

Neurologizing the Psychology of Affects

How Appraisal-Based Constructivism and Basic Emotion Theory Can Coexist

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ABSTRACT—*Abundant neurobehavioral data, not discussed by Lisa Feldman Barrett (2006), support the existence of a variety of core emotional operating systems in ancient subneocortical regions of the brain (Panksepp, 1998a, 2005a). Such brain systems are the primary-process ancestral birthrights of all mammals. There may be as many genetically and neurochemically coded subcortical affect systems in emotionally rich medial regions of the brain as there are “natural” emotional action systems in the brain. When emotional primes are aroused directly, as with local electrical or chemical stimulation, the affective changes sustain conditioned place preferences and place aversions, which are the premier secondary-process indices of affective states in animals. Humans are not immune to such brain manipulations; they typically exhibit strong emotional feelings. Human emotion researchers should not ignore these systems and simply look at the complex and highly variable culturally molded manifestations of emotions in humans if they wish to determine what kinds of “natural” emotional processes exist within all mammalian brain. Basic emotion science has generated workable epistemological strategies for understanding the primal sources of human emotional feelings by detailed study of emotional circuits in our fellow animals.*

How might we ever understand how emotional feelings are generated in the brain? In a deftly argued, wide-ranging paper in the first issue of *Perspectives on Psychological Science*, Lisa Feldman Barrett (2006) concludes that solid evidence for a diversity of basic emotional processes does not exist. She prefers

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an attributional–dimensional constructivist view of human emotions and postulates that positive and negative core affects are the basic feelings—the primary processes—from which emotional concepts are cognitively and socially constructed. This is largely theoretical conjecture rather than a conclusion derived from robust neuroscientific data.

Her critique of basic emotion theory is premised largely on the inability of modern brain imaging and peripheral psychophysiological data to consistently confirm the existence of many basic emotions. In this article, I consider three key questions: (a) Is Barrett’s argument, based largely on correlative analyses of human electrophysiological and brain imaging data, sufficient to harm basic emotion theory? (b) Do positive and negative valences/arousals exist as birthrights, or is our emotional nature composed of more complex affective qualities? (c) Can one adequately address these questions without analyzing the abundant causal evidence that has been obtained from a cross-species affective neuroscience? Many additional lines of evidence and argumentation need to be considered before we can accept Barrett’s thesis.

My aim is not to delve into the abundant facial and conceptual analysis on which basic emotion theory has been premised in psychology. I expect that many people have experienced pure fear, anger, and sadness at some point in their lives (I certainly have), even though these are fairly rare in modern societies that have strong social “safety nets” against the “slings and arrows” of misfortune that can shift one into such primary-process states of mind. Thus, most of our everyday emotions are such complex mixtures of primary (feeling), secondary (learning and thinking), and tertiary (thoughts about thought) processes that we can barely see the primary process emotions and affects that contribute to the cognitive jungles of our lives. We can use our faces and voices for many purposes, permitting seemingly endless arguments about the validity of those kinds of evidence for basic emotion theories (Ortony & Turner, 1990; Russell, 1995). It is

much harder to argue against the evidence derived from highly localized stimulations of mammalian brains. In this article, I argue that Barrett should consider causal evidence for various basic emotional systems derived from direct brain stimulation experiments in a variety of mammals, including humans (Denton, 2006; Heath, 1996; Panksepp, 1982, 1985, 1986a). Also, there is abundant neurochemical and neuroanatomical data that is consistent with a more diverse affective apparatus within human and animal brains than Barrett has considered (Panksepp, 1986b, 1993, 1998a).

A goal of scientifically comprehensive affect theories is to provide clear psychological and neuroscientific visions of how valenced experiences emerge from brain activities. Barrett would discard basic emotion theory and encourage more subtle psychological and open-ended developmental approaches to the study of human emotions. The latter goal is desirable, but it does not require the former (Panksepp, 2001). Thus, my aim here is quite limited—to question whether Barrett has adequately evaluated the neuroscientific evidence concerning how primary-process emotionality is organized in the brain. Available causal evidence suggests that basic emotion theory easily survives Barrett's critique and that intrinsic affective diversity is comprised of more than just core positive and negative affects. Indeed, social-constructivist and evolutionary agendas may become more robust with more realistic visions of what evolution has provided for our affective lives.

Substantial cross-species data, not discussed by Barrett, supports the existence of a diversity of basic emotional systems, each potentially elaborating distinct feelings. Such work needs to be considered by all who wish to envision what nature has provided for human emotional life. As Barrett has made no distinction between the affective properties of emotional and motivational processes such as hunger and thirst and sensory affects (e.g., pleasure of taste), it is worth introducing such distinctions and inquiring whether Barrett envisions core positive and negative affects to undergird just emotional feelings or also those that arise from homeostatic and sensory processes. Some investigators feel there is no meaningful distinction between emotions and bodily drives (Denton, 2006), and others use taste reactivity as a model for emotional processing (Berridge, 2000, 2003). I do not agree with such conflation, but this is a critically important issue for affect science and is presumably critically important for Barrett's core thesis. If all affective life emerges epigenetically from primordial positive and negative affect, they would presumably also be foundational for homeostatic and sensory affects. But even if she is only interested in the classic emotions, there is abundant evidence that more than two types of emotional affect are natural products of mammalian brain activities.

The basic emotion view advanced here is generally consistent with substantive research programs on basic human emotions (e.g., Ekman, 2003; Izard, Youngstrom, Fine, Mostow, & Trentacosta, 2006; Plutchik, 2003), which were the focus of

Barrett's critique. Her criticism of those theories, valid or not, should not be generalized to the more basic brain-based approach she did not cover (e.g., Panksepp, 1998a), which arises from a very different intellectual tradition. It is simply not fair to lump my views with others of the genera without delving into the diverse and different databases on which my views are based. Before discussing why my affective neuroscience emotion systems theory can survive her challenge, it is important to emphasize that most basic emotion approaches do not neglect the foremost concerns of constructivist approaches: The diverse neural tools that help generate basic emotional states have been envisioned to interpenetrate with the higher neocortical substrates that mediate the cultural and cognitive complexities that characterize the human species (e.g., Frijda, 1986; Izard et al., 2006; Panksepp, 1998a, pp. 320–323). Also, constructivism is strongly linked to a semantically based analysis of how secondary and tertiary emotional–cognitive interactions emerge from social learning, and it is perplexing that investigators interested in the full complexity of human emotions, often elaborated cognitively, do not find approaches whose goal is to deal neuroscientifically with evolved primary-processes to be quite useful for their thinking. The basic cross-species neuroscience and social constructivist approaches could work remarkably well together.

Unfortunately many of the affect systems postulated by modern emotion theorists, from Tomkins (1962, 1963) to the present day, remain to be causally studied in the brains of human beings. However, if we accept evolutionary continuities and homologies, especially in subcortical brain regions where homologies abound, animal models may help unravel the general principles by which the neural and genetic substrates of emotionality operate in all mammalian minds (“mind” in this case being, of course, thoroughly neurobiological).

Contrary to Barrett's arguments, correlative approaches, such as human brain imaging and psychophysiology, are not sufficiently robust to adjudicate what is “basic” about basic emotions (also see Panksepp, 1992). Autonomic physiology is regulated by generalized sympathetic and parasympathetic controls, such that high levels of differentiation among emotions should not be expected with such measures. With regard to modern brain imaging, simply consider the fact that the pseudocolor statistical maps of neural activity changes (rarely more than a few percentage points different than baseline levels) hide vast oceans of neurophysiological and neurochemical activities, with multiple functional systems interpenetrating, especially in subcortical regions, and with overlapping, interacting circuits generating affective mentality. Each imaged pseudocolor region of the human brain hides enormous complexities and individual variabilities that need to be considered. What we can surely say of the typical final products of most averaged results from human brain imaging studies, consisting of isolated islands of arousal, is that “the brain does not work that way.” The relevant causal neuroscientific work done in animals, most of it not pursuable in

human beings, is critically important for a balanced and comprehensive discussion of the state of the field. Neglecting findings from other mammals may be tantamount to ignoring the strongest neuro-evolutionary database that can clarify key issues in the contentious debate initiated by Ortony and Turner (1990), which remained unresolved in a subsequent debate (Panksepp, 1992; Turner & Ortony, 1992).

This commentary will provide a synopsis of important lines of evidence missing in Barrett's analysis. I do not question the utility of dimensional approaches to emotions. That has been empirically well affirmed ever since Wundt introduced this way of operationalizing and systematizing our observations in his 1874 *Fundamental of Physiological Psychology*. Abundant literature supports the utility of dimensional measures in psychological research (including my own; see Panksepp, Knutson, & Burgdorf, 2002), but that should not be used to diminish more detailed, neuroscientific views of how brain systems for emotionality are organized within mammalian brains. Obviously, many theoretical approaches are useful in addressing different aspects of emotionality. Cross-species neuroscience strongly supports the existence of many core emotional systems, apparently with the capacity to generate overlapping spectrums of affective properties as evaluated with human self-reports and choice measures in other animals.

CORRELATIVE VERSUS CAUSAL EVIDENCE IN EMOTION RESEARCH

The dilemma running through Barrett's analysis is the assumption that a psychophysiological correlational approach to emotions will reveal the nature of affective experience. Barrett highlights the chaotic patterns obtained from correlating various physiological, behavioral, and psychological changes during emotional episodes, along with ambiguities that presently exists in the rapidly expanding human brain imaging literature. However, a basic emotion perspective based on direct subcortical brain stimulation data is not harmed by such variability and inconsistencies. Core emotionality is ultimately a reflection of fine-grained but globally influential circuit activities that ethically cannot be vigorously recruited (and thus empirically studied) in traditional human laboratory settings. As psychology becomes fond of imaging the brain substrates of emotionality, many of which reflect complex cognitive correlates, it should continue to attend to lessons from animal brain research.

The cross-species affective neuroscience view provides abundant evidence of how interoceptive systems of the visceral brain and nearby emotional action generating circuits are involved in the generation of many distinct affective feelings (e.g., Denton, 2006; Panksepp, 1998a, 2000a). We will never understand affect if we do not fully consider the causal evidence about such matters derived from animal models (only ~ 2% of Barrett's research references were to animal emotion work and even

less of them referred to work that has specifically focused on the neural nature of affect).

It would be good to know Barrett's perspective on the nature of affect that has been culled from subcortical brain stimulation experiments across many species. She suggests that these systems merely control behavior, but there are many valuation types of experiments in which these systems were directly manipulated, yielding important causal evidence (for abundant references to conditioned approach and avoidance work, see Panksepp, 1998a, 2005a). Does she subscribe to a higher cortical "read-out" theory of affective states, as is common in the field? If so, how does she account for the reciprocal arousals of subcortical/medial-frontal emotional systems and frontal cortical working-memory functions (Goel & Dolan, 2003; Liotti & Panksepp, 2004; Northoff et al., 2004), as well as the abundant evidence for morphine reward sites being situated very deep in the brain, below the neocortex (e.g., Olmsted & Franklin, 1997) where subcortical control of primary-process consciousness is concentrated, much of which is affective (Denton, 2006; Merker, 2007; Panksepp 1998a, 1998b, 2003a, 2003b; Parvizi & Damasio, 2001)?

CONCERNS WITH HUMAN NEUROIMAGING DATA

Barrett looks to human psychophysiological and neuroimaging correlates to argue that the evidence for basic emotional systems is weak. She correctly points out that human brain imaging evidence, taken together, does not clearly support the existence of many classically postulated emotional processes. However, we must be selective in choosing which techniques are appropriate for addressing affective questions. For instance, one should do their best to get people into strong affective states, which are then best imaged with positron-emission tomography (PET) technology (increasingly rare because of its cost). This might permit a selective focus on affective states rather than intimately associated cognitive processes.

Because of the need for precise timing and because of its fast time resolution (< 2 s), functional magnetic resonance imaging (fMRI) technology is intrinsically biased to analyze the perceptual and cognitive aspects of emotional processes, which are bound to be highly variable, rather than the intrinsic dynamics of instinctual-emotional affects, the feeling aspects of emotional states, which surely have longer time constants and are more difficult to induce, in robust forms, within the confines of modern scanners. Further, if raw affect is intimately related to brain generators of instinctual actions, we know that those actions cannot be allowed in the confines of fMRI scanners (e.g., sobbing sadness, dynamic playful hilarity, the thrusting power of anger) even though one can use motor imagery to create weak simulacra (Gordon, Panksepp, Dennis, & McSweeney, 2005; Panksepp & Gordon, 2003). The need to sustain motor stillness and the widespread use of rapidly timed cognitive information, not well suited for evocation of robust affective states, is a

worrisome bias. Perhaps all meta-analyses of affective experience should focus on PET imaging, which can analyze longer time periods (minutes as opposed to seconds) and which permits participants to get into strong emotional states before the imaging starts (i.e., affect is induced before positron-emitting isotopes are infused into subjects).

Most PET studies that have focused on strong affective arousal have found a range of distinct subneocortical effects in brain regions that control animal emotionality (George et al., 1995; Lane, Reiman, Ahern, Schwartz, & Davidson, 1997). Strong feelings of sadness, fear, anger, and joy evoke robust brain arousals in distinct brain regions long implicated in animal emotionality (Damasio et al., 2000). Significant reductions in blood flow were evident in many neocortical regions (for an overall summary of those data, see Panksepp, 2003a, Figure 2, p. 10), indicating that cortical processing is diminished during intense emotional feelings (for a fuller discussion of the commonly observed reciprocity between higher and lower brain functions during cognitively and affectively intense brain states, see Liotti & Panksepp, 2004). A recent meta-analysis that has focused simply on the cingulate gyrus, weighted more toward PET studies (Vogt, 2005, see Fig. 5), has reported distinct representations of anger, sadness, fear, and happiness in this single brain region.

We await fMRI scans in which affective change is correlated to regional brain changes throughout the brain (i.e., independently of whether there were significant absolute “subtractive” changes in specific brain regions). In other words, there may be correlative relationships between brain changes and fluctuating affect in brain regions that show no significant absolute changes during emotional provocations. No such study has yet been done using specific emotion categories! From a subcortical emotional systems perspective, one would predict that medial subcortical and perhaps higher limbic changes will be positively correlated with affect, whereas more lateral cognitive cortical regions, if anything, would be negatively correlated (because those regions of the cortex tends to inhibit raw emotionality, as has long been known from animal decortication work; Siegel, 2005). Most constructivist-attributional views of affect should predict that the higher, more cognitive brain regions should show the most abundant positive correlations. Those kinds of studies will be more important for adjudicating affective issues than will the meta-analysis, emphasized by Barrett, of brain changes conducted by Murphy, Nimmo-Smith, and Lawrence (2003) and by Phan, Wager, Taylor, and Liberzon (2002).

The animal data strongly indicates that a variety of extensive, longitudinally coursing, highly overlapping, emotion-relevant circuits exist in slowly firing subcortical regions of the brain (Panksepp, 1998a; Swanson, 2003) reaching up into limbic cortical regions for self-representation (Northoff, Heinzel, de Greck, Bermpolh, & Panksepp, 2006), where the power of globally released molecules (e.g., neuropeptides) may be more important than the frequency of local action potentials. In

general, those tightly intertwined circuits are not as readily imaged as are those in the more expansive rapidly firing neocortical regions. Time-limited sensory affects are much easier to study in fMRI environments than are strong emotional feelings, helping explain why there is a bit more clarity on issues such as the insular dominance in disgust responses (Murphy et al., 2003) and orbitofrontal participation in various sensory pleasures (Rolls, 2005).

Thus, Barrett’s critical analysis needs to be supplemented by the qualification that there are many reasons why the lower “animalian” circuits for basic emotions are not readily imaged in human fMRI studies, not the least of which is that slowly firing, tightly organized neural systems that overlap with each other extensively would be harder to resolve with fMRI than would the more widespread rapidly firing networks of higher cortical reaches within the human brain (for a more in-depth coverage of the underlying physiological issues, see Panksepp, 1998a; e.g., Fig. 4.4). Most who recognize that association cortex, at birth, is largely a random access memory field are happy with the constructivist vision that most of the detailed aspects of emotional life are learned through the auspices of basic affective systems (Panksepp & Panksepp, 2000).

The cross-species affective neuroscience view is premised on the likelihood that a variety of raw emotional systems (i.e., in-born tools of learning and living) come into the world with few innate “object relations.” For instance the fear and anxiety systems of rats initially seems to only get aroused to the smell of cats, pain, sudden sounds, and other unexpected startling effects. Only through the experience of being threatened does the rat come to fear the image of a cat. Similarly, perhaps we mammals are not born with deeply social brains or mirror neurons; we simply have some rough-and-ready genetically provided subneocortical neural tools, such as separation-distress, social-bonding, and play urges, that epigenetically help create deeply social brains, especially if we have the good fortune of having good emotional upbringing (Sunderland, 2006). The basic emotional systems are envisioned to initially be largely objectless tools for living that rapidly get enmeshed in learning and are potentially idiosyncratically related to individual perceptual-cognitive abilities. This fact provides enormous latitude for constructivist views to explain how the final emotional patterns of individuals and cultures emerge via learning. Constructivism and a reasonably complex basic emotion theory can easily coexist.

BASIC EMOTIONS FROM A CROSS-SPECIES AFFECTIVE NEUROSCIENCE PERSPECTIVE

Barrett highlighted my own approach to the study of basic emotions in two epigraphs used to negatively contrast my position with her own. Unfortunately, anthropocentrism and neglect of the animal data remain prominent in Barrett’s updated advocacy of the Ortony and Turner (1990) critique of basic emotion

theory. I would again note that the critical issues raised at that time (Panksepp, 1992) still remain unaddressed by constructivists. I do not know of any way to reveal the nature of primary-process cross-species brain mechanisms except through animal research, but there is a tendency for psychologists interested in the human mind to neglect those findings. In all fairness, psychologists should not ignore the substance of basic emotion views based on the analysis of brain operating systems in a diversity of mammals. Thus, before discarding basic emotion theories and replacing them with theories based solely on the existence of core positive and negative affect or on little more than social learning, it is worth considering whether constructivist approaches might be strengthened by investing in some of the basic emotion approaches of the past.

At a minimum, constructivists should evaluate causal evidence obtained through the use of localized electrical stimulation of the brain (ESB) as well through a variety of other neuroanatomical, neurochemical, and neuropharmacological approaches. When specific subcortical circuits are stimulated by raw electrical energy (noninformational “garbage” applied to specific subcortical brain regions), affective experience can be easily generated from certain subcortical regions, and the ever-popular amygdala is not a prime site. ESB does not informationally “tell” the brain to do anything specific. ESB does not convey any discrete information. Nevertheless, both ESB and localized chemical stimulation of the brain (CSB) produce coherent emotional/behavioral effects, as well as various synergistic affective effects as monitored through place preference and avoidance paradigms (Panksepp, 1998a, 2005a).

Evidence for distinct emotional arousals in animals has long been supplemented by findings that humans who are stimulated in many medial mesencephalic (especially periaqueductal gray [PAG] sites) and diencephalic brain regions commonly report diverse affective experiences that are congruent with the animal data, suggesting deep homologies in the underlying brain systems (see Heath, 1996, and Panksepp, 1985). Here are just a few more recent examples: Bejjani et al. (1999) provoked acute sadness and depression with subcortical deep-brain stimulation. Panic and fear result from stimulation of other nearby brain sites (Shapira et al., 2006). Mirthful laughter and felt smiling can be evoked from yet other sites (Krack et al., 2001; Okun et al., 2004). All these effects are obtained from deep subcortical systems we share with other mammals.¹ Rapid mood-brightening effects are also obtained from deep limbic cortices where metabolic changes have previously been observed in depression (Mayberg et al., 2004), namely those anterior cingulate areas long implicated in animal emotionality (MacLean, 1990).

¹No clear and rapid affective emotional changes are observed during localized cortical stimulation: I have had most of my neocortex stimulated with rapid transcranial magnetic stimulation with no acute emotional arousals, only some very mild mood effects from frontal cortical stimulation, which is par for the course (for critique of relevant psychological issues, see Schutter, Van Honk, & Panksepp, 2004).

Such brain manipulations can serve as rewards and punishments to change instrumental behaviors as well as conditioned place preferences and aversions in animals (for abundant details, see Panksepp, 1982, 1998a, 2005a). Although Barrett agrees with the existence of such behavioral systems, she does not consider the likelihood that the behavioral choices of animals and their primary-process “instinctual” emotional vocalizations and action tendencies (Knutson, Burgdorf, & Panksepp, 2002; Panksepp, 1981a, 1981b, 1982) also speak loudly for the existence of various affective potentials in animal minds (Panksepp, 2005a, 2005b, 2005c). Perhaps she can logically ignore the many sensory and homeostatic affects (e.g., Berridge, 2000; Denton, 2006), but if she does, they will still need to be explicated either as variants of her core positive and negative affects or as yet other distinct affective processes of the brain. There is a more complex nature to affective experience that needs to be better assimilated into psychological understanding. For instance, a diversity of emotional affects are probably integrally linked to the instinctual actions of mammalian brains (Panksepp, 1986a, 1998a, 2005a).

If we get the cross-mammalian foundational issues right—a clear understanding of the evolutionarily dictated (i.e., primary-process) neuroanatomies and neurochemistries of emotional and affective networks that can be properly illuminated only through animal research—we could more ably contemplate the human secondary and tertiary cognitive complexities on which Barrett focused her keen analysis. By clarifying cross-species primary processes, we may also be able to make some headway on some of the neuro-endophenotypic underpinnings of psychopathologies (Panksepp, 2004, 2006a, 2006b) and potentially develop new medicinal concepts in our desire to help individuals who are affectively compromised (Panksepp & Harro, 2004). Indeed, human neuropharmacology also provides abundant evidence about the neuro-causal determinants of affective states (Depue & Collins, 1999; Panksepp, 1986b; Panksepp & Harro, 2004).

There are many fine tests to investigate the valuative aspects of affective arousal in animals (Peciña, Smith, & Berridge, 2006). The most compelling approaches analyze whether animals choose to activate or deactivate brain stimulations that evoke emotional behaviors and whether such states motivate conditioned place preferences and aversions (Panksepp, 2005a). Barrett did note that conditioned freezing in rats may index fear (e.g., LeDoux, 1996), which is consistent with the unconditioned ESB data (Aggleton, 2000; Panksepp, Sacks, Crepeau, & Abbott, 1991), but behavioral choices remain the gold standard for the existence of affective processes in animals. Animal behaviorists have found that opiate reward mechanisms are concentrated in the brain in very deep mesencephalic regions and that higher opiate-receptor rich regions, including the amygdala, typically do not sustain conditioned place preferences (Olmstead & Franklin, 1997).

Many subcortical brain systems, homologous in all mammals, sustain distinct affective preferences and aversions. For

instance, the chemistries that robustly control fear (e.g., benzodiazepines) do not strongly reduce separation distress (Kalin & Shelton, 1989; Scott, 1974), whereas those that reduce separation distress (e.g., very low doses of opioids) are not as effective in attenuating fear (Kalin, Shelton & Barksdale 1988; Panksepp, Herman, Vilberg, Bishop, & DeEsquinazi, 1980). Likewise, early medications that were effective for generalized anxiety disorders in humans (e.g., chlordiazepoxide and diazepam) are not the same as those that dramatically attenuate panic attacks (imipramine) and vice versa—see Klein (1964), for human data that corresponds well to the animal data described by Scott (1974). Such findings speak for distinct negative emotional affect systems, and there are many other brain chemistries that provoke aversive states in animals (Panksepp & Harro, 2004). Similar evidence is available for brain systems that mediate positive rewards in distinct regions of the brain (Ikemoto & Wise, 2004), including opioids and various other neuropeptides, dopamine, and, more recently, cannabinoids (Zangen, Solinas, Ikemoto, Goldberg, & Wise, 2006). Researchers are finding more and more examples like this, with the search for a role of oxytocin in feelings of love and trust motivated by prior work in animals highlighting psychological elaborations that can only be well studied in humans (Esch & Stefano, 2005; Kirsch et al., 2005; Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005; Panksepp, 1999; Zeki, 2007). One cannot fully discuss the nature of basic affects in humans without fully considering evidence collected from other animals.

WHAT IS AFFECTIVELY BASIC IN OUR EMOTIONAL LIVES?

Barrett wisely worries about the “arbitrary aggregation” of diverse facts under common emotional labels. Indeed, ever since Wittgenstein, it is widely recognized that vernacular concepts are slippery tools for scientific pursuits. However, to determine what is neurobiologically basic and “natural” about mammalian brain emotional systems, one must deal with the abundant ESB and CSB data (Buck, 1999; Panksepp, 1982, 1998a, 2005a). Much empirical evidence supports the existence of at least seven prototype emotional systems in all mammalian brains: SEEKING, RAGE, FEAR, LUST, CARE, PANIC, and PLAY (please note that capitalizations are used to avoid part-whole confusions [Bennett & Hacker, 2003]; to alert readers to the claim that these may be necessary brain systems for those types of emotional behaviors and feelings, although they are by no means sufficient for all the emotional manifestations that may arise from those systems in real world activities; and to highlight that specific psychobehavioral brain systems are the referents of these labels).

Barrett leaves open the existence of such systems in the brain for behavioral control in animal models, but she also needs to consider how these systems impact emotional feelings. Research using ESB and CSB has provided a massive amount of evidence indicating that these systems are basic emotional

valuation mechanisms of the brain (Panksepp, 2005a). Abundant progress has also been made on understanding the subcortical substrates of various sensory affects such as gustatory rewards (Peciña et al., 2006) and basic motivational or homeostatic affects (Denton, 2006, who unfortunately conflated emotional and homeostatic processes).

There are several key questions for evaluating these lines of evidence. Can one evoke such emotion patterns using ESB in homologous brain regions across species? The answer is yes. Do other organisms have various kinds of emotional experiences when thus provoked? To the best of our ability to evaluate experience indirectly with behavioral measures, the answer is yes, but we do need more discriminating research on affective states aroused by such brain processes. Do humans have such systems? They do (Heath, 1996; Panksepp, 1985), though they are much sparser in terms of relative brain size because of higher cerebral expansions. Attributional, dimensional, and constructivist theorists need to attend to such neuropsychological work in order to make adequate judgments about the quality of the evidence for basic emotion systems within the mammalian brain. Barrett’s deconstruction remains incomplete without analyzing such lines of evidence.

If we could aspire toward a foundational cross-species mind science, we might better appreciate how animal brain research helps illuminate the ancestral sources of core emotions, perhaps even the human capacity for grief (Panksepp, 2003b, 2005c) and joyful laughter (Panksepp & Burgdorf, 2003). Brain mechanisms for animal separation distress and human sadness appear to be related (Panksepp, 2003b; Zubieta et al., 2003). Tickle-induced 50-kHz ultrasonic vocalization circuits (generating rat “laughter”) may be homologous to those that facilitate human social joy (Burgdorf & Panksepp, 2006; Burgdorf, Wood, Kroes, Moskal, & Panksepp, 2007; Panksepp, 2000b, 2007), and such social emotional circuits are important motivational substrates for drug addictions (Panksepp, 1981a, Panksepp, Nocjar, Burgdorf, Panksepp, & Huber, 2004; Panksepp et al., 2002).

Are such emotional circuits “natural kinds” (i.e., coherent functions of the brain that are largely inherited)? It depends on how one uses such concepts (for a relevant philosophical analysis, see Zachar & Bartlett, 2001), but it seems evident that the basic connectivities and neurochemistries of the subcortical brain regions that seem essential for affective experience are genetically prescribed and experientially refined. As every neuroscience student knows, if you learn these aspects of rodent brains, one has a working knowledge of the subcortical terrain in humans and all other mammalian brains. Of course, it is axiomatic that all brains systems will exhibit abundant cross-species variability and epigenetic maturation. The fact that emotional feelings are probably reworked at the level of many cortico-cognitive regions, allowing abundant subtlety and reflective awareness of emotions in humans, is not an argument against the existence of basic emotional systems that contribute heavily to what humans feel and do.

BASIC EMOTIONAL INSTINCTS AND EMOTIONAL FEELINGS

Does the vast spectrum of human emotional feelings arise just from some kind of primordial positive and negative affect? Are emotional feelings based solely on primordial “reward” and “punishments” systems that promote approach and avoidance? Because of advances in neuroscience, we now should consider whether such simplified theoretical approaches were historically mandated largely by our inability to analyze the affective functions of the brain. Modern neuroscientific evidence suggests there are many types of rewards and punishments in the brain (e.g., many Thorndikian “satisfactions” and “discomforts” in human and animal minds; see the “Law of Effect” in Thorndike, 1911, p. 244; for updated discussion, see Panksepp, 2005b). Researchers subscribing to theories that assume that the neurobiological contribution is simply some kind of pervasive feeling of goodness and badness need to consider evidence that all mammals experience several distinct basic rewards, such as euphoric dopamine-based appetitive excitement (wanting/SEEKING) facilitated by psychostimulants (Berridge & Robinson, 1998; Ikemoto & Panksepp, 1999; Panksepp, 1981b, 1986a), the opioid-based reward that contributes to many sensory pleasures (Panksepp, 1998a), and the vigor of PLAY that can be dramatically reduced by psychostimulants (Beatty, Dodge, Dodge, White, & Panksepp, 1982). Also, how would Barrett explain other subcortical pleasures, such as feelings of orgasm (Holstege et al., 2003) and taste gratifications (Peciña et al., 2006), that have highly localized controls in the brain. Such affects may not be irrelevant for her thesis simply because some researchers do not consider them to be emotions, which obviously is a conceptual category, like motivation, rather than a natural kind.

In any event, there are abundant data that many positive emotional feelings, such as playful euphoria and caring nurturance, and negative ones, such as anger, fear, separation-distress, do arise, in part, from distinct brain systems, as do the diverse sensory and homeostatic affects. Abundant animal and human data affirms the existence of distinct anatomies and chemistries for such brain functions (Denton, 2006; Panksepp, 1998a, 2005a; Peciña et al., 2006). Indeed, who would ever confuse these feelings in human phenomenal experience? And is that not the most critical point for all human-based basic emotion theories from Tomkins onward?

It is unwise to prematurely close such issues on the basis of the weakest forms of available evidence (i.e., psychophysiological and human brain fMRI). Basic emotional responses (typically accompanied by affect) can be evoked readily and reliably across species and with highly localized ESB and CSB. For instance, RAGE responses are evoked essentially from the same brain regions in rats (Panksepp, 1971), cats, and primates (Siegel, 2005). Such localized ESB can activate many different types of instinctual tendencies, including SEEKING, RAGE,

FEAR, LUST, CARE, PANIC and PLAY, which are recognized as the major emotional operating systems in a cross-species affective neuroscience (Panksepp, 1998a, 2005a). Detailed monograph-length empirical summaries of certain systems are available, though these investigators do not use my terminological conventions, nor do they necessarily acknowledge that animals have emotional experiences (for self-stimulated SEEKING reward, see Olds, 1977; Rolls, 1975; for FEAR, see LeDoux, 1996; for LUST, see Pfaff, 1999; for RAGE, see Siegel, 2005; for PLAY, see Burghardt, 2005; and for maternal CARE, see Numan & Insel, 2003).

Fine brain mapping reveals that each of these emotional systems has, to a degree, distinct subcortical substrates. However, all of the systems exhibit abundant overlap and interactions (especially with very broadly acting, nonspecific arousal systems such as norepinephrine and serotonin) that may help reduce fMRI distinguished visibility of basic emotional processes and may also lead to many shared arousal components among different affects.

Animals can indicate, in many ways, that they do have affective experiences when emotionally aroused with ESB and CSB of subcortical brain regions that appear to be homologous across all mammals (Panksepp, 1998a, 2005a). Emotional vocalizations are excellent measures, as are conditioned place preferences and avoidances (Burgdorf, Knutson, Panksepp, & Shippenberg, 2001). Also, the correspondences between human and animal findings are striking. As previously noted, it is remarkable that the general dispersion of brain sites that can evoke separation distress calls in animals resemble the subcortical arousals documented by Damasio et al. (2000) during a PET analysis of human sadness (Panksepp, 2003b). Both the animal and human systems are modulated by opioids (Panksepp, 1981a; Panksepp et al., 1980; Zubieta et al., 2003). Admittedly, we need more research and better empirical tools to distinguish distinct emotional affects in animals, but, so far, evidence strongly indicates that the aroused emotional affects are an integral aspect of the subcortical circuitries that promote the instinctual emotional actions (Panksepp, 1998a, 2000a, 2005a, 2005b).

A reasonable alternative to the core affect view advocated by Barrett is that each basic emotional action system has the potential to contribute to psychologically distinguishable affective states that help code major categories of survival needs, thus helping animals better anticipate life-challenging situations. Raw affects, in this view, are genetically promoted anticipatory states that become rapidly connected to associated environmental events, thereby further facilitating survival through individual learning. This anticipatory, experience-expectant view of affects may help us understand why there would be so many distinct affects. This issue is best envisioned by focusing on homeostatic affects discussed by Denton (2006). Take the easily understood example of hunger. This feeling does not indicate energy reserves are dangerously low but rather that it may

be wise to top up energy reserves when opportunities arise. Thirst does the same for water needs. How could affects anticipate needs if they only came in primal positive and negative “flavors”?

Among such homeostatic affects, one would be hard put to argue against the likelihood that primitive brain mechanisms, with a great deal of genetic determination, regulate our capacity to feel hunger (Burdakov & Alexopoulos, 2005; Panksepp, 1974), thirst (Sewards & Sewards, 2000), salt appetite (Denton, 2006), fatigue, and sleep (Shen, Barbera, & Shapiro, 2006), as well as thermal and other imbalances of our body (Zeisberger, 1998). There also appear to be a vast number of aversive affects that are linked to our capacity to detect stimuli in the outside world, from disgust and pain (Craig, 2003; Panksepp, 2005c) to the many pleasures and delights of sensation (Berridge, 2000; Pecina et al., 2006). This is not even considering the diverse emotional affects with their abundant and distinct neuropeptide controls (Panksepp, 1993; Panksepp & Harro, 2004).

Animals with many affects are bound to be more capable of survival and thus more competitive than are those that can only feel good and bad in more generalized ways. Indeed, fluctuating affects may be the primary source of that pervasive, but phlogiston-like, concept called *reinforcement* (Panksepp, 2005b), but the jury remains out on that issue. In contrast, where is the evidence for the alternative view that the diversity of affective life is constructed simply from primordial positive and negative core affects (Barrett, 2006; Russell, 2003)? We await a credible neurobiological scenario and some preliminary evidence for that view. Considering the animal evidence for necessary subcortical loci of control, why would we assume that affective experience is constructed within the higher attributional regions of the brain?

WHERE ARE THE BRAIN MECHANISMS OF AFFECT CONCENTRATED?

Much of human brain imaging of affects has been premised on the likelihood that experience requires a neocortical “read-out” of some kind. My reading of the evidence is that raw emotional and homeostatic affects are largely elaborated subcortically. Those are the only regions of mammalian (Panksepp, 1998a, 2005a) and human brains (Heath, 1996; Panksepp, 1985) where causal manipulations (electrical and chemical stimulations) can promptly and dramatically modify emotional behaviors and emotional feelings conjointly (see Footnote 1). Indeed, Damasio et al.’s (2000) exquisite PET imaging of human affects and practically all other high-affect PET studies (for a summary, see Liotti & Panksepp, 2004) have highlighted arousal of subcortical areas long implicated in animals studies, with abundant concurrent neocortical inhibitions.

The subneocortical locus of control for affective processing, in the preponderance of PET studies that have focused on emotional feelings, reinforces the importance of animal models in working out the details of the basic emotional systems of the mammalian brain that Barrett did not discuss. In this context, we

must recall that when animals have been surgically deprived of the neocortex soon after birth, they still exhibit practically all instinctual–emotional behavior patterns (Kolb & Tees, 2000; Panksepp, 2005a). It is truly remarkable that juvenile rats play almost normally even if their neocortical mantle was surgically eliminated soon after birth (Panksepp, Normansell, Cox, & Sivi, 1994) and that humans can express joy, with many indices of apparent feeling, even if their higher brain is absent (Shewmon, Holmse, & Byrne, 1999).

If one considers all the evidence, it should be clear that the diversity of experienced emotional qualities are not simply generated by the higher attributional regions of the brain as Barrett envisions, even though those regions surely add something (instigation, inhibition, cognitive parsing, and regulation?) to the affective equation. When one extensively damages the medial strata of the brainstem from the PAG all the way up to the anterior cingulate, consciousness and emotional reactivity are dramatically changed toward emotional unresponsivity and akinetic mutism (Panksepp, 1998b; Watt & Pincus, 2004). In this literature, there is little to commend the view that the ability of the brain to generate affective experience is mediated by, as opposed to largely triggered by, cognitive appraisals. This view leaves open the possibility that higher attributional regions of the brain do contribute positively to the affective quality of learned emotional experiences (secondary and tertiary processes), but the causal data stream runs thin on that conjecture, especially when one considers the abundant neocortical inhibitions during strong emotional arousals (Damasio, et al., 2000; Liotti & Panksepp, 2004; Panksepp, 2003a).

Admittedly, more work is needed to clarify how well animals can discriminate arousal of the various emotional action systems, using procedures along the lines pioneered by Stutz, Rossi, Hastings, and Brunner (1974), which indicated that self-stimulation reward from the septal area could be discriminated from reward evoked along the medial forebrain bundle. In the absence of such difficult functional studies, which are not well supported in the ruthlessly reductionistic research-funding climate of modern behavioral neuroscience in which affective states are rarely considered, we can still note the remarkable state-dependent learning effects that can be achieved by manipulating the neurochemical infrastructure of the emotional brain (Jarbe, 1986; Overton, 1991). These studies indicate that animals can discriminate an enormous number of pharmacologically induced internal states that are known to generate different feelings in human beings.

THE NEUROCHEMISTRIES OF BASIC AFFECTS

The above neural diversities do not mean that many brain emotional action systems do not share some common arousal functions. Clearly, brain norepinephrine and serotonin systems influence every emotion (see Panksepp, 1982, Figure 2). Barrett

also highlights that dopamine can modulate affect, but she falsely suggests that our own work has supported a restricted “reward” viewpoint. In fact, we were among the first to argue that dopamine was a generalized facilitator of broad scale appetitive SEEKING urges, which are accompanied by a psychological quality more akin to foraging-related exhilaration that helps facilitate eager anticipation of rewards and escape from punishments (Ikemoto & Panksepp, 1999; Panksepp, 1992). Dopamine helps forge expectations about all kinds of worldly resources rather than the pleasure of sensation (Panksepp, 1981b, 1986a). This view has more recently been advanced in the more restricted “wanting” and “liking” concepts of Berridge and Robinson (1998).

Although there is some worth in postulating that brain dopamine is a generalized positive affect system whereas acetylcholine is a generalized negative one (for relevant data see Brudzynski, 2007), one would still have to consider the affective properties and enormous number of neuropeptides (Panksepp, 1993) that can control conditioned place preferences and aversions, leading to rather precise affective predictions in humans. As summarized in Panksepp and Harro (2004), corticotrophin releasing factor antagonists can reduce negative feelings associated with a host of stressors. It is predicted that antagonists for Substance P, a neuropeptide that mediates instinctual rage and perhaps anger (Siegel, 2005), may selectively reduce angry irritability in human beings. Vasopressin receptor antagonists are predicted to reduce the strength of male sexual pushiness and jealousy. Oxytocin should elevate feelings of nurturant care and confidence while reducing sadness. How might Barrett incorporate these diverse findings, and many others (Panksepp & Harro, 2004), in the context of dimensional theories she favors?

THREE CRITICAL CHALLENGES OF BARRETT’S VIEW

At present, there is no neuroscientific evidence that just a simplified positive–negative affective infrastructure controls emotional feelings in either animals or humans. There are currently many lines of evidence for multiple emotional affects, as well as other affect varieties (sensory and homeostatic). How would Barrett deal with the following three well-established affective phenomena from her point of view?

1. Although most addictive drugs ultimately use the dopamine-based SEEKING system as a final common arbiter of drug desire, the affective effects of various addictive drugs are distinct enough that both human and animal addicts have little trouble distinguishing the affective effects of opioids from cocaine and of nicotine from alcohol/barbiturates and benzodiazepines (Bevins, 2004), not to mention more arcane rewarding drugs (Ikemoto & Wise, 2004). Most of these drugs have been successfully used as cues to control state-dependent discriminations in learning tasks (Jarbe, 1986;

Overton, 1991). These patterns of results are not obviously consistent with a bidimensional core positive and negative affect point of view.

2. Our analysis of the basic emotional responses of young animals to social separation demonstrated that opioids, oxytocin, and prolactin are uniquely efficacious in reducing separation distress, which was long postulated to be a key substrate for human sadness (Panksepp, 1981a, 1998a). The psychostimulants and other drugs of abuse that can be rewards for both animals and humans were not highly effective in this way, nor were practically any of the other agents available in biological psychiatry (Panksepp, 1991; Panksepp, Normansell, Herman, Bishop, & Crepeau, 1988). These social effects are obtained at doses that do not affect many other emotional and motivated behaviors. Our early prediction that human sadness would be characterized by low-opioid activity in the brain was eventually affirmed in humans (Zubieta et al., 2003). Because chronic sadness is a main source of human affective disorders, we can predict that new nonaddictive opioids such as buprenorphine should be highly effective antidepressants. We further predict that all neurochemical agents that specifically reduce separation distress will tend to increase social strength and thus dominance, a prediction verified for opioids (Panksepp, Jalowiec, DeEsquinazi, & Bishop, 1985). Thus, cross-species basic emotion research makes rather precise predictions for humans that can be falsified. Can a core positive and negative affect view generate any comparably precise psychobiological predictions?
3. There are abundant specificities in psychiatrically effective drugs. Some agents greatly reduce anxiety, others reduce depressive and sad feelings, and yet others attenuate the excessive joy and optimism of mania (Panksepp, 2004). If one believes in a single negative affect systems, one also need to explain why various neuropeptides, including brain corticotrophin releasing hormone, dynorphins, adrenocorticotrophic hormone, cholecystokinin, and muscarinic cholinergics can all mediate negative affect (Panksepp & Harro, 2004). There has long been solid clinical evidence for distinct anxiety and panic systems (Klein, 1964), and such findings are fostering work to distinguish multiple fear–anxiety systems in the animal brain (Vianna & Brandão, 2003).

DECONSTRUCTING CORE POSITIVE AND NEGATIVE AFFECT

Although animal brain research as well as human phenomenal experience indicate that affective life has much more resolution than simply some kind of primordial positive and negative affects, it is often useful to communicate one’s states in terms of global categories of good and bad. However, our categorization of affective life into positive and negative groupings may reflect

conceptual categories rather than natural kinds. Clearly, the brain–mind is sufficiently complex to harbor a large number of basic affective urges and feelings. But even without the neuroscience, some key questions would need to be addressed by dimensional theorists: Why do the various distinct basic emotions so easily yield statistically independent personality factor loadings at the human psychological level? And why do the negative emotions (FEAR, RAGE, and PANIC) also load on a negative affect superfactor, whereas the positive emotions (SEEKING, CARE, and PLAY) load on a positive affect superfactor (Davis, Panksepp, & Normansell, 2003)? Might it not be that positive and negative affects, rather than being foundational for the construction of emotional life, are actually umbrella concepts generated by our remarkable linguistic–cognitive abilities and our communicative need to simplify our affective lives to ourselves and others?

An advantage of dimensional approaches to the emotional life is that they have provided clearer visions than has basic emotion theory of how higher order cognitive emotional concepts might emerge through learning. However, basic emotion theory also provides a vision of the emergence of certain emotional complexities from more primitive brain systems through evolutionary refinements. Indeed, we were the first in neuroscience to consider how emotional variety could have evolutionarily emerged from more simple affective structures: Our initial experiments in social neuroscience postulated that the separation distress mechanisms of the brain emerged evolutionarily from more basic pain systems (e.g., Panksepp, 1981a, 2005c; Panksepp et al., 1980). This possibility guided our neurochemical analysis of this system in productive ways across many species, highlighting how robustly opioids could inhibit paniclike separation-distress responses in guinea pigs, dogs, and chickens (Herman, Conner, Bishop, & Scott, 1978; Herman & Panksepp, 1978; Panksepp, Vilberg, Bean, Coy, & Kastin, 1978). On the basis of this animal data, we had postulated that human sadness would be a low-opioid state, which is a prediction that has now been affirmed, as previously noted, by studies evaluating human brain mu-opioid receptor occupancy (Zubieta et al., 2003). A complementary premise is that neurochemistries that could powerfully and specifically alleviate the pain of separation distress (namely opioids, oxytocin, and prolactin) could serve as affective substrates for the construction of social bonds and more subtle prosocial feelings (Nelson & Panksepp, 1998; Panksepp, 1981a, 1998a). These ideas should delight constructivists who wish to consider the applicability of a rich diversity of basic emotional tools for their own research concerns.

TOWARD A SYNTHESIS OF NATIVIST AND CONSTRUCTIVIST VIEWS

There has been a perennial philosophical conflict between nativist and constructivist view of mind—between those who are interested in what nature provides for the overall mind equation

(internalism) and those interested in what nurture provides (externalism). It is increasingly realized that views seeking to have one side prevail over the other are not facing up to reality. Only through interaction of such processes can minds be created.

It should be self-evident that psychology does need some level of naturalism, some kind of data-based neuro-genetic foundation that is consistent with the diversity of psychological experiences we have in the world. We humans are mammals who share homologous brain architectures below our neocortical crowns. These foundational processes are functionally similar across mammals, and they are essential for our emotional lives. We are also wise to envision how much of our mental life is constructed. However, modern psychology often marginalizes those aspects of the mind–brain to which human psychologists have little direct access, even though abundant neurological evidence indicates that they exist. Might it not be wiser to envision them as raw tools for the construction of higher mental functions?

The organization of those ancient brain systems is governed decisively by genetic influences operating in supportive environments. Conversely, these rough-and-ready genetically prescribed tools interact abundantly with more flexible cortical systems that vary considerably more between species and among members of the same species, yielding the unique epigenetic landscapes of individual lives. Still, without those ancient genetically dictated simple-minded systems, the complexities of mental life might not exist. Certainly damage to subcortical brain regions, ounce for ounce, is more disastrous to emotional life than is neocortical damage. Emotion researchers need to envision those subcortical emotional systems in more sophisticated ways (Merker, 2007) than as simply negative versus positive affects or light-switch type arousal systems. But even that primitive variant of nativism suggests we cannot escape some kind of basic affect theory in order to understand what kind of creatures we are. Fortunately, because of our vast neocortical abilities, those ancient systems do not strictly limit what kind of an individual each human being can become.

Unfortunately, because of the sustained disagreements between nativists and environmentalists in philosophy, between nature and nurture in behavioral psychology (Panksepp, 1990) and between the European ethological tradition that studied the genetically predisposed “instincts” and the Anglo-American animal behavioral tradition that focused on learning (for fine historical overview, see Burkhardt, 2005), the middle ground is only slowly becoming ascendant. A viable nativist agenda involves attempting to clarify those primary-process tools provided by nature without denying the impact of nurture. A viable constructivist agenda involves clarifying how secondary and tertiary processes control emotional learning, without denying that nature provides critically important tools for living, and learning about the worldly complexities in which we are immersed.

Barrett's thesis seems partly premised on a lingering anti-nativism in modern psychology. We need to cultivate a fruitful and realistic middle ground. In a critique of the nativist agenda (especially of the cognitive–linguistic varieties advanced by Chomsky, Fodor, and Pinker), philosopher Fiona Cowie, coming from a vigorous environmentalist perspective, made a remarkable semiconciliatory remark about nativism. She says, “it may be a mistake to regard the nativist’s metaphors as anything more than colorful *façons de parler*. They do, after all, often seem to be intended more rhetorically than explanatorily. . . . so, it is a mistake to rely too much upon them. Further, to insist on a literal reading of the nativist’s words is in many cases to violate a central imperative of interpretation—namely, that one shouldn’t be too quick to ascribe stupid views to smart people. Particularly problematic in this regard is the attribution of a commitment to ‘naïve’ or non-dispositional nativism to those who favor the ‘writing on the soul’ and ‘*prêt à porter*’ conceits. This view, according to which concepts or beliefs are, as it were, ‘fully present’ in the mind at birth, has for so long been known to be susceptible to so many and such obvious objections, that charity alone might prevent our attributing it to anyone, his or her taste in similes notwithstanding.” (Cowie, 1999, p. 6). Although there may be no innate ideas in the human brain, there certainly are a variety of innate potentials to feel and perhaps to think in various ways. What modern affective neuroscience emotion theory may provide is a basic dispositional explanatory infrastructure for the massively plastic and affect-rich human mind.

The status of basic emotions cannot be adequately resolved solely by psychological or behavioral research. One can understand Barrett’s wish to deconstruct basic emotion theory once again, in light of how modern correlative psychophysiological and brain imaging data are so frightfully confusing. However, such issues must also be considered from the perspective of robust causal neuroscientific approaches to such foundational mind issues. Surely, human brain imaging is not yet as robust an approach to causal issues as is localized stimulation of relevant areas of the brain. Cross-species brain research remains the most workable strategy providing critically important lines of detailed evidence, and theoreticians need to fully consider the available cross-species affective neuroscience data to evaluate if natural emotional dispositions do exist in mammalian brains.

If we focus on the animal data, in which causal manipulative studies of the brain have long been conducted, we can be confident that extensive networks of partly overlapping neural circuits help mediate diverse forms of emotional behaviors and positive and negative affects. Affect appears to be a property of coherently organized neural circuits extending, most critically, from central mesencephalic levels, such as the PAG, through medial regions of the diencephalon and extending to the orbitofrontal cortex and to both medial (anterior cingulate and medial frontal cortices) and more lateral regions of the forebrain, especially the temporal lobes and insula (Northoff et al., 2006; Panksepp, 1998b; Rolls, 2005). For instance, in the PAG,

negative emotions, such as separation distress, fear, and defensive behaviors, are generated in more dorsal areas; positive affect is generated in more ventral regions; and many other emotional “columns” are interspersed in between (Bandler & Keay, 1996; Watt, 2000), which may provide a primordial “self” representation that is essential for the neurodynamics of emotional feelings (Panksepp, 1998b). In dorsal PAG regions, we find fear responses as well as separation distress responses. Along the hypothalamic corridor that leads to the PAG, we find negative fearful effects concentrated in medial and anterior hypothalamic regions, whereas separation distress is more evident in medial regions of the thalamus, extending to bed nuclei of stria terminalis and nearby septal regions (Herman & Panksepp, 1981; Panksepp et al., 1988). Several positive affect systems within the hypothalamus are concentrated more laterally, running from the ventral tegmental area through much of the medial forebrain bundle that projects to the nucleus accumbens and medial septal regions. Several negative affect systems are concentrated the bed nuclei of the stria terminalis and in central and lateral regions of the amygdala, whereas the corticomедial areas are devoted more to socio-sexual issues. Many other kinds of negative sensory affects are elaborated in the insula, from disgust to pain (Craig, 2003). A recent remarkable neuropsychological discovery has been the elimination of the urge to smoke after insular damage, with no comparable reduction of the desire to eat (Naqvi, Rudrauf, Damasio, & Bechara, 2007). Likewise, the cingulate gyrus seems to participate in many affects, but with apparently remarkable regional loci of control for various emotions (Vogt, 2005). All these systems interact with yet higher cognitive structures (Borod, 2000).

With such complexities, what should we do with the ever-popular global positive and negative affect states? Are they natural kinds or conceptual categories? Barrett comes close to suggesting it is the former, but there is little relevant neuroscience data available, so the jury must remain out. Might those concepts be easier to understand if we recognize that certain basic emotional traits (e.g., SEEKING, CARE, and PLAY) cluster together in a positive emotional category and that others (e.g., FEAR, RAGE, and PANIC or separation distress) cluster in a negative affect category (Davis et al., 2003) just as they do in cross-cultural studies (Kuppens, Ceulemans, Timmerman, Diener, & Kim-Prieto, 2006)? Considering the cross-talk among emotional systems as well as their influences within higher cognitive structures, it is to be expected that specific negative emotions facilitate a cognitive sense of dissatisfaction and feelings of ill-being, whereas positive emotions promote general feelings of satisfaction and well-being (Diener, Lucas, & Scollon, 2006). How such cross-talk among affective and cognitive systems occurs is simply not known in any detail, but it is not hard to envision that higher working-memory brain regions such as the dorsolateral prefrontal cortices, which harvest emotional information from subcortical circuits, may not only

parse affective space with a host of cognitively elaborated emotions (from empathy to regret to shame, etc.) but also actively transform many affective messages into larger conceptual categories (positive and negative affect), perhaps for ease of communication.

On the other hand, if there does exist some kind of simplified positive and negative affective infrastructures at the evolutionary core of the human brain–mind, we must await solid neuro-empirical evidence for that scenario. For instance, both psychostimulants and opioids are addictive and generate distinct positive affects, and one would have to specify how both arise from an even more primordial form of positive affect. Of course, it is possible that both basic emotions and generalized affects do exist in the brain, but conceptual position taking will not suffice to resolve the issue. For now, we can be more confident about the existence of distinct brain emotional systems and a diversity of primary-process affects than about more generalized, bidimensional affective circuits.

The question of how to clearly discuss the nature of our pre-propositional psychological processes (i.e., raw affects) will remain a major challenge for scientific psychology for some time to come. I think we can all agree that neuroscience is more effective in illuminating the pieces of the emotion puzzle than the psychological wholes that are constructed from individual experiences in the world. As soon as we recognize that different approaches can work together to solve problems of mutual interest, we may no longer wish to have one approach prevail over the other. I am happy with the conclusion that most of the human mind is constructed from some rudimentary tools—for instance, the epigenetic molding of our social brains is probably strongly dependent on our capacity to feel primary-process separation distress, joyful play, lust, and caring nurturance (Panksepp, 2001).

SUMMATION

Many of Barrett's suggestions for a new research paradigm for understanding emotions are excellent, and a great deal of progress has already been made in that direction in both basic emotion theory as well as in constructivist approaches to understanding the human mind. There are some primordial "natural kinds" in this diverse psychological world of ours—often called primary processes by psychiatrists—but they are rather hard to objectify in human psychology because of the layers of experiential change (secondary and tertiary processes) that mold both our physical and psychological worlds and the lack of routine causal manipulations and adequate imaging methodologies to envision them unambiguously within human brains. Because "natural kinds" has become such a loaded term in psychology and philosophy, and because we cannot see clearly the brain mechanisms behind the surface of experience, perhaps it is better to speak about "practical kinds" (see Zachar & Bartlett, 2001) in our search for the dynamic epigenetic controls

that evolution has provided within the many genetically ingrained mechanisms of brain. Because of remarkable advances in molecular biology, in which it has been shown that experience-dependent gene methylation and acetylation can transform the social landscape across generations (e.g., Meaney & Szyf, 2005), we may all now agree that epigenetic-developmental processes help mold the genetic potentials for raw affectivity within our brains and minds into a diversity of seemingly seamless psychological wholes.

The remarkable aspect about this debate is that it is easy to integrate basic emotion theory fruitfully with all other forms of emotion theorizing that have been proposed, even psychoanalytic perspective (Panksepp, 1999). Had I selected an epigraph for this chapter, it would have been the following: "The concept that raw affects are initially objectless in the brain, allows a fruitful rapprochement between basic emotion theory and constructivist views of emotions. . . . Constructivist theories of emotions obviously need some basic tools for anything useful to be constructed. The intrinsic, evolutionarily provided emotional abilities revealed by affective neuroscience are such tools. Even though such emotionally valenced systems cluster into constellations of positive and negative affects, it seems unlikely that only two primal types of affective feelings are the raw materials from which all other affects are created within mammalian brains" (Panksepp, 2006a, p. 22).

Hopefully, all sides in this debate agree that, despite existing conceptual differences and scientific priorities, it is very important to understand the nature of anger, desire, fear, love, lust, playful joy, and "cryful" sadness in human lives, as well as their diverse cultural manifestations. And we should remember that if, in fact, there were no primary-process emotions that we share with other animals, we would still need to credibly answer why it is so easy, even for children, to recognize such a variety of distinct emotional states not only in each other but in other mammals as well (Darwin, 1872/1998). As Barrett wonders "How is it that people can automatically and effortlessly see *anger*, *sadness*, and *fear* in others?" Moreover, why do blind children show the species-characteristic facial expressions for so many emotions? Why do anencephalic children exhibit such clear emotionality (as long as they have been well cared for) even though they are missing essentially all higher regions of their brains (Shewmon et al., 1999)?

It does appear that James–Lange theory was misconceived in viewing emotions primarily as higher brain read-outs of bodily commotions. Indeed, had William James known of the visceral nervous system, with its very complex mapping of instinctual processes and autonomic parameters, he might never have proposed the view that has now captivated psychology for more than a century. Had he known of the limbic system, he might have instead suggested that higher emotional feelings were constructed by primitive emotional feelings, created within ancient reaches of the mammalian brain, influencing the higher cognitive apparatus. To the best of our knowledge, raw emotional

feelings are intimately linked to the instinctual action systems of the brain that are intermeshed with complex subneocortical visceral maps of the body (Panksepp, 1998b, 2000a)—maps that activate autonomic responses congruent with emotional actions, which also listen to feedback echoes from the body. Those feedback echoes are needed to regulate primary-process emotional circuits. They do not create emotions by sending bodily information directly to higher regions of the brain. Those higher regions can be eliminated, and all basic emotions remain relatively intact (Panksepp et al., 1994). In fact, James entertained many ideas about emotions, including the possibility that emotional experience was closely linked to the emotional–instinctual apparatus. James said, “In speaking of instincts, it has been impossible to keep them separate from the emotional excitements which go with them” and that “every object which excites an instinct excites an emotion” (See Denton, 2006, p. 7; see also abundant information for the localization of basic homeostatic affects in subcortical regions of the human brain).

Perhaps it is now wise to pay heed to William James’s alternative approach rather than just his more famous conjecture. Emotional affects are critically linked to the activities of “instinctual” emotional–behavioral circuitries of the brain. It is time to recognize that the most beautiful and robust constructions are made with abundant raw materials and with many rough-and-ready brain–mind tools. The search for such tools is not the same as the quest for biological determinism nor need it be envisioned as a simple-minded aspiration for ruthless reductionism. It is far better for us to be gentle reductionists who recognize the epigenetic complexities of the mind that Barrett wishes to move to the forefront of the emotion research agenda (Panksepp, 2001). Plurality is good in this area, but our understanding of the neural infrastructure of basic emotions and their affects remains woefully incomplete, as there are very few researchers in America working on such topics in the present intellectual climate.² We will make more progress if each discipline appreciates how its own area of expertise relates respectfully to those of others and to the whole. Mechanistic science is only adept at explicating “parts” rather than describing the “wholes.”

But perhaps this general principle has already been resolved in many minds. As Kuppens et al. (2006, p. 491) note, it is now widely accepted “that emotions are both biologically grounded and culturally shaped. Not surprisingly, then, the debate has now been settled more or less, with most authors acknowledging that there are both universal and culture-specific aspects to emotion.” I trust that we will all finally agree on that point.

²I offered a graduate seminar on *Affective Neuroscience* at Boston College during the Fall of 2005. Professors Lisa Barrett and Jim Russell were consistent and cordial participants. Many of the above issues were discussed in detail.

REFERENCES

- Aggleton, J.P. (2000). *The amygdala*. New York: Oxford University Press.
- Bandler, R., & Keay, K.A. (1996). Columnar organization in the midbrain periaqueductal gray and the integration of emotional expression. *Progress in Brain Research*, *107*, 285–300.
- Barrett, L.F. (2006). Are emotions natural kinds? *Perspectives on Psychological Science*, *1*, 28–58.
- Beatty, W.W., Dodge, A.M., Dodge, L.J., White, K., & Panksepp, J. (1982). Psychomotor stimulants, social deprivation, and play in juvenile rats. *Pharmacology, Biochemistry, & Behavior*, *16*, 417–422.
- Bejjani, B.P., Damier, P., Arnulf, I., Thivard, L., Bonnet, A.M., Dormont, D., et al. (1999). Transient acute depression induced by high-frequency deep-brain stimulation. *New England Journal of Medicine*, *340*, 1476–1480.
- Bennett, M.R., & Hacker, P.M.S. (2003). *Philosophical foundations of neuroscience*. Malden, MA: Blackwell.
- Berridge, K.C. (2000). Measuring hedonic impact in animals and infants: Microstructure of affective taste reactivity patterns. *Neuroscience and Biobehavioral Reviews*, *24*, 173–198.
- Berridge, K.C. (2003). Comparing the emotional brain of humans and other animals. In R.J. Davidson, K.R. Scherer, & H.H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 25–51). New York: Oxford University Press.
- Berridge, K.C., & Robinson, T.E. (1998). The role of dopamine in reward: Hedonics, learning, or incentive salience? *Brain Research Reviews*, *28*, 308–367.
- Bevins, R.A. (Ed.). (2004). *Nebraska symposium on motivation: Vol. 50. Motivational factors in the etiology of drug abuse*. Lincoln: University of Nebraska Press.
- Buck, R. (1999). The biological affects: A typology. *Psychological Review*, *106*, 301–336.
- Borod, J.C. (Ed.). (2000). *The neuropsychology of emotion*. New York: Oxford University Press.
- Budzynski, S. (2007). Ultrasonic calls of rats as indicator variables of negative or positive states: Acetylcholine-dopamine interaction and acoustic coding. *Behavioural Brain Research*, *182*, 261–273.
- Burdakov, D., & Alexopoulos, H. (2005). Metabolic state signalling through central hypocretin/orexin neurons. *Journal of Cellular and Molecular Medicine*, *9*, 795–803.
- Burgdorf, J., Knutson, B., Panksepp, J., & Shippenberg, T. (2001). Evaluation of rat ultrasonic vocalizations as predictors of the conditioned aversive effects of drugs. *Psychopharmacology*, *155*, 35–42.
- Burgdorf, J., & Panksepp, J. (2006). The neurobiology of positive emotions. *Neuroscience and Biobehavioral Reviews*, *30*, 173–187.
- Burgdorf, J., Wood, P.L., Kroes, R.A., Moskal, J.R., & Panksepp, J. (2007). Neurobiology of 50-kHz ultrasonic vocalizations in rats: Electrode mapping, lesion, and pharmacological studies. *Behavioural Brain Research*, *182*, 274–283.
- Burghardt, G.M. (2005). *The genesis of animal play*. Cambridge, MA: MIT Press.
- Burkhardt, R.W., Jr. (2005). *Patterns of behavior: Konrad Lorenz, Niko Tinbergen, and the founding of ethology*. Chicago: University of Chicago Press.
- Craig, A.D. (2003). How do you feel? Interoception: The sense of the physiological condition of the body. *Nature Reviews. Neuroscience*, *3*, 655–666.
- Cowie, F. (1999). *What's within? Nativism reconsidered*. Oxford, United Kingdom: Oxford University Press.

- Damasio, A.R., Grabowski, T.J., Bechara, A., Damasio, H., Ponto, L.L.B., Parvizi, J., & Hichwa, R.D. (2000). Sub-cortical and cortical brain activity during the feeling of self-generated emotions. *Nature Neuroscience*, 3, 1049–1056.
- Darwin, C. (1998). *The expression of emotions in man and animals* (3rd ed.). New York: Oxford University Press. (Original work published 1872)
- Davis, K.L., Panksepp, J., & Normansell, L. (2003). The affective neuroscience personality scales: Normative data and implications. *Neuro-Psychoanalysis*, 5, 21–29.
- Denton, D. (2006). *The primordial emotions: The dawning of consciousness*. New York: Oxford University Press.
- Depue, R.A., & Collins, P.F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences*, 22, 491–517.
- Diener, E., Lucas, R.E., & Scollon, C.N. (2006). Beyond the hedonic treadmill: Revising the adaptation theory of well-being. *American Psychologist*, 61, 305–314.
- Ekman, P. (2003). *Emotions revealed*. New York: Times Books.
- Esch, T., & Stefano, G.B. (2005). The neurobiology of love. *Neuroendocrinology Letters*, 26, 175–192.
- Frijda, N.H. (1986). *The emotions*. London: Cambridge University Press.
- George, M.S., Ketter, T.A., Parekh, P.I., Horowitz, B., Herschovitch, P., & Post, R.M. (1995). Brain activity during transient sadness and happiness in healthy women. *American Journal of Psychiatry*, 152, 341–351.
- Goel, V., & Dolan, R.J. (2003). Reciprocal neural response within lateral and ventral medial prefrontal cortex during hot and cold reasoning. *NeuroImage*, 20, 2314–2321.
- Gordon, N.S., Panksepp, J., Dennis, M., & McSweeney, J. (2005). The instinctual basis of human affect: Affective and fMRI imaging of laughter and crying. *Neuro-Psychoanalysis*, 7, 215–217.
- Heath, R.G. (1996). *Exploring the mind-body relationship*. Baton Rouge, LA: Moran Printing.
- Herman, B.H., & Panksepp, J. (1978). Effects of morphine and naloxone on separation distress and approach attachment: Evidence for opiate mediation of social affect. *Pharmacology, Biochemistry, & Behavior*, 9, 213–220.
- Herman, B.H., & Panksepp, J. (1981). Ascending endorphinergic inhibition of distress vocalization. *Science*, 211, 1060–1062.
- Holstege, G., Georgiadis, J.R., Paans, A.M., Meiners, L.C., van der Graaf, F.H., & Reinders, A.A. (2003). Brain activation during human male ejaculation. *Journal of Neuroscience*, 23, 9185–9193.
- Ikemoto, S., & Panksepp, J. (1999). The role of nucleus accumbens DA in motivated behavior, a unifying interpretation with special reference to reward-seeking. *Brain Research Reviews*, 31, 6–41.
- Ikemoto, S., & Wise, R.A. (2004). Mapping of chemical trigger zones for reward. *Neuropharmacology*, 47(Suppl. 1), 190–201.
- Izard, C.E., Youngstrom, E.A., Fine, S.E., Mostow, A.J., & Trentacosta, C.J. (2006). Emotions and developmental psychopathology. In D. Cicchetti & D.J. Cohen (Eds.), *Developmental psychology: Theory and method* (2nd ed., Vol. 1, pp. 244–292). New York: John Wiley & Sons.
- Jarbe, T.U. (1986). State-dependent learning and drug discriminative control of behaviour: An overview. *Acta Neurologica Scandinavica*, 109, 37–59.
- Kalin, N., & Shelton, S. (1989). Defensive behaviors in infant rhesus monkeys: Environmental cues and neurochemical regulation. *Science*, 243, 1718–1721.
- Kalin, N., Shelton, S., & Barksdale, C. (1988). Opiate modulation of separation-induced distress in non-human primates. *Brain Research*, 440, 285–292.
- Kirsch, P., Esslinger, C., Chen, Q., Mier, D., Lis, S., Siddhanti, S., et al. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *Journal of Neuroscience*, 25, 11489–11493.
- Klein, D. (1964). Delineation of two drug-responsive anxiety syndromes. *Psychopharmacology*, 5, 397–408.
- Knutson, B., Burgdorf, J., & Panksepp, J. (2002). Ultrasonic vocalizations as indices of affective states in rat. *Psychological Bulletin*, 128, 961–977.
- Kolb, B., & Tees, C. (Eds.). (2000). *The cerebral cortex of the rat*. Cambridge, MA: MIT Press.
- Kosfeld, M., Heinrichs, M., Zak, P.J., Fischbacher, U., & Fehr, E. (2005). Oxytocin increases trust in humans. *Nature*, 435, 673–676.
- Krack, P., Kumar, R., Ardouin, C., Dowsey, P.L., McVicker, J.M., Benabid, A.L., & Pollak, P. (2001). Mirthful laughter induced by subthalamic nucleus stimulation. *Movement Disorders*, 16, 867–875.
- Kuppens, P., Ceulemans, E., Timmerman, M.E., Diener, E., & Kim-Prieto, C. (2006). Universal intracultural and intercultural dimensions of the recalled frequency of emotional experience. *Journal of Cross-Cultural Psychology*, 37, 491–515.
- Lane, R.D., Reiman, E.M., Ahern, G.L., Schwartz, G.E., & Davidson, R.J. (1997). Neuroanatomical correlates of happiness, sadness, and disgust. *American Journal of Psychiatry*, 154, 926–933.
- LeDoux, J.E. (1996). *The emotional brain*. New York: Simon & Schuster.
- Liotti, M., & Panksepp, J. (2004). On the neural nature of human emotions and implications for biological psychiatry. In J. Panksepp, (Ed.), *Textbook of biological psychiatry* (pp. 33–74). New York: Wiley.
- MacLean, P. (1990). *The triune brain*. New York: Plenum Press.
- Mayberg, H.S., Lozano, A.M., Voon, V., McNeeley, H.E., Seminowicz, D., Hamani, C., et al. (2004). Deep brain stimulation for treatment-resistant depression. *Neuron*, 45, 651–660.
- Meaney, M.J., & Szyf, M. (2005). Maternal care as a model for experience-dependent chromatin plasticity? *Trends in Neurosciences*, 28, 456–463.
- Merker, B. (2007). Consciousness without a cerebral cortex: A challenge for neuroscience and medicine. *Behavioral and Brain Sciences*, 30, 63–134.
- Murphy, F.C., Nimmo-Smith, I., & Lawrence, A.D. (2003). Functional neuroanatomy of emotion: A meta-analysis. *Cognitive, Affective, & Behavioral Neuroscience*, 3, 207–233.
- Naqvi, N.H., Rudrauf, D., Damasio, H., & Bechara, A. (2007). Damage to the insula disrupts addiction to cigarette smoking. *Science*, 315, 531–534.
- Nelson, E.E., & Panksepp, J. (1998). Brain substrates of infant-mother attachment: Contributions of opioids, oxytocin, and norepinephrine. *Neuroscience & Biobehavioral Reviews*, 22, 437–452.
- Northoff, G., Heinzel, A., Birmpohl, F., Niese, R., Pfennig, A., Pascual-Leone, A., & Schlaug, G. (2004). Reciprocal modulation and attenuation in the prefrontal cortex: An fMRI study on emotional-cognitive interaction. *Human Brain Mapping*, 21, 202–212.
- Northoff, G., Heinzel, A., de Greck, M., Birmpohl, F., & Panksepp, J. (2006). Our brain and its self: The central role of cortical midline structures. *NeuroImage*, 15, 440–457.
- Numan, M., & Insel, T.R. (2003). *The neurobiology of parental behavior*. New York: Springer.

- Okun, M.S., Bowers, D., Springer, U., Shapira, N.A., Malone, D., Rezai, A.R., et al. (2004). What's in a smile? Intra-operative observations of contralateral smiles induced by deep brain stimulation. *Neurocase*, *10*, 271–279.
- Olds, J. (1977). *Drives and reinforcements: Behavioral studies of hypothalamic functions*. New York: Raven Press.
- Olmstead, M.C., & Franklin, K.B.J. (1997). The development of a conditioned place preference to morphine. Effects of microinjections into various CNS sites. *Behavioral Neuroscience*, *111*, 1324–1334.
- Ortony, A., & Turner, T.J. (1990). What's basic about basic emotions? *Psychological Review*, *97*, 315–331.
- Overton, D.A. (1991). A historical perspective on drug discrimination. *NIDA Research Monographs*, *116*, 5–24.
- Panksepp, J. (1971). Aggression elicited by electrical stimulation of the hypothalamus in albino rats. *Physiology & Behavior*, *6*, 311–316.
- Panksepp, J. (1974). Hypothalamic regulation on energy balance and feeding behavior. *Federation Proceedings*, *33*, 1150–1165.
- Panksepp, J. (1981a). Brain opioids: A neurochemical substrate for narcotic and social dependence. In S. Cooper (Ed.), *Progress in theory in psychopharmacology* (pp. 149–175). London: Academic Press.
- Panksepp, J. (1981b). Hypothalamic integration of behavior: Rewards, punishments, and related psychobiological process. In P.J. Morgane & J. Panksepp (Eds.), *Handbook of the hypothalamus: Vol. 3, Part A. Behavioral studies of the hypothalamus* (pp. 289–487). New York: Marcel Dekker.
- Panksepp, J. (1982). Toward a general psychobiological theory of emotions. *Behavioral and Brain Sciences*, *5*, 407–467.
- Panksepp, J. (1985). Mood changes. In P. Vincken, G. Bruyn, & H. Klawans (Eds.), *Handbook of clinical neurology* (Vol. 45, pp. 271–85). Amsterdam: Elsevier.
- Panksepp, J. (1986a). The anatomy of emotions. In R. Plutchik (Ed.), *Emotion: Theory, research and experience: Vol. 3. Biological foundations of emotions* (pp. 91–124). Orlando, FL: Academic Press.
- Panksepp, J. (1986b). The neurochemistry of behavior. *Annual Review of Psychology*, *37*, 77–107.
- Panksepp, J. (1990). Can “mind” and behavior be understood without understanding the brain? A response to Bunge. *New Ideas in Psychology*, *8*, 139–149.
- Panksepp, J. (1991). Affective neuroscience: A conceptual framework for the neurobiological study of emotions. In K. Strongman (Ed.), *International reviews of emotion research* (pp. 59–99). Chichester, United Kingdom: Wiley.
- Panksepp, J. (1992). A critical role for “affective neuroscience” in resolving what is basic about basic emotions. *Psychological Review*, *99*, 554–560.
- Panksepp, J. (1993). Neurochemical control of moods and emotions: Amino acids to neuropeptides. In M. Lewis & J. Haviland (Eds.), *Handbook of emotions* (pp. 87–107). New York: Guilford Press.
- Panksepp, J. (1998a). *Affective neuroscience: The foundations of human and animal emotions*. New York: Oxford University Press.
- Panksepp, J. (1998b). The periconscious substrates of consciousness: Affective states and the evolutionary origins of the SELF. *Journal of Consciousness Studies*, *5*, 566–582.
- Panksepp, J. (1999). Emotions as viewed by psychoanalysis and neuroscience: An exercise in consilience. *Neuro-Psychoanalysis*, *1*, 15–38.
- Panksepp, J. (2000a). Affective consciousness and the instinctual motor system: The neural sources of sadness and joy. In R. Ellis & N. Newton (Eds.), *The caldron of consciousness: Motivation, affect and self-organization, advances in consciousness research* (pp. 27–54). Amsterdam: John Benjamins.
- Panksepp, J. (2000b). The riddle of laughter: Neural and psychoevolutionary underpinnings of joy. *Current Directions in Psychological Sciences*, *9*, 183–186.
- Panksepp, J. (2001). The long-term psychobiological consequences of infant emotions: Prescriptions for the 21st century. *Infant Mental Health Journal*, *22*, 132–173.
- Panksepp, J. (2003a). At the interface of affective, behavioral and cognitive neurosciences: Decoding the emotional feelings of the brain. *Brain and Cognition*, *52*, 4–14.
- Panksepp, J. (2003b). Feeling the pain of social loss. *Science*, *303*, 237–239.
- Panksepp, J. (Ed.). (2004). *Textbook of biological psychiatry*. New York: Wiley.
- Panksepp, J. (2005a). Affective consciousness: Core emotional feelings in animals and humans. *Consciousness & Cognition*, *14*, 30–80.
- Panksepp, J. (2005b). On the embodied neural nature of the core emotional affects. *Journal of Consciousness Studies*, *5*, 158–184.
- Panksepp, J. (2005c). Social support and pain: How does the brain feel the ache of a broke heart. *Journal of Cancer Pain & Symptom Palliation*, *1*, 59–65.
- Panksepp, J. (2006a). The core emotional systems of the mammalian brain: The fundamental substrates of human emotions. In J. Corrigan, H. Payne, & H. Wilkinson (Eds.), *About a body: Working with the embodied mind in psychotherapy* (pp. 14–32). Hove, United Kingdom: Routledge.
- Panksepp, J. (2006b). Emotional endophenotypes in evolutionary psychiatry. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *30*, 774–784.
- Panksepp, J. (2007). Neuroevolutionary sources of laughter and social joy: Modeling primal human laughter in laboratory rats. *Behavioral Brain Research*, *182*, 231–244.
- Panksepp, J., & Burgdorf, J. (2003). “Laughing” rats and the evolutionary antecedents of human joy? *Physiology & Behavior*, *79*, 533–547.
- Panksepp, J., & Gordon, N. (2003). The instinctual basis of human affect: Affective imaging of laughter and crying. *Consciousness & Emotion*, *4*, 197–206.
- Panksepp, J., & Harro, J. (2004). The future of neuropeptides in biological psychiatry and emotional psychopharmacology: Goals and strategies. In J. Panksepp (Ed.), *Textbook of biological psychiatry* (pp. 627–660). Hoboken, NJ: Wiley.
- Panksepp, J., Herman, B., Conner, R., Bishop, P., & Scott, J.P. (1978). The biology of social attachments: Opiates alleviate separation distress. *Biological Psychiatry*, *9*, 213–220.
- Panksepp, J., Herman, B.H., Vilberg, T., Bishop, P., & DeEsquinazi, F.G. (1980). Endogenous opioids and social behavior. *Neuroscience and Biobehavioral Reviews*, *4*, 473–487.
- Panksepp, J., Jalowiec, J., DeEsquinazi, F.G., & Bishop, P. (1985). Opiates and play dominance in juvenile rats. *Behavioral Neuroscience*, *99*, 441–453.
- Panksepp, J., Knutson, B., & Burgdorf, J. (2002). The role of emotional brain systems in addictions: A neuro-evolutionary perspective. *Addiction*, *97*, 459–469.
- Panksepp, J., Normansell, L.A., Cox, J.K.F., & Siviy, S. (1994). Effects of neonatal decortication on the social play of juvenile rats. *Physiology & Behavior*, *56*, 429–443.

- Panksepp, J., Normansell, L.A., Herman, B., Bishop, P., & Crepeau, L. (1988). Neural and neurochemical control of the separation distress call. In J.D. Newman (Ed.), *The physiological control of mammalian vocalizations* (pp. 263–300). New York: Plenum.
- Panksepp, J., Nocjar, C., Burgdorf, J., Panksepp, J.B., & Huber, R. (2004). The role of emotional systems in addiction: A neuroethological perspective. In R.A. Bevins (Ed.), *Nebraska symposium on motivation: Vol. 50. Motivational factors in the etiology of drug abuse* (pp. 85–126). Lincoln: University of Nebraska Press.
- Panksepp, J., & Panksepp, J.B. (2000). The seven sins of evolutionary psychology. *Evolution & Cognition*, 6, 108–131.
- Panksepp, J., Sacks, D.S., Crepeau, L., & Abbott, B.B. (1991). The psycho- and neuro-biology of fear systems in the brain. In M.R. Denny (Ed.), *Aversive events and behavior* (pp. 7–59). New York: Erlbaum.
- Panksepp, J., Vilberg, T., Bean, N.J., Coy, D.H., & Kastin, A.J. (1978). Reduction of distress vocalization in chicks by opiate-like peptides. *Brain Research Bulletin*, 3, 663–667.
- Parvizi, J., & Damasio, A. (2001). Consciousness and the brainstem. *Cognition*, 79, 135–160.
- Peciña, S., Smith, K., & Berridge, K.C. (2006). Hedonic hot spots in the brain. *Neuroscientist*, 12, 500–511.
- Pfaff, D.W. (1999). *Drive: Neurobiological and molecular mechanisms of sexual behavior*, Cambridge, MA: MIT Press.
- Phan, K.L., Wager, T.D., Taylor, S.F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: A meta-analysis of emotion activation studies in PET and fMRI. *NeuroImage*, 16, 331–348.
- Plutchik, R. (2003). *Emotions and life: Perspectives from psychology, biology, and evolution*. Washington, DC: American Psychological Association.
- Rolls, E.T. (1975). *The brain and reward*. Oxford, United Kingdom: Pergamon Press.
- Rolls, E.T. (2005). *Emotions explained*. Oxford, United Kingdom: Oxford University Press.
- Russell, J.A. (1995). Facial expressions of emotion: What lies beyond minimal universality? *Psychological Bulletin*, 118, 379–391.
- Russell, J.A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, 110, 145–173.
- Schutter, D.J.L.G., Van Honk, J., & Panksepp, J. (2004). Introducing repetitive transcranial magnetic stimulation (rTMS) and its property of causal inference in investigating the brain-function relationship. *Synthese*, 141, 155–173.
- Scott, J.P. (1974). Effects of psychotropic drugs on separation distress in dogs. In *Proceedings of the 9th Congress on Neuropsychopharmacology, CINP* (pp. 735–745). Paris: Excerpta Medica Amsterdam.
- Sewards, T.V., & Sewards, M.A. (2000). The awareness of thirst: Proposed neural correlates. *Consciousness and Cognition*, 9, 463–487.
- Shapira, N.A., Okun, M.S., Wint, D., Foote, K.D., Byars, J.A., Bowers, D., et al. (2006). Panic and fear induced by deep brain stimulation. *Journal of Neurology, Neurosurgery, and Psychiatry*, 77, 410–412.
- Shen, J., Barbera, J., & Shapiro, C.M. (2006). Distinguishing sleepiness and fatigue: Focus on definition and measurement. *Sleep Medicine Review*, 10, 63–76.
- Shewmon, D.A., Holmse, D.A., & Byrne, P.A. (1999). Consciousness in congenitally decorticate children: Developmental vegetative state as self-fulfilling prophecy. *Developmental Medicine and Child Neurology*, 41, 364–374.
- Siegel, A. (2005). *The neurobiology of aggression and rage*. Boca Raton, FL: CRC Press.
- Stutz, R.M., Rossi, R.R., Hastings, L., & Brunner, R.L. (1974). Discriminability of intracranial stimuli: The role of anatomical connectedness. *Physiology & Behavior*, 12, 69–73.
- Sunderland, M. (2006). *The science of parenting*. London: Dorling Kindersley.
- Swanson, L.W. (2003). *Brain architecture: Understanding the basic plan*. New York: Oxford University Press.
- Thorndike, E.L. (1911). *Animal intelligence*. New York: MacMillan & Co.
- Tomkins, S.S. (1962). *Affect, imagery, consciousness: Vol. 1. The positive affects*. New York: Springer.
- Tomkins, S.S. (1963). *Affect, imagery, consciousness: Vol. 2. The negative affects*. New York: Springer.
- Turner, T.J., & Ortony, A. (1992). Basic emotions: Can conflicting criteria converge? *Psychological Review*, 99, 566–571.
- Vianna, D.M.L., & Brandão, M.L. (2003). Anatomical connections of the periaqueductal gray: Specific neural substrates of different kinds of fear. *Brazilian Journal of Medical and Biological Research*, 36, 557–566.
- Vogt, B.A. (2005). Pain and emotion interactions in subregions of the cingulate gyrus. *Nature Reviews. Neuroscience*, 6, 533–544.
- Watt, D.F. (2000). The centrencephalon and thalamocortical integration. Neglected contributions of periaqueductal gray. *Consciousness & Emotion*, 1, 91–114.
- Watt, D.F., & Pincus, D.I. (2004). Neural substrates of consciousness: Implications for clinical psychiatry. In J. Panksepp (Ed.), *Textbook of biological psychiatry* (pp. 627–660). Hoboken, NJ: Wiley.
- Zachar, P., & Bartlett, S. (2001). Basic emotions and their biological substrates: A nominalistic interpretation. *Consciousness & Emotion*, 2, 189–221.
- Zangen, A., Solinas, M., Ikemoto, S., Goldberg, S.R., & Wise, R.A. (2006). Two brain sites for cannabinoid reward. *Journal of Neuroscience*, 26, 4901–4907.
- Zeisberger, E. (1998). Biogenic amines and thermoregulatory changes. *Progress in Brain Research*, 115, 159–176.
- Zeki, S. (2007). The neurobiology of love. *Federation of European Biochemical Societies Letters*, 581, 2575–2579.
- Zubieta, J.K., Ketter, T.A., Bueller, J.A., Xu, Y., Kilbourn, M.R., Young, E.A., & Koeppe, R.A. (2003). Regulation of human affective responses by anterior cingulate and limbic mu-opioid neurotransmission. *Archives of General Psychiatry*, 60, 1145–1153.