental emotional systems can become sensitized or desensitized by repeated affective experiences. And each emotional experience can promote various forms of implicit and explicit learning as it interacts with our representations of internal and external reality. By such molding of chronic feeling-thinking patterns, people's attitudes can become rigid and negativistic, diminishing more fluid positive reasoning.

Thus feeling can become extreme in a variety of sustained ways and for many reasons. No doubt, most of what we need to know about these processes remains to be discovered. But depression, being the "common cold" of psychiatric disorders, is a key problem to focus on, especially because of its high and seemingly ever-increasing prevalence in modern societies. It is possible, of course, for the increase to be only apparent, partly driven by pharmaceutical companies which try to generate demand for their highly profitable, often marginally effective, antidepressant medications through sophisticated marketing strategies. Surely, many people throughout history have had short bouts of depression in their lives, but what is new is that up to 20% of the population currently seeks medical

assistance for the symptoms. And often, medications are not provided as needed but chronically. It now seems likely that sustained use of antidepressant medications can produce sustained shifts in brain neurochemical patterns (e.g., through development of receptor supersensitivities), in ways that lead to even stronger negative feelings, when medications are terminated (for a fine overview, see Marcia Angells analysis in *The New York Review of Books:* http://www.nybooks.com/articles/archives/2011/jun/23/epidemic-mental-illness-why/).

But we simply do not know enough about the depressed BrainMind yet to have any definitive conclusions. As patient Andrew Solomon (2001, p. 29) put it, "Let us make no bones about it: We do not really know what causes depression. We do not really know what constitutes depression. We do not really know why certain treatments may be effective for depression. We do not know how depression made it through the evolutionary process. We do not know why one person gets a depression from circumstances that do not trouble another." Our failure to understand depression may arise, in part, from the fact that neuroscience has not yet studied the most relevant ancient affective circuits of the mammalian MindBrain closely enough.

Indeed, the discussion so far has not conceptualized which types of brain emotional systems are most strongly impacted by depression. To help remedy that, Watt and I (2009) recently provided a comprehensive synthesis of a basic affective-social neuroscience view, with peer commentaries and extended responses, that may promote progress on this recalcitrant problem. Our proposal was that to understand depression(s), one needs to understand the psychic pain that arises from sustained separation distress—excessive and sustained arousal of the GRIEF system—one of the most important social-emotional systems of the brain. In addition, perhaps because of the sustained arousal of GRIEF, the arousability of the SEEKING system becomes diminished in depression (as noted earlier, and see Coenen, et al., 2011, as well as Panksepp & Watt, 2011; and Zellner et al., 2011).

We have already seen that the GRIEF system—concentrated in the anterior cingulate gyrus, the ventral septal nuclei, dorsal preoptic area, as well as the bed nucleus of the stria terminalis (BNST), dorsomedial thalamus, and the periaqueductal gray (PAG)—figures heavily in the generation of emotions of sadness and grief and the urge to cry (Herman & Panksepp, 1981; Panksepp et al., 1988). Brain chemistries that exacerbate

feelings of distress (e.g., the release of Corticotropin-Releasing Factor) and those chemistries that powerfully alleviate distress (e.g., brain opioids, oxytocin, and prolactin) are the ones that figure heavily in the genesis of social attachments and all may play a role in the regulation of social bonding (Nelson and Panksepp, 1998) and thereby depressive affect. Helen Mayberg and colleagues (2005) have provided substantial relief from treatment-resistant depression with deep brain stimulation of the subgenual anterior cingulate, where the higher brain loci that mediate GRIEF-type ruminations can be inhibited. It is likely such brain stimulation is scrambling the neural sources of the psychic pain engendered by the GRIEF system. Indeed, the chronic overarousal of this system may be one of the major sources of depressive dysphoria.

A global shutdown of SEEKING, that characterizes the transition from "protest" to "despair", may be a pivotal BrainMind change in sustained depression. It is well established that early experiences of separation and loss predispose people to depression or can trigger a first depressive episode (Heim & Nemeroff, 1999). Also, the mechanisms that mediate attachment and separation are much more sensitive in females, who are twice as likely as males to suffer from depression. We have also known for a long time that the affectively positive opioids that regulate the brain's separation/attachment mechanisms have powerful antidepressant properties. If it were not for the addictive risks of opioid drugs, they might still be used as antidepressants the way they were before the advent of modern psychopharmacology in the 1950s. Thus, depression may, in part, reflect diminished activity of those natural brain chemicals that make us feel good when we are safely and securely attached to others (see Chapter 9). In part, depression may reflect the failure of our natural endogenous opioids to provide an adequate sense of security—in short, loving social-attachment bonds are a primal form of addiction (Panksepp, 1981a).

As noted earlier, a safe, nonaddictive antidepressant drug (the mixed opiate receptor agonist/antagonist, buprenorphine) is currently available (Bodkin et al., 1995), albeit double-blind, placebo-controlled trials to test its efficacy remain to be conducted. Still it is a "safe opioid", and, at low doses, buprenorphine can directly counteract psychic pain. It will not be severely addictive, unlike most opioids, because as doses are increased the agent exerts opioid antagonistic effects. It is to be expected that such agents may also be quite effective in reducing suicidal ideation. Further study of

the GRIEF system should yield various new medications for disorders ranging from depression to social phobias. However, such translational research into clinical issues will only be effective if we realistically entertain the nature of the underlying affective processes.

Although these opioid-driven attachment systems may be pivotal in depression, there may be many associated mechanisms that mediate the various depressive subtypes. For example, the dynorphin-facilitated shutdown of dopamine-driven appetitive systems (when an individual mentally "gives up" in despair) may form an independent etiological mechanism in a subset of cases (Knoll & Carlezon, 2010). Another type of depression may arise from the emotions associated with material loss—especially the feeling of defeat that arises in dominance encounters as organisms compete for resources (Panksepp, Moskal et al., 2002).

Parenthetically, in the most extreme emotional circumstances, precipitous arousal of the separation-distress system may be one of the underlying causes for panic attacks (Panksepp, 2006a; Preter & Klein, 2008). Our understanding of the psychobiology of social attachments, which has largely arisen from work on these neurochemistries, also links up with a preliminary understanding of childhood disorders such as autism. It is possible that some children with this condition may be socially aloof because they are addicted to their own self-released opioids, as opposed to those activated by significant others (Panksepp, Lensing et al., 1991). More recently, the idea of an oxytocin component to autism has been entertained (Panksepp, 1992b to Green & Hollander, 2010).

If this analysis is correct, then we need vigorous new research efforts into the brain mechanisms of attachment and separation distress (the GRIEF system), as well as their role in the etiology, mechanisms, treatment, and prevention of depression. Just like traditional behavioral neuroscience research, such investigations would cover the full gamut of methodologies from the genetic and molecular levels, through anatomy and chemistry, to functional imaging, brain stimulation, and drug trials (Watt & Panksepp, 2009). However, according to this research model, special emphasis would be placed on the integration of psychological and neurological approaches (Panksepp & Watt, 2011).

As already noted, modern preclinical research, with a few exceptions, continues to focus on psychiatrically relevant external symptoms, while ignoring the importance of affects (and the brain systems that generate

emotions). For example, investigators who target fearfulness rarely discuss the nature of the FEAR circuitry, focusing instead on the conditioning of anxiety-like behavior and, more recently, on how their work may relate to resolving PTSD symptoms (Davis et al., 2010).[2] Researchers may not even consider the GRIEF system when discussing the sources of anxiety, but they have discovered a new form of "anxiety" in rats that is integrated by part of the "extended amygdala" called the BNST. However, it has long been known that this is the brain area where one can very easily evoke separation calls in animals; thus their discovery may have been of this social emotional response rather than just a variant of the traditional fear response. There are two distinct anxiety-type systems in the brain, FEAR and GRIEF, and they both promote negative feelings. If we do not distinguish these two "anxiety"-promoting systems, we may succumb to many errors in thinking and therapeutics. At present, analyses of mammalian brain emotional systems, with attempts to model psychiatric disorders in light of affective circuit imbalances, remain rare (Panksepp, 2010b).

EMPATHIC AFFECTIVE NEUROSCIENCE: VIEWS ON SELECTED CULTURAL PRACTICES

A better understanding of emotional brain systems also points the way to promoting better childrearing practices and hence functioning societies. The implications for childhood thriving that may arise from prolonged breastfeeding, mothers and infants sleeping together, and the importance of abundant early childhood physical play have recently been extensively discussed (e.g., Narvaez et al., 2012) as has the quality of marital relationships for the mental health of children (Code, 2009).

Such issues extend through the life span, in ways too complex to cover here. However, let me consider a most common issue that faces everyone—the death of a loved one. What are the most effective ways to grieve? With the rapidly shifting tides of modern cultures, excellent examples of how to navigate such life passages are less and less evident. Traditionally family members have coped in the context of community support. While this also remains a central feature of grieving in modern societies, the practice has changed and weakened in many ways. In traditional societies, the safety nets of perceived community support were often much stronger than they

typically are today. Although many of the outward forms of mourning remain intact, the affective support that people obtain in modern cultures, with the gradual narrowing of "communities," has often become more fragile. The social flux that characterizes so many individualistic modern cultures has depleted the level of overall social support many individuals feel in the midst of mourning. Here is a vivid description by Ellen Dissanayake of how the tragic passage of life can be structured to sustain one's social connections and to obtain needed emotional support from an extended "family":

> Traditional ceremony and custom . . . play a much larger part in the life of a Sri Lankan than in ours. After a person dies in Sri Lanka, the mourners arrive during the course of the day at the home where the deceased is lying in an open coffin on a table in the living room, surrounded by flowers. The bereaved family members greet each visitor at the door, breaking down in sobs with each new arrival as they talk about the circumstances of the death and the merits of the deceased. The guest enters the house and joins other guests; they chat quietly with each other about any subject (we heard discussions about movies, business, and political matters); and after a decent interval, they leave. Eventually the family and close friends go to the place of cremation or burial where Buddhist monks join them and recite the appropriate Palwe texts—reflections on birth, death, decay, and reincarnation. Three days after the disposition of the body, the family and priests hold an alms-giving ceremony; other alms givings in memory of the deceased occur after three months, one year, and at yearly intervals thereafter. We realized that this kind of formalized handling of grief, with regular, community-sanctioned opportunities to weep and express one's loss at greater and greater intervals of time, gave to the bereaved a sort of patterned program to follow, a form that could shape and contain their feelings. Instead of having to suppress their grief and sense of loss in the interests of being brave or "realistic," or having to release it haphazardly or in solitude, the bereaved is enabled—compelled—by the ritual of mourning to acknowledge and express it publicly, over and over again within a preordained structure. The temporal structure of the mourning ritual, simple as it is, assures that thoughts and feelings about one's loss will be reiterated at prescribed times. Even if one might not consciously have proper mournful feelings, the custom of successive alms giving ensures that these feelings are elicited. The prescribed formal ceremonies become the occasion for the extended social network publicly expressing their sorrow. (2003, pp. 19–20)

This is a societal form of affect "therapy." These progressions highlight how a traditional culture has learned to deal with our ever-present sorrows with grace, compassion, and solidarity. People in traditional societies commonly care deeply about each other's lives, and this allows for individuals who have experienced loss to progress through the stages of grieving, and thereby to be less likely to descend into depression. It is also hard to imagine how psychotherapy could yield successful outcomes in the absence of a fundamental sense of attachment between clinician and patient.

Thus, the quality of the therapeutic relationship has long been recognized as a key to effective treatments.

This view was advanced by Carl Rogers (1902–1987), a humanistic therapist, through his concept of unconditional positive regard (Rogers, 1961, 1980). If therapists cannot assume a stance wherein they can empathize with the psychic pain of others, there can never be that sense of trust that is critically important for the healing touch. Without that trust, the foundation of the ever-present and wonderful "placebo effect", the endogenous opioid mediated feeling of social support, cannot take hold in the patient's mind. Without genuine empathy, which should lie at the core of every therapeutic interaction, there will always be a residue of suspicion, a feeling of being manipulated, as opposed to the deep acceptance that opens the portals for positive change—for feelings of redemption and salvation. Compassion (as in spiritually present counseling: Brammer, 2011) may be critically important for the caretaking stance that is essential for effective therapy.

TOWARD A SYNTHESIS OF AFFECTIVE NEUROSCIENCE AND THERAPEUTIC PRACTICES

The above perspectives may currently be the minority view in biological psychiatry, but I believe that they reflect a natural and reasonably structured way to relate neurobiological causes to the foremost emotional concerns of psychiatry and experiential, emotion-focused therapies. Indeed, sensitive clinicians are coming to realize how such conceptual maps, experimental inquiries, and neuroscientific findings can inform their efforts (Valliant, 2008). Although this may not always have been clear in my recitation of many preclinical facts in earlier chapters, my overall hope is that an evidence-based understanding of how primal affects are engendered in the MindBrain will promote clinical thinking. Such approaches may also eventually allow us to better envision the nature of emotional problems in future psychiatric diagnostic schemes, where thinking needs to be restructured along the lines of emotional endophenotypes rather than artificial syndromal thinking (Panksepp, 2006a). At the same time, it is clear that research on primary-process emotions in biological psychiatry remains in its early stages.

Obviously, psychotherapists do not need to be told that emotional dysregulation is the key problem afflicting their patients. This is self-evident. And many therapists currently recognize that optimal progress will only be achieved if they engage sincerely with patients' emotional dynamics, and to work creatively and sensitively to facilitate the restructuring of patients' affective lives, without neglecting that humans are also fundamentally cognitive beings. So far, this sort of multidimensional therapeutic work remains more of an art than a science. Just as in the skilled playing of a musical instrument, clinicians need solid, rigorous, and practiced techniques, as well as broad-based knowledge of relevant, empirically founded theory, to support the flights of inspiration and breakthrough engagements that mark true clinical artistry. The more psychotherapeutic practice is grounded in affectively sound thinking and technique, the more consistently effective it should become. The structures of affective neuroscience can help clinicians become more systematic in their methods, moment to moment and day to day, and thereby less dependent on serendipity and clinical intuition.

Children, in particular, become more responsive to therapeutic help if one keeps their real affective concerns in focus during clinical interactions. For better treatment of their social-affective disorders, we probably would be wise to consistently recruit their PLAY energies. There is a jester in all of us. (Thank goodness, for it can make play out of work—including, at times, psychotherapeutic work.) Like all primal emotional urges, the impulse to PLAY emerges from networks below the neocortex. However, it is becoming clear that the act of playing has remarkable effects on the cortex, programming it to become fully social, with many changes in gene expression that are allowing us to envision new treatments for depression (Burgdorf et al., 2010, 2011). As long as ludic energies are well used in clinical practice, clinical interventions are bound to move ahead positively as fast as possible.

For instance, shared laughter may index therapeutic moments of great value. If a therapist, in the midst of dealing with very difficult life circumstances, is able to promote a positive affect, even to the point of laughing with a client, might new discoveries in memory research, such as *reconsolidation*, be brought to bear on the attempt to more permanently soften the painful edges of life? All memories are labile when they are retrieved. They tend to return to their semipermanent "storehouses" and to

carry along the most recent affective structuring of experience. According to this view, the capacity of a therapist to shift a client from negative feelings and despair to periods of positive affect, before moving on to other issues, should serve as a vehicle for ensuring that those negative memories have lost some of their power over clients' feelings about themselves and their life circumstances.

Thus, perhaps the positive affect of PLAY is as important for adults as it is for children. The capacity for emotional resilience is increased by direct *physically* playful engagements. Those real-life interpersonal delights are seldom used in traditional psychotherapy, even with children. It is impossible for children to play without moving their bodies. Of course adults can have fun with just verbal interactions, but one must wonder whether it might not be useful for therapists to focus on the body and encourage clients who are very tense to assume different bodily postures, from sitting in a chair, to sitting on the floor, to standing and perhaps engaging the therapist with various nonaggressive emotional gestures (an approach that I have seen demonstrated by Pat Ogden and colleagues (2006), who incorporate sensorimotor aspects into their psychotherapeutic approaches). Because primary-process emotions are all about dynamic movements, perhaps such therapeutic flexibility might open emotionally expressive "doors" for genuine playful social interactions with abundant long-term therapeutic impact.

In short, we need to learn how to wrap agonizing negative, even traumatic, memories within new, positive affective "wrappings"—a possibility that new memory research, especially on reconsolidation, as already noted, coaxes us to consider. Perhaps there is no better way to soften troubling memories than to evoke positive emotional arousals soon after the reliving of intensely negative emotional memories. If the therapist is able to move their interactions with patients gracefully into a positive affective, or playful, space, would the memory reconsolidate in a less painful way? If our vision of learning is correct (see Chapter 6), namely that new, unconditioned primary-process emotional states regulate the learning process (with a bottom-up control of information consolidation), we should be able to provide new affective-contextual variables for old memories originally laid down in negative affective states. I would predict that the painful, splintered edges of past memories can be "sanded down" in order to allow positive affect to recontextualize troublesome memories. My

colleagues and I have already observed such effects in preclinical studies: Play after stress can diminish depressive responses; indeed, if one tickles a rat after it has been exposed to a fearful situation, the power of the negative affect can be diminished.

Of course, in using such strategies, one must remember that the genuine experience of play can also evoke negative emotions; that is especially common in childhood play. In our attempt to evaluate the utility of play interventions in young children (Scott, 2001), we found that problematic behaviors could be minimized if addressed immediately, gently, with a return to play as being the reward. Also, there are bound to be many special problems to be confronted in work with previously traumatized children, but we anticipate that social joy, if it can be facilitated in such children within an atmosphere of trust, can have substantial benefits above and beyond the efficacy of more cognitive therapies (Panksepp & Scott, 2012). In order to achieve such goals, there still needs to be abundant research, as well as training of therapists in the art of facilitating natural physical play in the context of social safety.

Any therapist who can capture the therapeutic moment in mutually shared joy episodes will have brought the client to the very doorstep, the wellspring of happy living. To the extent that the patient can remain there, in both body and mind, one may have offered one of the greater emotional gifts that psychotherapy can ever provide. PLAY should have a very special place in psychophysical therapies, from childhood to old age. Of course, since there is also the dark side of humor, where someone ends up being the butt of a joke, humor as a form of play can be a double-edged sword. Thus, therapists must be ready to identify emerging crises and deal with them in the present moment.

There are also therapeutic possibilities in the manipulation of the CARE and GRIEF systems. These are the Janus-faced twins of social attachments. Expressions of distress in infants arouse a mother's urge to nurture. In more general contexts, we feel a natural compassion for the suffering of others. Deep subcortical emotional resonance, including deep empathy when others are in distress, appears to be a natural property of the mammalian brain. Indeed, perhaps therapists need to be especially adept at using their cortical "mirror-neuron" systems to promote affectively meaningful contacts and interpretations. In other words, their bodies need to resonate and harmonize

with the emotional states of their patients, as opposed to simply being an unexpressive "talking head."

It will be interesting to see how chemical agents derived from the shared chemistries of the CARE and GRIEF systems, including safe opioids (e.g., buprenorphine) and oxytocin, can eventually be used therapeutically. These are most probably the brain chemistries that facilitate our capacity to create positive intersubjective spaces with others. Medicinal use of such social chemistries may one day allow clinicians to selectively enhance the prosocial emotional feelings that promote therapeutic progress. Supplementing the therapeutic situation with nurturant activities may increase the release of endogenous opioids, oxytocin, and prolactin. However, supplementation with such hormones—for example, intranasal oxytocin before a session in couples therapy—might also enhance therapeutic flow by allowing both members of the couple to work more effectively in the present moment.

The vast amount of social-attachment research and the brain mechanisms of social pain have been well summarized by Macdonald and Macdonald (2010). In addition to the socially induced soothing effects of brain opioids, oxytocin in the brain has now been shown to mediate trusting behaviors in economic decision-making and perhaps the capacity to read other minds more sensitively (Pincus et al., 2010). Such changes in the tertiary-process aspects of the mind may largely arise from the fact that oxytocin diminishes separation anxiety and loneliness; in other words, it promotes confidence (Panksepp, 2009c). In preclinical models it can reverse some of the deleterious and depressive effects of social isolation (Grippo et al., 2009). It is to be expected that, with help from this natural chemical, people who are overly timid, who suffer from "social phobias," may feel more comfortable interacting with others.

SUMMARY

Affective neuroscience aspires to clarify the actual primary-process affects that exist in the mammalian MindBrain. The triangulation method—integrating evidence from behavioral, neural and mental analyses—is straightforward and not reliant on conjecture. I hope it provides a more stable platform for further study of foundational issues that need to guide clinical thought, psychiatric research, and the development of new affect-

focused therapeutic practices. Using this approach, preclinical investigators can focus their efforts on specific and hopefully relevant emotional brain networks as opposed to vague behavioral indicators of psychiatric disorders. Because the organization and functions of emotional systems can be studied and evaluated in a wide variety of species and in well-controlled experimental situations, affective neuroscience aspires to provide a more coherent empirical base for thinking about primary-process emotions than has so far been available. It also allows us to see why most of our thoughts, our cognitions, are so dramatically anchored by our affective states. Feelings came first in MindBrain evolution.

This is not to deny that most psychotherapeutic relationships have to also be negotiated at the cognitive level. Thus, primary-process affective neuroscience has not yet provided solid evidence for how the emotional powers of the mind need to be dealt with in therapeutic environments. However, it provides an alternative vision, hopefully a clear one, of how diverse negative affects may contribute to distress and suffering and how positive affects can be better used to counteract negative affects. Such principles may better contextualize optimal therapeutic practice and theory than earlier visions of affective life, where no solid neuroscience-base was available.

At the very least, in order to understand the core nature of emotional feelings, we must recognize the pre-propositional affective processes that emerge early in BrainMind development, and how they can exist independently of the enormous complexities of higher mental processes in humans. During early infancy, primary-process affective states are not enmeshed with the cognitive and linguistic processes with which they always interact later in life. By understanding the extremely plastic neurobiological nature of our cognitive apparatus (Doidge, 2007), which is dynamically and developmentally constructed largely from affectively laden life experiences, we are in a better position to understand how we might undo troublesome higher affective programming, some of which has epigenetically become part of the maladaptive hardware of the brain.

We should note that the distinction between cognitive and affective processes made throughout this book is still rather novel, even unpopular in certain quarters of contemporary cognitive and neural sciences. A majority of investigators interested in emotions, many of them arriving from language-based constructivist traditions in psychology, claim that we cannot

draw meaningful and useful distinctions between cognitive and emotional processes. However, at a primal neural level that is possible, and is an essential stepping stone for future neuroscientific progress.

Still, emotions and cognitions are so interactive that distinctions become difficult at the top of the brain—within the learned tertiary-process functions of neocortical networks. But they can be easily distinguished at subcortical primary-process BrainMind levels. This in no way seeks to deny the importance of understanding how the cognitive and affective realms interact, especially for psychotherapy, and other dynamic systems theories of mental qualities (e.g., see Lewis, 2005).

To recap, cognitions are those brain information-processing functions that are integrally linked to the sensory-perceptual portals of the mind, while raw emotions and affects reflect some of the most important within-brain organizing principles. The cognitive aspects are more closely linked to the programming of each individual's higher brain development, while the raw emotions and affects represent the ancestral, inherited tools for living. Although the interaction of emotion and cognition is inextricably interwoven in the unique puzzle of each individual's higher mind, we must be able to envision cognition and emotion as different, albeit interconnected, types of mental processing. Anatomically speaking, they are as distinct and interactive as our hearts and skeletal muscles. In order to think clearly about foundational issues, we must consider the unique contributory aspect of such distinct levels of control within the brain (Cromwell & Panksepp, 2011).

This penultimate chapter has been geared toward all those in the helping professions who have been drawn to understanding the foundations of the human mind—our elemental mammalian affective nature—and how this knowledge may relate to increasing our understanding of a variety of human psychiatric problems. We can anticipate that a closer study of emotional-affective networks will eventually yield knowledge that will be helpful in the service of those whose lives have been ensnared by emotional distress. In the final chapter, I will elaborate on the philosophical implications of this knowledge for understanding some long-standing scientific dilemmas, as well as some concluding thoughts about the nature of human consciousness and the animal mind.

Epilogue: Recent Personal Experiences with PTSD, EMDR, and Reconsolidation

It is especially important for future researchers to clarify how certain emerging psychotherapeutic interventions work to modify the emotional tone of the brain. I recently underwent a personal experience with a novel form of psychotherapy—the procedure called Eye Movement Desensitization and Reprocessing (EMDR)—that has become popular during the past few decades, but is not universally accepted, for the treatment of PTSD and other dissociative states in which intense emotion-laden memories are compartmentalized, almost dissociated from the rest of the mind, rather than being integrated within a unified, emotionally well-functioning personality. Here, I present an account of my personal experiences with this therapy along with some hypotheses as to why this form of trauma-therapy may work so effectively.

* * *

As an introduction, let me just note that I have had my share of traumas in my life. The first "big item" was during 1944 when I nearly died at 1 year of age, after being scalded on my lower body while my family fled Estonia in advance of the Red Army. Those traumatic infantile memories are probably still partly alive, somewhere in the recesses of my brain, albeit diminished and, from the perspective of my explicit cognitive memories, totally unconscious. Still, such an experience surely left some kind of affective residue, perhaps reflected in a constitutional tendency toward anxiety and excessive worry, perhaps depression. Indeed, abundant evidence indicates that early traumas can increase the severity of the incidence of adult PTSD and future depressions (for a summary, see Watt & Panksepp, 2009). However, I fortunately have the kind of temperament, call it courage, to muscle through hardships.

My most recent traumas were related to a series of increasingly harsh cancer therapies that I was receiving, across a full year, while working on this book. While being treated at a world-class institution in Seattle—fondly known as the 'Fred Hutch'—Dr. Sandra Paulsen (author of *Looking Through the Eyes of Trauma and Dissociation* [2009]) kindly offered me

several sessions of treatment with EMDR (Dr. Paulsen has also contributed illustrations to this volume: Figures 1.7 and 5.1). She was interested to see if I, with my affective neuroscience perspectives, could shed some light on why EMDR has produced such robust therapeutic effects in numerous patients over the years. She guided me through a sampler of EMDR therapies for infantile as well as cancer traumas.

Let me establish a medical context for all of this. After almost 10 years of remission, I was once again in treatment for a malignancy of the lymphatic system—a non-Hodgkin's lymphoma. Back in 1998, a fist-size tumor was discovered in my lung thanks to a routine X-ray prior to double hernia repair. This helped explain the heaviness I had felt in my chest for many years. But because no cardiologist had found anything wrong, I had rationalized it as a residue of profound chronic grief following the death of my daughter Tiina (on Good Friday, 1991). Perhaps my core SELF, which is embedded in cardiovascular control systems, was experiencing persistent pangs of GRIEF. Wrong. Those chest symptoms disappeared following cancer treatment, suggesting it was largely the massive tumor pressing on my pericardium.

But before I headed onto the right track for a cure, there was a profound shock! When the initial pathology report on my tumor biopsy arrived, the young surgeon who had discovered it sat my wife and me down and informed us that, at best, I had a year to live. Indeed, his diagnosis of a small-cell carcinoma of that size statistically mandated that my days were numbered. Fortunately, after a month of profound worry (during which I was setting my affairs in order), pathologists at the Mayo Clinic correctly rediagnosed the tumor as a small-cell lymphoma, which, with great relief, I henceforth dubbed a "wimpoma," having learned it was treatable. Although my first oncologist was already offering me various harsh chemotherapies, at the end of an informative discussion of treatment options, I asked the key question: "Doc, have you ever treated this kind of lymphoma before?" With a shy shrug, he replied, "No." With a friendly nudge, I promptly shared my heartfelt wish: "Well, I sure would like to be treated by someone who has." He smiled back, and cordially tapped my shoulder, saying "Good choice!" Across the span of 6 weeks of daily radiation, the tumor was successfully treated at the University of Michigan Hospital oncology unit, where I was the last patient of Allen Lichter, who later retired as the dean of the medical

school to assume a leadership role at a medical foundation in Washington, DC.

The recurrence of my lymphoma in 2007—with the same type of tumor, but concentrated in the stomach—suggested a totally new flareup of the same disease (indeed, my contact doctor, Oliver Press, was interested enough to do molecular biology comparing this and the previous malignancy, but it turned out not to be a residue of the previous tumor). This time, however, the troublesome tissue—the new clone—had already disseminated, and invaded my bone marrow. My wife, as fate would have it, was diagnosed at that same time with a different non-Hodgkin's lymphoma. We had recently moved to Pullman, Washington, so that I could join the Veterinary College at Washington State University. We were both soon beset by a host of medical issues, heralded by a near fatal pneumonia with sepsis, which kept me in intensive care "at the brink" for 5 days. Then my first chemo cycle failed miserably. Then I went through several courses of much more aggressive "combo" chemotherapy, known ominously as R-CHOP, which produced a partial remission. Unfortunately, within 6 months, my disease had advanced well beyond the stage discovered at diagnosis, suggesting it had rapidly become resistant to conventional chemotherapies. If not treated with a stronger therapy soon—namely a stem-cell transplant—two doctors independently gave me the gloomy prognosis of having no more than a year and a half to live. As fate would have it, my wife, Anesa, had achieved full remission with R-CHOP, but she also suffered a relapse within 6 months.

We decided it was wise for both of us to seek treatment at the Seattle Cancer Care Alliance, where stem-cell transplants had been perfected. We had great confidence in the expertise and experience of the entire medical staff, while at the same time we faced the considerable stress of relocating for an extended medical leave. As I completed the first draft of this book in the spring of 2009, I also had just completed 4 months of treatment that had included several minor and one major life-threatening side effect (an antibiotic resistent "superbug"). I was in partial remission, but the residue was mopped up with focal stomach radiation. My wife had also achieved full remission. Throughout this journey she served as my full-time caregiver, because no one is admitted to the transplant clinic without someone ready to assist at every hour of the day. Together we survived multiple runs to the emergency ward for me, and also one for her. I thank

her for the devotion and courage she showed throughout. For me there had been half a dozen successive treatment regimens, increasingly aggressive, with ever more fatigue and various forms of physical discomfort. Overall, the experience was mildly traumatic, to say the least, and I was eager to see if psychotherapy, especially EMDR, might provide some benefits.

In my EMDR session, Dr. Paulsen first used an "early trauma" protocol designed to deal with implicit infantile memories. However, I was unable to access any explicit memories of infantile trauma (perhaps I could have, using Art Janov's revolutionary approach—see his 2007 book on *Primal Healing*), so we proceeded to the standard EMDR protocol that she had devised for dealing with cancer trauma issues. Most who have gone through arduous medical procedures, with the many iatrogenic side effects—that is, the additional medical problems induced by the treatment itself—have much to complain about. I certainly did.

My mind was full of fresh and troublesome memories from the autologous stem-cell transplant, during which I experienced blood and gastrointestinal infections by super-bugs. These led to frequent hospital visits and more failed antibiotic treatments than I wish to recall, requiring more diagnostic tests than I would ever want to repeat. This led to the repeated removals and reimplantations of my surgically placed intravenous Hickman Line (a catheter positioned near my heart, which, if it works well, obviates the need for endless venous punctures) for fear it might be the source of infection, followed by two PICC lines (peripheral intravenous chronic catheters), that all too often had to be pulled out, due to leaking and other problems. Eventually, my blood infection was brought under control with a solid month of the self-administration of intravenous infusions of Meropenem three times per day. I was also fed up with medically induced emotional side effects—from profound daily fatigue and apathy, to many 4 A.M. wakenings in terror (why, at such wee hours of the morning, does negative affect usually prevail?), often with the fear that I might lose my wife, my life, and the chance to finish this book if the last available antibiotic failed to kill the gram-negative super-bug before it killed me. Indeed, I was ready to sample some EMDR.

The therapeutic effects of EMDR for trauma were discovered by Francine Shapiro (2001, 2002). Treatment consists of a systematic retrieval of traumatic memories, followed by therapist-guided lateral eye movements to defuse the affective intensity of such memories. It has been reported that

the power of traumatic memories fades with the bilateral repetition of various simple attentional activities. Indeed, instead of lateral eye movements, one could focus on an alternate tapping of the knees, or listening to tones first in one ear, then the other (in other words, different types of bilateral stimulation). Dr. Paulsen and I decided to use a straightforward "standard EMDR" approach. As she put it, we would try "to clear out any unresolved disturbance about the cancer diagnosis itself, the treatment (including iatrogenic effects), and disturbances such as fury at the medical profession, the insurance industry, etc., fear for loved ones, fear of death, or anything else that put emotional obstacles in the way of clear sailing."

The several hours I spent with Sandra were eye-opening (not to mention "eye-moving") experiences: She had me systematically retrieve emotional feelings related to my cancer treatment (not at all hard to do), after which she promptly had me follow a row of lights flashing back and forth on a bar about the length of a yardstick. While my self-induced emotional feelings were clear and distinct, not hard to retrieve through a stockpile of autobiographical memories, as soon as I started to move my eyes, the feelings faded promptly. This was replicated time and again with different feelings: anger, anxiety, grief, and so on. It always worked very rapidly. In other words, as soon as I shifted into an external sensory-attentional framework, as a result of the bilateral eye stimulation, the intensity of affect melted like butter on a hot skillet (but without the sizzle). If done repeatedly, this type of therapy is claimed to defuse traumatic memories as rapidly and effectively as any other psychotherapy ever devised. Although that needs more empirical evaluation, the EMDR struck me as a rather direct ABT. At this point in time, no one fully knows what is happening in the brain (van der Kolk, 2006), but however it works, EMDR can be profoundly helpful.

So what is occurring in the brain during EMDR therapy? Why would such a simple procedure produce such dramatic emotional benefits? Let me share some theoretical speculations about how it may work. There are quite a few untested theories floating about, and I am often asked what my favorite viewpoint might be. The point I usually raise is that the exploratory eye movements represent a basic primate SEEKING response. Such scanning movements are organized in the deep layers of the superior colliculi, just above the PAG, which is the most important brain region for

elaborating all of the primary-process emotions (with more negative emotions being concentrated in the dorsal PAG, closer to the eye-movement circuits, while more positive networks are situated ventrally). There are neural connections downward from the eye-movement regions, especially to the dorsal negative affective regions of the PAG. If they turn out to be largely inhibitory (e.g., perhaps full of GABA), we may have a ready explanation for why negative affect would rapidly diffuse with the onset of exploratory eye movements. They may actively inhibit some of the most distressing neural circuits in our brains. Of course, there are many other possibilities—from cognitive refocusing, limited attentional resources, top-down regulation of emotions, and so on (that is the nature of scientific possibilities for every observation)—but none of these have yet been cashed out in terms of critical evidence.

Why would the affective benefits be lasting, with traumatic memories affectively "softened," albeit not forgotten? There are quite a few untested theories floating around, but here the concept of "memory reconsolidation" may again play a role (see Chapter 6) as it has for many possibilities discussed in the main part of this chapter. First, the EMDR therapist typically establishes an "island of safety" within which traumatic memories can be systematically reprocessed. For instance, in the psychotherapist Katie O'Shea's EMDR protocol, clients are (i) first instructed how to set aside unresolved emotional material in an "imaginary container," then (ii) trained to "access the ability to feel safe in safe situations," such as the therapeutic alliance, and finally (iii) to use EMDR to "reset automatic Emotional Circuits to a healthy level of response" (2009, p. 290). In other words, the EMDR situation may allow one to access affects associated with traumatic memories and then to recontextualize them, fairly rapidly, within a realm of safety.

This last possibility is consistent with the intriguing discovery of "reintegration" or "reconsolidation" in recent basic learning and memory research. Abundant preclinical work with animal models has now shown that memories that are retrieved tend to return to their memory banks with modifications (Nader & Einarsson, 2010). If such memory modifications can be at a primary-process affective level, one can readily understand how cognitive information about past traumatic events is no longer suffused with negative affect. In other words, the emotional sting has been extracted and

the affective "inflammation" has been soothed by gradually recontextualizing the affectively negative memory within feelings of safety.

Of course, this is currently just a theory as opposed to demonstrated neuroscientific fact. Indeed, most of what we know about the benefits of psychotherapy currently remain in that category, even though there are increasing numbers of brain-imaging studies that have shown how various therapies—cognitive-behavioral, psychoanalytic, and interpersonal—may change the balances of affective arousal within brain regions that are known to control emotionality (Cozolino, 2002, 2010; Doidge, 2007).

In contrast, the view advanced in this book about the "locus of control" of primary-process emotions and the fact that other mammals experience similar emotions are based on a mountain of facts. Anyone who does not believe such conclusions has the responsibility of explaining all that data some other way. I strongly urge the many behavioral neuroscientists who still do not believe animals have emotional feelings, to not just argue against the conclusion, as most are prone to do, but to experimentally negate the data on which the conclusions are based. That is how science works, but for some reason, on this topic, opinion has long ruled over facts. Because of that, I have recently chosen to write a series of frank papers in 2011 about the need for behavioral neuroscience to get real about the emotional feelings of other animals (e.g., Panksepp, 2011b, 2011c).

CHAPTER 13

Coda

Philosophical Reflections: Can We Go From Mice to Men and Back Again?

When they (my elders) named some object, and accordingly moved towards something, I saw this and I grasped that the thing was called by the sound they uttered when they meant to point it out. Their intention was shown by their bodily movements, as it were the natural language of all peoples: the expression of the face, the play of the eyes, the movement of other parts of the body, and the tone of voice which expresses our state of mind in seeking, having, rejecting, or avoiding something. Thus, as I heard words repeatedly used in their proper places in various sentences, I gradually learnt to understand what objects they signified; and after I had trained my mouth to form these signs, I used them to express my own desires

—St. Augustine (343–430), epigraph to Wittgenstein's *Tractatus*

WE KNOW THAT ALL HUMAN languages are learned. Whether we have an intrinsic "language instinct" within our brain circuitries or just an evolved desire to communicate remains unknown. But we do know that our urge to communicate is closely linked to ancient subcortical processes (Lieberman, 2001). So are all of our cognitive abilities (Koziol & Budding, 2009). The higher human mind, like that of all the other mammals, is grounded in our ancestral feelings, and for language acquisition, perhaps in our social affects: Indeed, little babies first become engaged with the prosodic intonations and melodies of their languages before they begin to assimilate

the propositional contents. Our "musical" emotional intonations may be the gateway to language acquisition (Panksepp, 2008b; Panksepp, 2009/2010).

In St. Augustine's reflections above, used by the philosopher Ludwig Wittgenstein as an epigraph for his *Tractatus*, we find an intuitive grasp of brain processes that learn to reflect the world—the role of mirror neurons in refining language and social understanding (Iacoboni, 2009b; Rizzolatti & Sinigaglia, 2008). St. Augustine reflected on those mysterious affective states of mind behind our innate tendencies to have, to reject, and to avoid aspects of the world—our raw emotional "intentions in action" (see Figure 1.4). Few modern scholars of the mind have envisioned the importance of our innate affective feelings for the kinds of cognitive creatures that we become (but see Davies, 2011), as we grow into our species-typical predilection to *acquire* language (for a summary, see Panksepp, 2008b, 2009/2010).

David Hume (1711–1776), the naturalistic philosopher who wrote the renowned *Enquiry Concerning Human Understanding* (1748/1910) on the heels of his *Treatise of Human Nature* (1739), was a prominent partisan of the affective view of intentionality. He advanced the view that human behaviors are strongly influenced by emotional feelings, a view that lay largely dormant in his era of rationalism, through many centuries (see McGilchrist, 2009), until Damasio resurrected it pointedly in his *Descartes' Error* (1994). But Damasio advanced his own potential error at that time: namely, that human feelings arise largely from higher brain functions. We have already noted that as we finish this book, Damasio has fully reconsidered that perspective in his fourth book, *Self Comes to Mind* (Damasio, 2010), where he endorses a deeper subcortical evolutionary view about the origins of mental life, not unlike the one developed here and elsewhere, (Panksepp, 1982, 1998b), but until recently he did not fully accept that raw affects are already fully developed in subcortical brain regions. In his new view, he does.

Still, the majority of neuroscientists and psychologists remain silent, agnostic, or in denial about the subcortical sources of mind. They use rewards and punishments to train—to reinforce—their animals, in abundant studies on learning. But many still seem to believe, as did our behaviorist forebears, that animals feel nothing—that the brain mechanism of affective feelings do not contribute to the processes of learning and memory. Human research has long suggested otherwise. The evidence from animal research

has also long supported the opposite conclusion. But at present, the silence in cross-species brain science is deafening about the role of affective experiences in controlling animal behaviors.

Why most neuroscientists choose to remain silent about the affective feelings of the animals they study is a mystery to "outsiders," . . . but the answer is simple: Research thrives successfully on the ruthlessly reductionistic principle that brain mechanisms and behavior count but that mental activities in the brains of animals do not. That is where the funding is. This bias has long been detrimental to our scientific understanding of human emotions and a scientific appreciation of the roots of mental life by the intellectual community at large. It also leaves most citizens—who could benefit from a better understanding of diverse affective feelings and of how ancestral emotions control their higher intentions—more baffled about their minds than they deserve to be in this postmodern age.

Clearly, emotional feelings have evolved. As Darwin surmised in the *Descent of Man* (p. 127), differences in the mental lives of mammals are ones "of degree and not of kind." Our emotional feelings have a long evolutionary history, and the ancestral roots for these feelings are still shared by many living species. This is good news for science, and it should be good news for humans as well. This kind of knowledge can resituate our species in the framework of mental lives that preceded us, while respecting our vast, and surely unique, ability for cognitive depth. This concluding chapter aims not only to provide a conceptual synopsis of this book but also to deal forthrightly with cultural resistance, inside and outside brain science, to recognize the importance of understanding the raw emotional experiences of other animals for understanding the sources of our own feelings.

To contextualize the many strands of evidence summarized in this book, let us recall how Jeremy Bentham (1748–1832), the father of utilitarianism, suggested we envision human feelings. He stated that "utility" reflects "that property in any object, whereby it tends to produce benefit, advantage, pleasure, good, or happiness . . . or . . . to prevent the happening of mischief, pain, evil, or unhappiness" and reinforced it with "Nature has placed mankind under the governance of two sovereign masters, pain and pleasure. It is for them alone to point out what we ought to do, as well as to determine what we shall do" (*Introduction to the Principles of Morals and Legislation*, 1779/1879, p. 1). Clearly, then, as should have been clear to all

behaviorists, the "property in any object" they use to reward animals routinely works because of its capacity to evoke positive feelings. And would it not be the same for "punishments" although, as with many feelings that mediate rewards, there are a large variety of negative affects? If there is no so-called mind-dust in the universe (a phrase coined by William James to suggest that nonliving matter may contain some kind of proto-consciousness), then the inanimate material world, when it coalesced into complex life a billion-some years ago, found the solution for signaling intrinsic values that support life. This property was affective experience, constructed completely from neural activities. Mind arose with certain types of neural circuit activities, probably going back at least to the ancient networks that created organismic emotional coherence deep in the brain.

At present, we can be confident that mammalian brains have many intrinsic affective values, still shared, in kind if not the precise form, by humans and all other mammals, as well as birds, and probably many other types of creatures as well. However, few neuroscientists or psychologists know (or study) how affects are constructed in mammalian brains, largely because of the polarizing effects of behaviorism and more recently because of the failure of the psychologically oriented research community to acknowledge, perhaps even appreciate, the importance of animal models for addressing the nature of consciousness. This must surely be due to the still deafening silence that animal brain researchers maintain on the topic. However, answers to some of our most urgent questions in psychiatry must come from the use of "preclinical" models of affective states, arising from emotional systems' order and disorder.

The BrainMind is clearly an evolutionarily layered organ, grounded on affects, where major passages are still evident in brain organization—the more ancient functions are concentrated in lower and more medial brain regions, and the more recent ones are in higher and more lateral regions. Within an evolutionary framework, animal brain research can provide the most profound guidance in understanding the foundations of human feelings. Indeed, with such work, we may eventually come to understand how human affective experiences arise from mammalian brain dynamics. This is not to suggest that animals develop the sophisticated cognitive-affective sentiments of humans, nor do they ruminate about their misfortunes the way we do, but we should come to recognize that the primary-process affects, genetically built into animal BrainMinds in their

raw form, are not all that different from the ones that come to guide the affective proclivities of human brains. Sadly, the seemingly endless conceptual debates in human psychology and philosophy often drown out the empirical signals that neuro-evolutionarily sensitive animal research has long provided: All mammals are intensely affective creatures.

THE MOST IMPORTANT QUESTION IN NEUROSCIENCE?

To consider the philosophical issues alluded to above in practical and clinical terms, I would again pose the following question to all neuroscientists and biological psychiatrists interested in the mind: What is the most important question in all of neuroscience? Surely there would be a vast diversity of answers ranging from the molecular nature of memory to the neural "computations" that mediate cognitions, with an occasional vote for the nature of free will. Perhaps many biological psychiatrists would currently cast their hopes with specific brain and genetic substrates for psychiatric disorders, with a few votes for the nature of conscious experience. I would cast my vote for: "How are raw affective experiences created in the brain?" Why is this so important? The answer could help to clarify the foundational nature of experience in general (i.e., primary-process consciousness), as well as the diverse affective disturbances that human souls can suffer (Solms & Panksepp, 2012).

Thus, for depression, I would specifically ask: "Why does depression feel so bad?" Why does depression hurt? Why is it so psychologically painful? What does it mean to experience social pain (MacDonald & Jensen-Campbell, 2011)? Few neuroscientists have been willing to ask such questions, but some working hypotheses have been garnered from affective neuroscientific perspectives on primary-process emotionality, based on John Bowlby's seminal view that the arousal of GRIEF—the acute psychological distress engendered by separation from maternal CARE—if prolonged, leads to the sustained despair that is the gateway to depression (Panksepp & Watt, 2011; Watt & Panksepp, 2009). Likewise, addictions are sustained not only by positive feelings, but also by the potential for strong negative feelings that build up internally, as one seeks pleasure through mind-altering drugs (Kassel, 2010). Through the clarification that laboratory rats have a distinct set of affectively positive vocalizations, we can now use these measures as direct indicators of where animal minds are

in an "affective space," and this can provide novel understandings of addictions, depression, and general well-being (Brudzynski, 2010; Burgdorf et al., 2007; Knutson et al., 2002; Panksepp, Knutson et al., 2002; Zellner et al., 2011).

Psychologists, who rarely take deep neuroevolutionary perspectives on the nature of mind, are beginning to accept that certain aspects of positive and negative affects are part of the evolved physical landscapes of the human mind (Lambie & Marcel, 2002). Even some diehard social constructivists and those who ascribe to dimensional visions of emotions, a robust force in current academic psychology, are ready to accept a biological foundation for human feelings (Barrett, 2006; Russell, 2003, 2009). However, such investigators of human emotions do not readily accept the evidence for any more highly resolved affective life than feeling good or bad at the primary-process level.

The general failure of the psychological science community to recognize the primary-process emotional aspects of brain organization (there are exceptions: e.g., Buck, 1999; Izard, 2007) leave many debates like this unanchored by neural considerations, and thus restricted largely to very difficult and intrinsically confusing tertiary-process considerations: Those higher levels of mind are surely largely socially constructed, leading to great idiographic variety. But animal brain research indicates that there must also be many inborn feelings in human brains, not only because of the empirical evidence discussed in this book, but also because that would be a wise way for evolution to build brains.

If primary-process affects have any evolutionary function at all, besides simply guiding learning, it is to intrinsically anticipate future survival needs. For instance, if affects provide immediate unconditional "valuative" guidance of behavior, then it would be most useful to have accurate affective signaling of diverse internal states and external stimuli that threaten survival as well as those that promote satisfying, even happy, living. Of course, this is not to deny that some primary-process emotions may cut across various affects (e.g., especially the *desire*- and *interest*-generating urges of the SEEKING system), nor do we suggest that higher brain functions could not further parse affective feelings and meanings in uniquely human ways. A complex human reflective-affective consciousness emerges with learning and thought. Given the hierarchical systems that are present at many levels of BrainMind evolution, many of the complexities

are instantiated in the nested hierarchies of BrainMind functions, where the lower affective brain functions become re-represented in higher functions. With time and education, the higher functions develop recursive supervisory (executive) control over emotional expressions (see Chapter 2, Figure 2.3).

But at their core, primal affects are internal valuative processes that promote survival. Existentially, they are brain processes that make our experiences important to us, not only in terms of survival but in terms of everyday values. They are the rewards and punishments—the unconditioned stimuli and responses—that behaviorists use to mold animal learning to almost any form they wish, except when they try to go against the strongest instincts of animals. For instance, it is next to impossible to train rats to run backwards down a maze for food. Across the years, many of my students who came in with behavioristic biases have tried but failed. That is because rats were designed, through evolutionary selection, to pursue the fruits of the world with their noses rather than with their butts.

What would our lives be without the great variety of emotional feelings, from love to hate, that color the fabric of our days with meaning—from the everyday joys and torments to the subtle, at times sublime, affective richness of great music, dance, theater, and other arts? Our affective lives coax us to treasure and detest various events and objects of the world, many of which would have no psychological depth, no profoundness, without our affective capacities. Thus, whatever *basic* values do exist in this human psychological world of ours, they reside inside human brains, and to a substantial degree, in the ancestral minds we inherited from earlier animals.

Our core values arise from the evolved emotions—and incentive-responsive properties of many ancient networks of our brains—especially those concentrated in the medially situated subcortical brain regions that all mammals share, in homologous networks of complexity, because of their common ancestry. These primal powers of the mind become connected to secondary life experiences through learning. Of course, if we humans did not have emotional feelings, we would not bother to seek them in the brains of other animals. But in pursuing such issues scientifically, across species, modern affective neuroscience finally assures us that we are not just indulging in idle anthropomorphism (as if reality is situated in the lower right quadrant of our truth diagram, Figure 1.5). This makes the study of comparative neurophenomenology—the study of the internal psychological

contents of MindBrains—a critically important scientific undertaking, across species (Panksepp, 1999). In other animals, affective states are the easiest contents of their minds to study, because the neural circuits that engender emotional *actions* (not just "responses") are easily observed and are intimately intertwined with animals' emotional feelings. This allows a dual-aspect epistemology, whereby observable behaviors can be used as proxies for hidden feelings (Panksepp, 2005b).

Still, affective feelings are thoroughly subjective, and no physical science has yet accepted the existence of any subjectivity in the hidden recesses of the material world. Despite Darwin's (1872) seminal acceptance of animals' feelings in *The Expression of Emotions in Humans and Animals*, the reign of behaviorism and logical positivism early in the twentieth century has imposed a severe, century-long constraint on the scientific discussion of whether other animals have affective feelings that guide their behaviors. At the experimental level, especially in brain research, that conversation has barely been re-engaged (e.g., Mendl et al., 2010; Panksepp, 2010a).

As a result of the marginalization of animal feelings, a neuroscience of basic human values (i.e., affective states) became disconnected from relevant animal models that had the power to empirically address such issues and that would have illuminated the nature of our own emotional feelings. Despite modern brain imaging, the foundational neural mechanisms of such feelings cannot yet be studied in any *causal detail* in human beings, even though *correlative* analyses strongly indicate we can be confident that the major sources of control are subcortical (e.g., Damasio et al., 2000; Northoff et al., 2009), in ancient brain regions we share with other animals (Panksepp, 1982, 1998a). Now, with the ever-increasing acceptance of evolutionary views in the mind sciences, animal models can begin to fill the many gaps in our understanding of the primary-process affective foundations of human minds.

Currently, the functional details of human "mind flesh" and how it generates internal, subjectively experienced feelings must be inferred from imprecise measures—namely, subjective self-reports coupled with modern functional brain imaging, which finally support the basic emotion view (Vytal & Hamann, 2010). However, it has recently been noted that the correlations between brain and psychological changes using such techniques are quite consistently and suspiciously high (see Vul et al., 2009, with six commentaries). Because of the massive amount of averaging

needed to make functional sense of the data, many of the observed brain-psychology relationships may be due, in large part, to statistical artifacts that emerge in correlational analyses when data are pooled before computing correlation coefficients, a bias that stymied my own work in energy-balance regulation for a while (Panksepp, 1973).

Regrettably, and despite our scientific hubris, our impressive human brain-imaging tools in this area still resemble Galileo's spyglass more than the Hubble space telescope. Modern functional brain imaging largely provides evidence about *regions of interest* in the brain that deserve detailed experimental scrutiny. Such fine scrutiny is next to impossible to achieve in human research. But the techniques can give us statistical estimates of how various brain regions are working together (correlations in regional blood changes that yield statistical estimates of *connectivity maps*), which can be related to actual connectivities. Recent refinements allow the visualization of major tracts in the brain (pathways connecting brain regions) by using *diffusion tensor imaging* (DTI), which can even highlight what surgeons may have been doing to emotional networks during the era of refined psychosurgery—for example, converging evidence suggests that antidepressant neurosurgeries may have been amplifying the positive feelings of the SEEKING system (Schoene-Bake et al., 2010). This suggests that direct stimulation of the SEEKING system should exert antidepressant effects (see Schlaepfer's and Coenen's work below).

Some of the available *causal* tools in humans (for instance, psychopharmacological interventions and deep brain stimulation [DBS]) can be linked to psychological processes by correlations to subjective state changes. Recently, strong antidepressant effects have been observed in patients who had not responded to many other treatments, during localized deep brain stimulation (DBS) in the anterior cingulate region, especially Brodman Area 25 (Mayberg et al., 2005), which is the affective headwater of the GRIEF system (see Figure 9.1). Presumably the DBS of Area 25 disrupts functioning of the GRIEF system, providing rapid relief from depressive psychological pain. Using DBS in the anterior reaches of the SEEKING system (the nucleus accumbens) has yielded similar effects. For instance, Thomas Schlaepfer and colleagues (2009) report the following:

> After switching the stimulation on, one patient . . . spontaneously reported that he realized that he was in Cologne, that he never visited the famous Cologne Cathedral, and he planned on doing this in the immediate future, which he indeed did the day following the operation. Asked about

depressive symptomatology, he did not report any acute subjective changes. A second patient's immediate (60 s) reaction to stimulation was quite similar; she did not report any acute changes in depressive symptomatology but spontaneously mentioned that she wished to take up bowling again (a favorite pastime of hers 12 years ago, before onset of her depression). She noted, "This would be quite pleasurable." These immediate and unprompted behavioral responses demonstrate a sharp increase in exploratory motivation, consistent with the accumbens' role in reward-seeking behaviors. This is especially noteworthy given these patients' severe lack of motivation during their long depressive episode.

As already noted, we can now estimate how various distinct psychosurgical techniques that were utilized in past treatments of human mental disorders (for instance, treatment-resistant depressions) may yield benefits in individuals who have not received relief from other treatment. The effects seem to be due to convergent influences on hedonic pathways such as the medial forebrain bundle of the SEEKING system, providing another reasonable target for treatment-resistant depressions (Coenen et al., 2011; Schoene-Bake et al., 2010). Of course, experimental research questions such as this can only be piggybacked secondarily, with strict informed consent, on previously prescribed medical treatment strategies. Still, to develop such novel psychiatric tools, feasibility studies need to be conducted.

Techniques that are available for animal brain research, including electrical and chemical brain stimulation of specific neural systems, along with very detailed measures of regional brain chemistries, including gene expression profiles, are vastly more precise for guiding novel *causal* studies (Burgdorf et al., 2010). Because animals can't talk about their experiences, however, too many investigators believe we will never have access to their subjective minds. As argued throughout this book, such long-term (almost century-long) biases are demonstrably off the mark, so long as we recognize that the "rewards" and "punishments" obtained by artificial stimulation of specific brain regions are proof that certain brain changes *matter* to animals.

An appreciation of our mental emergence from a dim ancestral animalian past, coupled with an understanding of the subtle ways of evolution, strongly suggests that many other animals have affective survival values that are quite similar to our own. Thus, the sources of our primal emotional feelings are easiest to clarify through cross-species affective neuroscience. We must remember how much experimental work on our fellow animals has promoted medical advances of great importance for bettering human lives. Without animal research on insulin, tens of millions of children would

have died prematurely during the past century. Animal research can illuminate the basic principles of the neural mechanisms that govern our primary affects and related secondary-process learning mechanisms. Psychology, psychiatry, and psychotherapy will be changed, and enriched, when more scholars begin to work on these issues that are so directly germane to the problem of consciousness and human and animal well-being. There are reasons to believe that we can even re-envision the foundations of our cultural institutions, ranging from philosophical to religious perspectives (Davies, 2011; Thandeka, 2005; also see the symposium on the philosophy of affective neuroscience in the *Journal of Consciousness Studies* [Panksepp, Asma, et al., 2012]).

THE ANCESTRAL SOURCES OF CONSCIOUSNESS

The goal of this book was to provide an overview of our knowledge of these primal animalian substrates for the human spirit, and implications for helping humans, leaving out many details that could further supplement the general arguments. We have not fully discussed how ancient emotional systems interact with the higher cognitive abilities of humans, in the context of recognizing that evolution has yielded a branching bush of, at times, increasingly complex living beings, rather than a ladder of ascent. At present, we have less precise knowledge about these important interactions than about raw emotional processes and simple forms of learning such as classical and instrumental conditioning. However, it is surely our vast cerebral "thinking cap"—our extensive cortico-cognitive apparatus—that distinguishes us mentally from our animal ancestors. That adds layers of complexity that cannot be readily addressed with animal models (e.g., Harmon-Jones & Winkielman, 2007; Northoff et al., 2011).

As mammalian cerebral mantles enlarged and became more complex, our cognitive consciousness expanded accordingly, yielding higher (e.g., tertiary-process) forms of consciousness (Damasio, 1999), as well as a self-centered claim that language-based rationality is the foundation of human consciousness (Fogelin, 2003; McGilchrist, 2009). As discussed in Chapter 11, these "extended" forms of cognitive consciousness remain inextricably tethered to the more ancient, affective forms of being. An alternative view, not yet supported by existing neuroscience, is that cognitive consciousness emerged fundamentally from first-order capacities to perceive the external

world, with no critical linkages to affective feelings. That view seems plainly wrong, because when the subcortical affective substrates are largely destroyed, so are all forms of consciousness (Panksepp, 2005b, 2007a). Language is our most unique cerebral skill, but even that emerges through emotional guidance. Through language, however, we can uniquely study the *extended* tertiary-process cognitive-affective consciousness of humans. And this is why there continues to be enormous growth in descriptive (i.e., nonneuroscientific) emotion studies in psychology (Davidson et al., 2003; Lewis et al., 2008).

Damasio states that "extended consciousness is a bigger subject than core consciousness and yet it is easier to address scientifically" (1999, p. 201). We agree, even though that science is bound to be less mechanistic, and hence less informative, at least as far as causal issues are concerned. Because of the ease of study, however, approaches to the study of emotions that have their basis in various uses of human language tend to implicitly hinder the study of primary-process core affective consciousness in animals, just as strictly behaviorist views have tended to do. Research funding, and hence rapid progress, requires consensus in the scientific community. There is none when it comes to our primal animal emotions. Thus, little explicit neuroscientific work is being done on the emotional feelings of animals (with potentially profound implications for understanding human feelings), especially when compared to the renaissance of research on human emotions. The more ancient and foundational levels of such questions are being woefully neglected in the western intellectual tradition, even though these aspects are of great importance for revealing the nature not only of human emotional feelings but also the associated psychiatric disorders that afflict so many human lives.

I would suggest, in line with Damasio, that at this moment in Mind-Brain science, the topic of core affective consciousness is hard to empirically study in humans. But for substantive progress on many core issues that concern psychologists, it may now be more critically important to understand the *primary-process* evolutionary sources of human and animal feelings, rather than the extended cultural consciousness that gets so much attention. Many leaders within this field may not see it this way, but I believe they have not thought through the issues for all relevant neuroevolutionary perspectives, such as primary to tertiary levels of analysis. Indeed, if the primary-process affective gifts of nature are the

brain functions upon which our complex human mental apparatus still rests, it would be tragic to neglect the opportunities available to understand our deeper nature, which simply can't be illuminated as readily through human studies.

Although human beings may be justified in having considerable pride in the special qualities of our extended cognitive consciousness—such as our capacity to speak symbolically, which has created culture, civilizations, and our rich and detailed mental life—we have no robust ways of understanding the affective foundations of our own minds. That can only be revealed by studying comparable processes within other animals. Many of our higher mental functions are more like "tools of consciousness"—fully grounded and totally dependent on the integrity of subcortical processes described in this book. Thus, we are wise to cherish our perceptual apparatus—especially our acute hearing and vision—but if they are lost, we "only" lose many treasured *contents* of consciousness, while remaining fully conscious beings. Some components of the more ancient perceptual apparatus, namely our vestibular senses, are rarely experienced explicitly until they are injured. We would be wise to recognize that a scientific study of core affective consciousness in animals provides avenues, perhaps the only paths, to understanding that ancestral mind that supports our higher mental apparatus.

Because of this, our focus throughout this book has remained on the nature of primary-process affective forms of consciousness in animals without attempting to discuss the possible emotion-laden thoughts that *may* also exist in their tertiary-process minds—processes that are much harder to study in animals (Mendl et al., 2010; commentary by Panksepp, 2010a). We need to open up and invigorate the ongoing discussion about the nature of animal minds that was sealed—supposedly forever closed—by the behaviorist juggernaut almost a century ago. The behaviorists' vision, perhaps appropriate for their times, has now proved to be shortsighted in the era of neuroscience. It led to a premature discarding of the primary-process affective mind simply because the tertiary-process cognitive mind could not be studied well in animals. And their choices were made too easy because of the many premature uses of mentalistic concepts to explain animal behaviors by the intellectual descendants of Darwin (Romanes, 1882).

The residue of those decisions persists in our universities, in robust but incalculably negative ways, to this day. As behavioral neuroscientists

choose to be constrained by those old behavioristic concepts, scrubbed free of affect (e.g., "the reward system" and "reward prediction error"—see Chapter 3), there continues to be a tradition of practically no forthright discussion of affective processes in animals by neuroscientists. Emotional feelings have come to be scorned even by those who should know better. Rigorous scientists do not wish to see their work brushed off as anthropomorphism (Figure 1.3). But the preponderant weight of neuroscientific evidence indicates that those entities of the world called "rewards" and "punishments" are in fact constituted by affective changes within the brain. Indeed, the *unconditioned emotional response* systems of the brain that control the coordination of a symphony of emotional actions are not unconscious. Those brain networks are the very source of emotional feelings. That small shift in perspective could do much to enliven how we use preclinical models of psychiatric disorders. We live in an era where the widely used scientific *concept* of "reinforcement" should be seen as potentially little more than a shorthand summary term for the complex way that core affects—the unconditioned stimuli and unconditioned responses of animals—operate in the midst of the fluctuating events of the world. The pervasive *procedure* of reinforcement is highly effective, but the *process* of reinforcement remains an assumed, rather than a demonstrated, function of the brain. Now, "reinforcement" is widely assumed to be a *real* brain *process*, but it may turn out to be little more than the phlogiston of behavioral analysis (see footnote 2 in Chapter 3) that has gradually been accepted as a description of reality.

Because the Pandora's Box of the animal mind was sealed several generations ago, an adequate discussion of animal emotional functions remains to be fully engaged, especially by those in the academic community best situated to do the necessary research. Had the debate been opened up among neuroscientists in a timely manner (at least by the 1970s), as some earnestly tried to do in cognitive ethology (e.g., Donald Griffin, 1915–2003), we might be in a better position now to address the subjective aspects of ancestral affective minds in both humans and other animals.

In any event, the cognitive forms of consciousness—that is, thoughts about our specific circumstances in the world that integrate declarative and autobiographical memories—are intrinsically harder to study mechanistically than core affects, especially in animals. One simple reason is that, as far as we know, specific cognitions do not have the clear-cut

neural pathways that primal emotions have. Further, neurochemical codes for the core affects include a host of neuropeptides that regulate specific affects (Figure 13.1). That fact is a blessing for cross-species predictions. Future neuropeptide research should be able to test whether those neural controls can produce comparable affective changes in both animals and humans; these types of effects have already been well validated with oxytocin and social feelings (see Panksepp, 1992, 2009c; Pincus et al., 2010).

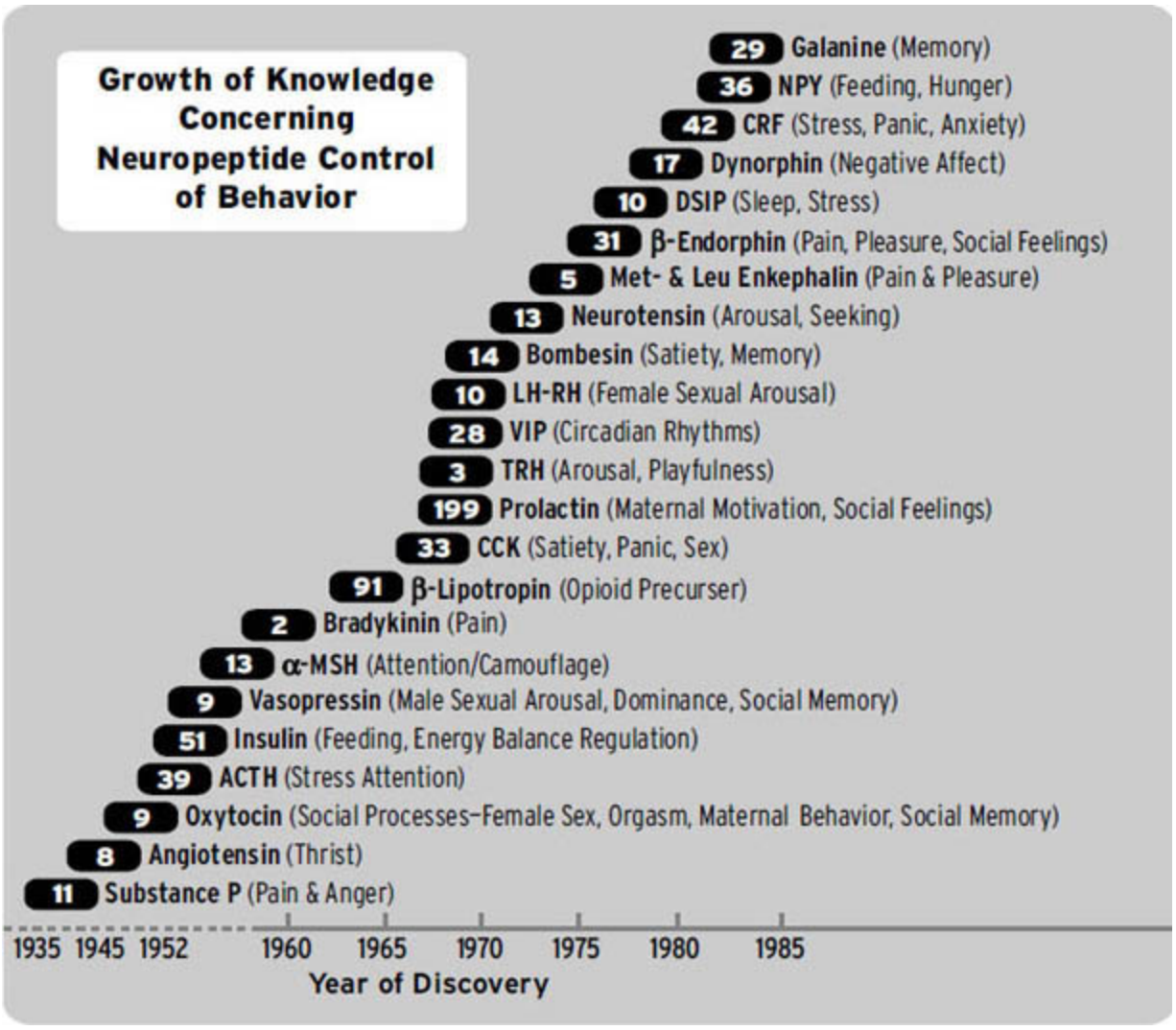

Figure 13.1. The time line of the discovery of major neuropeptides that participate in various brain functions related to the control of behavior and various emotional and motivational processes. Progress was slow in the

beginning (see the dotted line) but sped up enormously around 1970. The numbers inside squares indicate the number of amino acids in each of these neuropeptides (from Panksepp, 1998a; adapted with the permission of Oxford University Press).

In other words, the abundance of neurochemical coding of affects, as compared to cognitions, allows a rich commerce in predictions to flow across the solid neuroscience bridge from animal brain to human mind research, and back again. In contrast, all our cognitions ride *more strictly* along dynamically changing glutamatergic excitatory transmissions, sculpted by GABAergic inhibitory guidance mechanisms. In addition, cortical cognitions and perceptions also surely require more complex and rapid neuronal firing patterns than do subcortical core affective feelings. The massively complex neurodynamics that control cognitions are bound to vary much more from species to species. That would make a neuroscientific understanding of higher mental processes a far more difficult problem than the cross-species study of evolutionarily conserved primary-process affects.

Although thoughts about thoughts, mediated by propositional language—so well developed in mature humans—surely do not exist in most other animals, we know of no way to rule out such possibilities. It is possible that other mammals think more in terms of internal perceptual images, which may better reflect ancestral forms of animal memory and thought (Grandin, 2005). But that is hard to evaluate except through the *correlates* of global brain imaging (e.g., PET scans). Good causal research is currently next to impossible to conduct. Thus, we must hold off on any firm conclusions concerning homologies at the tertiary-process level of human and animal cognitions. On the other hand, basic secondary-process learning mechanisms that underlie conditioned behaviors, largely unconscious neural mechanisms, allow very effective cross-species translations (LeDoux, 1996, 2007).

However, there is no evidence that such conditional controls add much diversity to the *types* of affective feelings that animals experience, because learning just modifies the intensity as well as the temporal and spatial expression of emotions. For instance, as conditioned responses come to replace unconditioned responses (UCRs), it is to be expected that affect diminishes substantially. The critically important UCR mechanisms of the

brain may be essential not only for generating affect, but for providing the "glue"—the "reinforcement"—whereby conditioned stimuli come to evoke conditioned responses (see Chapter 6).

Likewise, an understanding of primary-process affects and associated conditioning processes may be of critical importance for our own tertiary-process, self-reflective tendencies, although we currently have no clear scientific data on how that happens. Obviously, all our higher mental complexities—from the conduct of science to philosophy, psychoanalysis, and the arts—require us to keep in mind many symbolic, language-based memories as well as vast patterns of past and future possibilities. We know of no animal with comparable cerebral skills, but we do know that many other animals are quite smart in their own ways (Romanes, 1882, to Griffin, 2001) and that they are surely vibrant affective creatures. This gives us very special responsibilities for the way we conduct our research and for the way that we care for all animal life. The implications of this knowledge for animal-welfare issues are vast (Bekoff, 2000; Grandin, 2005; McMillan, 2005).

THOSE EVER-PRESENT COGNITIVE-AFFECTIVE INTERACTIONS

We have intentionally not dwelled on the nature of our highly resolved perceptual-cognitive mental apparatus. In doing so, we do not deny the importance of our unique extended consciousness in making us fully and deliberatively human. Our goal has been to open up intellectual space for a realistic and effective scientific confrontation with the ancestral sources of human passions by cultivating a view that exhibits full respect for the emotional lives of other animals. I have no wish to diminish the many important differences that each species of animal and each unique human being and human culture brings to the rich banquet of mental existence in this world of ours. I have primarily sought those general principles of mind that still bind us to each other . . . like an extended family that needs to review its shared ancestral treasures. There are now many empirical findings that support the views I have advanced, and there is much more to be unearthed.

The widespread claim that affects are just a variant of cognitions seems little more than a word game to me, even though I certainly accept that the

many valenced (good and bad) feelings of the nervous system are always interacting with cognitions (imagination, learning, memory, thoughts) within the full complexities of most human and animal minds. The same can be said for attention and primal motivation (e.g., thirst and hunger) and indeed for all the ancestral faculties of the mind. Just like the organs of the body, everything inside us interacts. The first lesson that I learned in neuroscience is that the frequency-specific ripples of a stroboscopic visual signal entering the eye can be measured in practically every corner of the brain. This does not mean that in our pursuit of real progress in understanding vision we should not distinguish the visual system from the auditory system, or from other brain sensory systems.

For those who insist that affects and cognitions are totally conflated in the human MindBrain (which would probably include most psychologists and philosophers these days), I would suggest they consider their arguments not only from their perspective, but also the bottom-up perspectives advanced here. When we look down on mental life from a corticocognitive pedestal, yes, everything interacts. However, if we understand that cognitions are often "handmaidens" (or emissaries for the affects) such conflations no longer work. Positive emotions, which so far cannot be "computed" in any meaningful way, can directly promote bottom-up facilitation of positive affective homeostasis, or a restoration of well-regulated mental balance, when humans are beset by negative feelings. Top-down cognitive skills also effectively serve to seek out a large variety of positive affects that can counteract negative feelings.

Thus, here is the critical question: Do cognitions and affects operate by very similar neuronal principles, and in the same regions of the brain? The evolutionary bottom-up view preferred here gives us more than three good reasons to insist that primary-process affects have an independent existence that goes back much further in MindBrain evolution than the brain processes typically subsumed by the concept of cognition:

1. The emotional-affective presence of animals and humans remains remarkably intact after they lose their prime cognitive territories—their neocortex—early in life.

2. To this question, "Are there major neurophysiological differences between the premier territories of cognitive processing (i.e., the thalamic-neocortical axis) and those enriched in emotional-affective processing (i.e., the subcortical and cortical midline systems, or SCMS [see Northoff & Panksepp, 2008], traditionally called the extended limbic system)?" The answer is *yes*. Just in terms of firing rates of neurons, cognitive-somatic territories are enriched in very highly firing neurons (e.g., hundreds of action potentials per second), while the affective-

visceral ones abound in very slowly firing neurons (e.g., it is hard to find many that fire more than ten times a second).

3. There is no place in the normal neocortex or thalamus where you can stimulate a local region of the brain and consistently get the same cognition or thought over and over again (albeit Penfield demonstrated that one can obtain stereotyped perceptual phenomena by stimulating certain temporal lobe regions that border on the limbic system). By contrast, it is easy to find places within the SCMS where one can repeatedly stimulate the same brain locations and get the same affective states.

The ancient MindBrain substrates for emotional affects are not only governors of how we behave, but they also prompt us to dwell on the complexities of our lives as we navigate social worlds. More than anything, the distinction between affects and cognitions, interwoven as they are in the intact brain, allows us to grant that other animals also have experiences along the full spectrum of intrinsic survival values. They have affectively experienced states of the nervous system that are not terribly dissimilar to our own.

The recognition of this fact gives us a special responsibility to do our research with an abiding sensitivity, with deep respect and concern for the animals we sacrifice to obtain such knowledge (McMillan, 2005). Regrettably, a fuller understanding of the human condition and its emotional travails cannot be developed without scientific work on the relevant brain systems in animals. This conclusion is inextricable from the fact that they are sentient beings, and their affective capacities arise from the same type of neural soil as we have. Humans may be abundantly more "rational," and more "reflective" about their states of mind, but mammals all experience emotions affectively. And as the clinical studies of Merker (2007) and Shewmon et al. (1999) have revealed, those feelings arise from very deep regions of both human and animal brains (Figure 13.2). Obviously, we humans can dwell on the existential aspects of our lives more deeply than any other species. After all, we can speak and think symbolically. But this does not give us privileged access to raw affective experiences. What a terribly empty and lonely world it would be if we humans were the only conscious creatures within the inextricably interwoven fabric of life. What a wonderful relief it is when we realize that there are bubbles of consciousness wherever our fellow animals roam the earth.

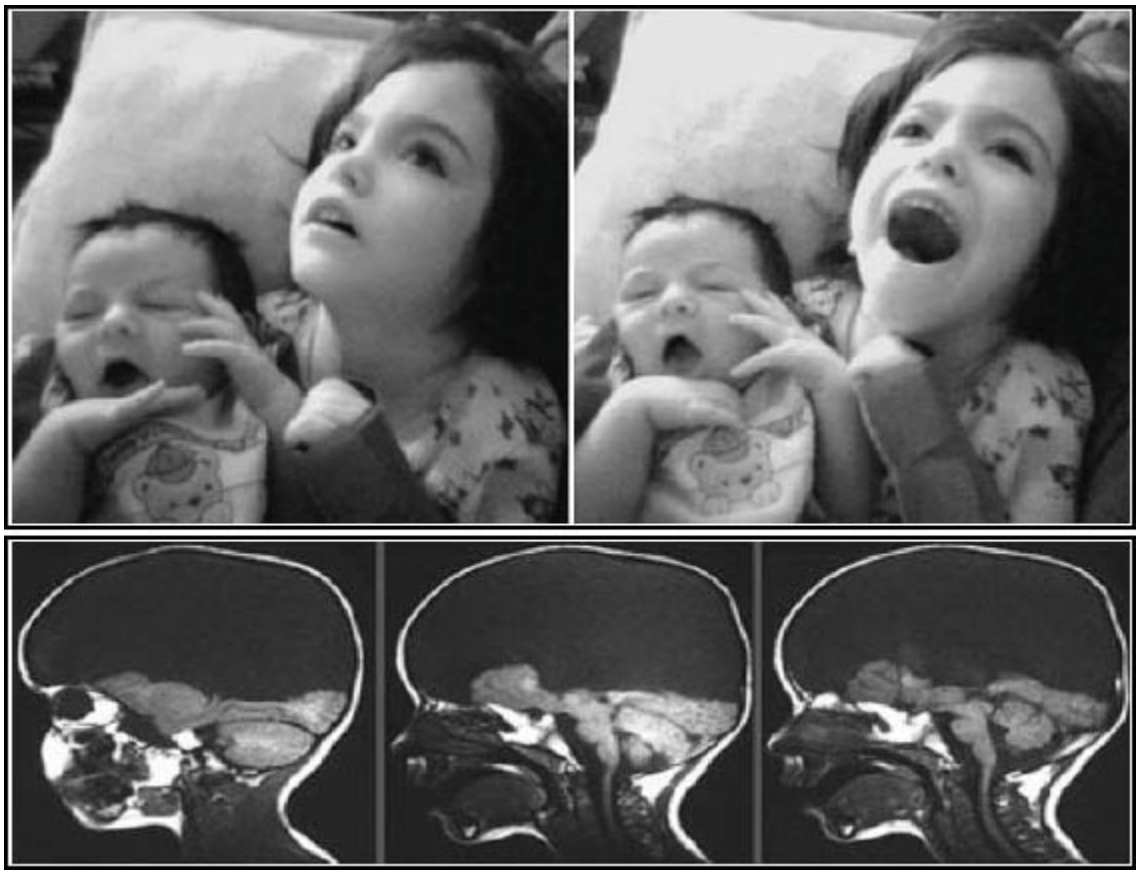

Figure 13.2. The emotional response of an anencephalic child to a baby being placed on her lap (top). The type of brain dysgenesis of such children (bottom) (data from Merker, 2007; I thank Bjorn Merker for use of the photographs. Radiographs reprinted with permission of the American College of Radiology (ACR Learning File, Neuroradiology, Edition 2, 2004). No other representation of this material is authorized without expressed, written permission from the American College of Radiology).

THE LOSS OF "MEANING" DURING THE TWENTIETH CENTURY

Early in the twentieth century behavioral science lost its connection to the intrinsic values of our mental apparatus. Along with remarkable advances in physics, astronomy, chemistry, and brain sciences during the twentieth century, the prevailing scientific view emerged that human existence, just as physical science had affirmed for the material world, is value-free, or inherently meaningless. Many psychologists, especially behaviorists,

assumed that was the only correct way to proceed in our studies of both mice and men. Subjectively experienced states of the nervous system were discarded from scientific discussions, albeit not from intellectual debates. Academic psychology thereby became alienated from affective feelings. The cold hard evidence harvested from animal bodies and their behaviors was all that mattered.

To a substantial extent, this was reflected in intellectual life at large. The classic "existentialist" position was that little of intrinsic value is the birthright—the "essence"—of each individual, and people have to generate meaning afresh by the lives they lead every day. Life was intrinsically absurd, with no more meaning than people constructed within their own existence. On one hand, this engendered nihilism; on the other, it encouraged the view that all "meaning" was created by each person. This may well be true for the tertiary aspects of mind, where mirror neurons, empathy, and fellow feelings have to be infused into neocortical matrices by culture, education, learning, and individual development. But it does not well describe the affective tools that evolution has built into the lower reaches of our mental apparatus (Panksepp & Northoff, 2009).

With the emergence of scientific analyses of behavior and the mind, remarkably simple and effective ways to study learning and memory were developed. One could use any old neutral signal—a tone, a flash of light, a tap on the shoulder so to speak—as the CS (conditioning stimulus) and then follow it with an electric shock or some equally potent UCS (the unconditioned stimulus, or stimuli), and within a handful of trials animals would begin to flee and freeze and poop, with their blood pressure rising as their hearts pitter-pattered, just from the presentation of the CS. All this only worked because so many UCS were capable of producing so many UCRs, which were profoundly important instinctual functions of the nervous system. These same UCSs could be used to train animals to do any of a variety of things in "instrumental conditioning" (e.g., running mazes) or operant (e.g., lever-pressing) procedures. It all worked like clockwork, but was this because animals were "just" machines?[1] Or did all this happen because evolution had built primal affective experiences into the neural matrices of many UCS and UCR networks of the brain? Scientists simply regarded animals as unfeeling machines. Largely left unstudied were the instinctual UCRs, which, at least in the realm of emotions (e.g., the FEAR

system), are essential for fear-conditioning to proceed as effectively as it does.

An often forgotten historical note is that few of the scientists who studied all the environmental parameters of conditioning, and eventually the brain mechanisms of emotional conditioning, paused to intensively study the nature of their experimental subjects' UCRs. Of course, that would have been difficult in the heyday of behaviorism, for it would have required considerable knowledge of the nervous system and intensive brain research. It might also have required scientists to entertain the realistic possibility that UCRs were constituted, in substantial part, by affective primary-process changes within a BrainMind. And thus, the various MindBrain consequences of UCS that these scientists used to train their animals, under the rubric of "rewards and punishments," were never conceptualized as provoking raw affective states evolutionarily embedded within the nervous system. After Thorndike's Law of Affect was transformed into a Law of Effect (i.e., the "satisfactions" and "discomforts" of the world were transformed into "rewards" and "punishments"; see Chapter 2), the scientific conversation about emotional feelings in animals almost ceased.

Now that we know that most unconditional stimuli that are used in experimental animal psychology derive their power from the fact that they evoke not only objective behavioral changes but also subjectively experienced affective changes within the brain (i.e., various distinct types of emotional UCRs), it is of utmost importance to develop clearer visions, and more affectively focused research programs, in order to unravel the nature of the many affective "instincts" of the brain. But that is now more difficult than it should be, for even the word "instinct" fell into disrepute during that era of ultra-positivism. Also, for contemporary scientists to shift their research priorities, there needs to be a societal shift in "reinforcement contingencies," specifically, the magnanimous sources of research funding (e.g., the National Institutes of Health and National Science Foundation) need to encourage a more flexible neuropsychological approach to the study of animal emotions, especially on the many affective UCRs of animal brains. If we do not do that, we will never know how human emotions evolved. That has yet to happen, so few scientists speak frankly (and in evidence-based ways) about the affective states that other creatures experience. If we understood these primal emotions, we would also have a better way of conceptualizing the psychological and motoric coherence of

organismic actions, and we could thereby better understand how the core-SELF and the whole brain works as a unit.

Although the breadth and depth of our human consciousness has been widened enormously by the intellectual potentials of our enlarged brains and cultures, we are, in fact, inheritors of ancient biological values that constitute the very ground of meaning within our minds. Regrettably, this affective ground of meaning can be difficult to talk about. Thus, our greatest recent gift, the discovery of language, is both a blessing and a curse. Besides bringing us beautiful songs, poems, and other literature, it is also ideally designed for sowing disagreement, dissension, and the marginalization of other humans, along with our shared animalian nature. The primary aspects of mind cannot be understood simply through the use of words. This understanding requires neuroscientific inquiries that do not reject the mental attributes of ancient brain functions. Twentieth-century philosophy was not often of much assistance in breaking through the cultural resistances that are finally leading to neuroscientific progress in understanding mental dynamics. Ludwig Wittgenstein's influential philosophy gives us a window to the dilemma that has prevented progress in understanding the mental life of humans and especially other animals.

THE AGONY OF WITTGENSTEIN: MAN'S SEARCH FOR MEANING

During the past century, many sought human meaning in the way we use words. While the poets and composers demonstrated how well we could artistically symbolize our deepest longings, joys, and despairs, others sought the grounding of our being and meaning within the logic of language. Ludwig Wittgenstein (1889–1951) proceeded along that path in his search for the ultimate logic of language. In his *Tractatus Logico-Philosophicus*, he sought to provide a definitive statement about how the structure of language was related to the structure of the world. This manifesto consists of seven propositions, with a host of subpropositions. The most relevant for our discussion of emotions is Proposition 6.5:

> When the answer cannot be put into words,
> neither can the question be put into words.
> *The riddle* does not exist.
> If a question can be framed at all, it is

also possible to answer it.

If one applies this rule to the affective topics covered in this book, the question is can there be a credible scientific answer about the nature of emotional feelings? I believe that this can finally be achieved, but only because of recent advances in neuroscience. We have now learned enough about the intricacies of the remarkable brain to envision how mind emerges from neurodynamics, constructed in close association with bodily states and environmental conditions. In a subproposition (6.52), Wittgenstein goes on to assert:

> We feel that even when all *possible* scientific questions have been answered, the problems of life remain completely untouched. Of course there are then no questions left, and this itself is the answer.

In the epistemology of emotions, one could suggest that this is an assertion of profound skepticism about the possibility of ever addressing the deepest aspects of nature, such as affective consciousness. Indeed, there are scholars who believe that it is impossible to study the source of our basic values scientifically. But the brain sciences of Darwin's day, not to mention Wittgenstein's, were primitive in comparison to what we now have. The closest Wittgenstein ever came to acknowledging feelings was in his cryptic outline of mental propositions in 6.522:

> There are, indeed, things that cannot be put into words. They *make themselves manifest*. They are what is mystical.

In short, his logic of language could not deal with affective mysteries. Wittgenstein summed up his views in the *Tractatus* in his introductory remarks as well as his final standalone seventh proposition: "What can be said at all can be said clearly, and *what we cannot talk about we must pass over in silence*." Emotional feelings were, in his time—during the era of *behaviorism* in psychology—among those "mystical" aspects of the world that lay outside the realm of propositional logic, beyond words, impenetrable to science. Such spooky aspects of mind seemed to lie forever outside coherent scientific analysis. To this day, it is still not widely recognized that this kind of scientific understanding is now possible, or that

it must be critically linked to identifying the brain processes that create our emotional feelings and values (Panksepp, 1998b; Russell, 2003).

Soon after finishing his "definitive" statement on the linguistic foundations of knowledge, Wittgenstein recognized that his world system was deeply flawed. He spent the rest of his tortured emotional life probing how meaning emerged from the incredibly flexible ways we use words. In his second renowned book, *Philosophic Investigations* (1953/1967), on which he labored for 20 years, published 2 years after his death, Wittgenstein made almost a 180-degree shift away from the starkness of the *Tractatus*, and he started to probe how we create meaning out of the infinite variety of "language games."

His intellectual legacy was to leave us with a view of mental life where meaning was a flexible manifestation of how we play with words. This has remained one of the hallmarks of postmodern culture, as well as of the trajectories of emotion research in the social sciences—a study of how we use words and how we semantically construct emotions. Only recently have scholars working in that tradition entertained the idea that there is a deeper neurobiological reality to emotional feelings—that, at the very least, there are natural mechanisms for the dimensions of positive and negative affects within the brain (Russell, 2003). This is progress, but it is short of the mark when it comes to primary-process emotional systems. Perhaps dimensional theories of emotion work best at the tertiary-process level, where the varieties of our affective lives are translated in simplified emotional concepts that can facilitate research (Zachar & Ellis, 2012; also see the special issue of *Emotion Review* [2011] that Jim Russell and colleagues have edited to better capture what "basic" emotion theorists are talking about). In any event, basic emotion and dimensional views of emotions can work well together if they reflect different levels of Mind-Brain organization (Panksepp, 2007d). As already noted, robust evidence for basic emotions does exist from modern human-brain imaging (Vytal & Hamann, 2010).

As one ponders human nature, it seems that ultimately few in mind science can resist the siren-song attractions of naturalism. Even Wittgenstein, in the aforementioned second book, remarked how "the human body is the best picture of the human soul" (Part II, p. 178). It is just such a vision—with a focus on the "body" represented within the brain—that we have independently sought in order to understand how the primary-

process emotional systems actually create feelings within mammalian brains (Panksepp, 1998b).

Damasio's theorizing is progressing along this path, shifting gradually away from the cortical sources of our feelings to the recognition that emotional-affective processes arise from deep subcortical regions. As I concluded this book, I was pleased that Damasio's (2010) views, as was previously noted, are shifting closer to my own. I trust many will follow this wise scholar. In *Self Comes to Mind*, he recognizes the diverse subcortical affective networks of the brain, where through evolution, the primal mind arose from (or with) the primal SELF. Varieties of ancient *emotional, homeostatic*, and *sensory* affects are intrinsic functions of the brain, triggered and modulated by various bodily inputs (Denton, 2006). However, only the emotional affects are of profound importance for a scientific psychiatry yielding, I hope, better conceptualizations of what clinicians need to achieve through psychotherapy.

It is ironic that Wittgenstein was personally so close to, yet intellectually so far from, an understanding of preverbal mentality tethered to ancient BrainMind realities. As is so movingly depicted in his choice of an epigraph for his second book—from St Augustine's *Confessions* (section I.8; see the beginning of this chapter)—language emerges, in part, from a child's affective engagements with the social world. Out of Wittgenstein's confusions about the fundamental nature of the mind, and what we can and cannot understand through neuroscience, he wrought an excessively relativistic view of human nature and human interactions, quite appropriate for tertiary-process regions of the human MindBrain. But this incomplete vision currently prevents psychology from becoming a whole science that fully seeks to understand the true evolutionary undergirding of mental life.

Perhaps it goes back to the difficult and prolonged process whereby we learn to speak. Attuned human intersubjectivity, which is central to the language-acquisition enterprise, includes the rhythmic nonverbal social signals—a natural body language—that is related to the "seeking, having, rejecting, or avoiding" of worldly objects that can become the targets of our "own desires." Human existence is not *just* a matter of sensory associations, even though those associations eventually fill our mental landscape to a point where, at times, we recognize little else. It should be kept in mind that even though the expansive human neocortex is a relativistic organ of the kind that Wittgenstein envisioned, it is not the subcortical terrain we share

with all other mammals. The vast computational spaces of our neocortex are quite empty of psychological content at birth, and practically everything it eventually comes to know—*noetic* and *autonoetic consciousness*, in Endel Tulving's terms (see Vandekerckhove & Panksepp, 2009)—is learned. That is not the case below the neocortex, where our *anoetic* consciousness, without understanding, resides.

Our ancestral brains contain special types of meaning based on genetic inheritance; the potentials for raw feelings are built into the instinctual (i.e., inherited) neural action apparatus of the body. And there are many varieties of those feelings. Some are closely linked to sensory inputs from the outside (the pleasures and displeasures of sensation), others are linked to internal bodily inputs to the brain (e.g. hunger, thirst, and their satisfactions), and yet others that reflect the action dynamics are evolutionarily built into the brain, at least in raw form. All of these tools for living are plastic to a degree; they can be strengthened and weakened by experiences. These raw feelings are closely linked to our intrinsic urges to reach out into the world in certain ways and to respond to the archetypal challenges we encounter. They do not have higher order intentionality (i.e., "intentions *to* act") but they do have intrinsic intentionality ("intentions *in* action"; see Panksepp, 2003a, Figures 1.4 and 1.8). Feelings are what make us active organisms as opposed to simply passive information-processing machines.

Many may agree that emotional feelings are the roots of our earliest human communications. But many still regard them as a variant of sensory rather than motor processes, that is, more passive (i.e., *the feeling of what happens*—probably a tertiary-process viewpoint) than active (i.e., *By God, I'm going to make this happen!*—a primary-process perspective). In fact, emotional feelings and consciousness itself may be premised as much on motor-action processes as on sensory-perceptual ones (see Chapter 12). Our perceiving minds as well as our ancestral affective minds appear to be anchored in action coordinates, which are the various instinctual emotional actions that we can easily recognize across mammalian species. The central role of the instinctual action apparatus has traditionally been marginalized in the analysis of emotional feelings and consciousness. It is often seen simply as an "output" of the nervous system rather than as a complex integration process. As Darwin suggested, emotionally expressive actions provide coherent images of our basic emotional nature. Our earliest engagements with the world are spontaneously active. Just look at any

infant, any child, any young vertebrate: SEEKING lies at the foundation of all of their aspirations.

AFFECTIVE OPTIONS AND OPINIONS

We are faced with a stark choice. Either we and other animals are inheritors of a variety of intrinsic values, representing the affective potentials of our brains or we are nonfeeling zombies who can be studied as pieces of machinery. Which option do we choose? How we answer this question in neuroscience will determine what type of knowledge, and perhaps what kind of culture, we will create.

Let us not underestimate the magnitude of the scientific problem before us. All aspects of consciousness emerge in animal and human brains as the result of the interactions of widespread neuronal networks. There is no single circuit or "center" for consciousness, even though there are critical convergence points (Sukhotinsky, et al., 2007). As I have long argued, the PAG may be the most important location in the brain, because it is richly connected to both higher and lower brain functions. It is a Grand Central Station for our affective life, and it is essential for the primal integration of diverse emotional experiences. It sends its tentacles far into the lower and higher regions of the brain. Much of this kind of "dark energy" in the brain is not easily visualized with modern brain-imaging technologies (Zhang & Raichle, 2010), but with the right tasks, remarkable images can be generated (Mobbs et al., 2009).

The PAG and its related brain-stem networks are essential for the construction of the higher mind, where distributed but specialized network models of the brain are more realistic than highly predetermined modular specializations, as many evolutionary psychologists are prone to assume. When we begin to envision the myriads of neurons and neural networks, with their seemingly endless neurochemistries, influencing each other in multiple re-entrant loops of activities—feeding upon each other and themselves—and generating diverse global field dynamics that are presently almost impossible to measure, we are humbled, at the outset, by the complexity of the task of deciphering how the BrainMind actually works in detail. But if we want to know ourselves, we have to proceed down this path, step by step. Understanding how the brain generates primal

emotional feelings may be the most solvable—the "simplest"—problem in consciousness studies.

Consciousness is surely not a single global property of the brain in action. It has a long evolutionary history that goes back to ancient systems that encode brain and body states that are essential for survival. Psychologically, those "ancestral voices of the genes" that arise from the neurodynamics of a variety of intrinsic brain systems are experienced as raw feelings or primitive affective states. We have focused on other mammals (and some birds; see Bernroider and Panksepp, 2011) largely because the neuroanatomical and neurochemical homologies are quite striking, allowing credible cross-species generalizations.

The issue of consciousness among invertebrate species is a more difficult issue because of diminishing neural similarities. But as we previously mentioned, even crayfish (basically large insects) exhibit conditioned place preferences for drugs that humans abuse and that other mammalian species find rewarding (Huber et al., 2011; Nathaniel et al., 2009, 2010; Panksepp & Huber, 2004). Thus, it is wisest to remain open-minded about these issues in the "lower" species and to see where the predictions lead us. But there is a core dilemma in neuroscience. In mind science, we would like to understand large-scale processes—the "wholes"—but neuroscience is best at studying small discrete phenomena, or the "parts" of the "wholes." Because of this tendency, we are very susceptible to mixing up the two, yielding mereological fallacies, namely part–whole confusions (Bennett & Hacker, 2003). And currently neuroscience is giving so many parts—so many brain mechanisms—but what functions they perform in the mind, the 'whole', is more difficult to decipher.

Scientists would like to understand the world, but they know that their techniques are much better at studying the parts of nature rather than its composite wholes. Different people have different solutions to this dilemma. A common one is to focus on rather narrow problems (out of sheer necessity, this is favored by scientists), where one begins to see each leaf on a tree ever more clearly, but then they all too often lose sight of both the trees and the forests. Most are bound to pay heed to Rene Descartes' (1596–1650) third rule of science in his *Discourse on Method*: "*To think in an orderly fashion when concerned with the search for truth, beginning with the things which were simplest and easiest to understand, and gradually and by degrees, reaching toward more complex knowledge, even treating,*

as though ordered, materials which were not necessarily so." Or, as Einstein is reputed as saying, "*Simplify, but not more than is necessary*" (emphasis added). That is the path we have taken here in our attempt to understand affective consciousness in humans.

Living brains, along with their minds—the invisible manifestation of their network-level neurobiological functions—reflect a delicate balance, as yet poorly understood, among vastly interacting neural circuits that work in and for living bodies and that respond to the challenges of the world by creating desired circumstances and avoiding those that are harmful. Emotional feelings are the experienced affective manifestations of such interactions; they are the subjective qualities of mind, aspects of which can finally be studied systematically, in detail, in other creatures. Thereby, we can begin to neuroscientifically understand our own minds. That understanding cannot be achieved without studying the relevant processes in other animals. Just as with the other success stories in biological science that have heralded medical progress, understanding has been guided, every step of the way, by findings from animal research. As Charles Darwin recognized, the knowledge we gain will have profound implications for understanding the human condition. As a species we still have much to learn about ourselves. What are we waiting for?

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First Edition

Book design by MidAtlantic Publishing Services
Production manager: Leeann Graham

Library of Congress Cataloging-in-Publication Data

Panksepp, Jaak, 1943-
The archaeology of mind : neuroevolutionary origins of human emotions / Jaak Panksepp, Lucy Biven. -- 1st ed.
p. cm. -- (A Norton professional book) (The Norton series on interpersonal neurobiology)
Includes bibliographical references and index.
ISBN 978-0-393-70531-7 (hardcover)
1. Emotions. 2. Emotions and cognition. 3. Memory. I. Biven, Lucy. II. Title.
BF531.P36 2012
152.4--dc23

2012010323s

ISBN: 978-0-393-70531-7
EISBN: 978-0-393-70731-1

W. W. Norton & Company, Inc., 500 Fifth Avenue, New York, N.Y. 10110
www.wwnorton.com
W. W. Norton & Company Ltd., Castle House, 75/76 Wells Street, London W1T 3QT

1 2 3 4 5 6 7 8 9 0

[1]We must remember that subcortical neurons in emotional regions typically fire much more slowly than in higher sensory-perceptual regions of the thalamus and neocortex. In many systems, the clustering of neural firing as opposed to their higher patterned rates is more important. Often the power and sustained actions of certain affective-visceral neurochemicals (e.g., neuropeptides) is more important in the patterning of emotional feelings and responses than elevations of neural firing rates, and the resulting increases in blood flow, which techniques such as PET and fMRI monitor. Thus, these techniques are not as sensitive to lower-affective brain functions as to higher-cognitive ones, which yields an undesirable bias in the way these techniques are being used to study emotions: They are more sensitive to the accompanying cognitive-thoughtful correlates of emotions than the affective substrates.

[2]The current era of brain imaging, although based on much more accurate brain activity measures (blood flow and metabolic changes), is still yielding images of brain functions that do not accurately reflect the underlying brain activities that generate mental events. They simply give us a more accurate estimate of which brain areas may be most important to investigate in order to tell us how the Mind-Brain is organized. As most practitioners know, this new "phrenology" still has many challenges to face and troublesome flaws in any attempt to translate brain activity to mind processes.

[1]At present there is increasing interest in animals that become especially interested and interactive with stimuli that predict rewards, namely those that are “sign-trackers” and those that seem most interested in the forthcoming food, the so called “goal-trackers”. The former exhibit more brain dopamine arousal in response to the anticipatory stimuli than the rewards themselves, while the latter continue to exhibit more modest arousal to both the predictive and goal stimuli. This seems to reflect a temperamental characteristic of the underlying SEEKING system. Sign-trackers are more likely to get addicted to drugs like cocaine than goal-trackers (Flagel et al., 2011).

[2]"Phlogiston" was the name early physicists gave to an imaginary (theoretically postulated) substance that combustible materials contained that allowed them to be burned. After they were burned down to ashes, the substances were thought to be "dephlogistonated." This, of course, proved to be a name for an entity that did not really exist; it was used to generate a feeling of understanding, before there was any.

[1] An *opponent process* is one that tends to directly dampen the effects of a process that precedes and triggers it. For instance, the positive affect of certain addictive drugs is internally counteracted by the build-up of negative affective feelings inside the brain, which leads to the distress of drug withdrawal.

[1]Benzodiazepines are also called minor tranquilizers or antianxiety agents, the earliest of which were chlordiazepoxide and diazepam (the brand names are Librium and Valium). Now there are many other kinds of minor tranquilizers, some of which are also sold as sleeping pills or muscle relaxants.

[1] http://www.emory.edu/INTELNET/fi.hasid.html

[1]For instance, we have long been puzzled at the fact that very low levels of electrical stimulation of the FEAR system in the hypothalamus can promote freezing while increasing the current can provoke flight (Panksepp, Sacks et al., 1991). How can such diametrically different fear responses share a common neural substrate? One theoretical way out of this conundrum is to suppose that at the higher current levels our localized brain stimulation is spreading to the SEEKING system, which unloosens animals from immobile freezing, into the remarkable strides of flight as they "seek safety"—clearly a state that should feel more pleasant to the animal. It should be full of hope! This suggests a fascinating theoretical possibility of how the terror of FEAR may explode into the optimistic eagerness, at times almost playfulness, of flight. Unfortunately, this idea remains to be subjected to rigorous neuroscientific evaluation, so it should not be seen as a conclusion but only as a working hypothesis for further research.

[1]This and the following chapter were written entirely by Jaak Panksepp in order to share a vision (along with some personal reminiscences) about how knowledge of mammalian emotions could help advance the science of biological psychiatry as well as the development of new psychotherapeutic approaches that may be quite controversial.

[2]It should be noted that a great deal of wonderful work on the details of the unconditional mammalian FEAR system has emerged from several laboratories in Brazil, most prominently from investigators working with Frederico Graeff (e.g., Del-Ben & Graeff, 2009) and Marcus Brandão (Brandão et al., 2005).

[1]The computer revolution promoted the notion that deeply biological minds could be computed on silicon platforms—a vision that prevailed in the new cognitive sciences and seems alive and well in many other corners of the academy (see Panksepp, 2008c).