Of Mice and Men

Natural Kinds of Emotions in the Mammalian Brain? A Response to Panksepp and Izard

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ABSTRACT—*For almost 5 decades, the scientific study of emotion has been guided by the assumption that categories such as anger, sadness, and fear cut nature at its joints. Barrett (2006a) provided a comprehensive review of the empirical evidence from the study of emotion in humans and concluded that this assumption has outlived its usefulness. Panksepp and Izard have written lengthy papers (published in this issue) containing complementary but largely nonoverlapping criticisms of Barrett (2006a). In our response, we address three of their concerns. First, we discuss the value of correlational versus experimental studies for evaluating the natural-kind model of emotion and refute the claim that the evidence offered in Barrett (2006a) was merely correlational. Second, we take up the issue of whether or not there is evidence for "coherently organized neural circuits" for natural kinds of emotions in the mammalian brain and counter the claim that Barrett (2006a) ignored crucial evidence for existence of discrete emotions as natural kinds. Third, we address Panksepp and Izard's misconceptions of an alternative view, the conceptual act model of emotion, that was briefly discussed in Barrett (2006a). Finally, we end the article with some thoughts on how to move the scientific study of emotion beyond the debate over whether or not emotions are natural kinds.*

> "It would be very surprising indeed if the brain were organized into spatially discrete units that conform to our abstract categorizations of behavior."
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> (Valenstein, 1973, pp. 142–143)

According to the National Academy of Sciences in the United States, a theory is a well-substantiated explanation of a phenomenon. A theory is the end point of science—it is what scientists know to be true when observations have been confirmed by repeated experimentation (National Academy of Sciences, 1998). A hypothesis, on the other hand, is a tentative statement that must be tested. Barrett (2006a) demonstrated that after a century of empirical research, the natural-kind view of emotion is not yet a theory. It remains a set of hypotheses—or what we might call a model—subject to the same rules of scientific verification as any other model of emotion. It is a fact that people experience phenomena that are called (in English) *anger*, *sadness*, and *fear*. It is a fact that people experience these psychological states as discrete events that are bounded in time and that people often (but not always) experience these states as psychologically distinct from one another. It is also a fact that people easily and effortlessly see anger and sadness and fear in the behaviors of other people, including babies, and in nonhuman animals. People even see these emotions in the behaviors of shapes (squares, circles, and triangles) that move in a particular relation to one another (Heider & Simmel, 1944). It is the task of science to explain these facts: to explain how the events that people experience as anger, sadness, or fear are caused and how they are entailed in the brain. It is compelling to believe that "SEEKING, RAGE, FEAR, LUST, CARE, PANIC, and PLAY" (see Panksepp, 2007, this issue) or "interest, joy/happiness, sadness, anger, disgust, and fear" (Izard, 2007, this issue) are biologically basic and derive from architecturally and chemically distinct circuits that are hard coded into the human brain at birth. Statements to this effect, no matter how often or forcefully made, are not yet facts; they are hypotheses. Panksepp (2007) and Izard (2007) are eminent scientists who have contributed both empirical observations and conceptual analyses to the literature on emotion, and, as they both correctly point out, there is some evidence to support the idea that emotions are natural kinds. As Barrett (2006a) illustrates, however, there is also a tremendous amount of evidence that is inconsistent with this idea.

In the scientific study of emotion, one particular fault line has been emphasized: The psychological events referred to by the

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English words *anger*, *sadness*, *fear*, *disgust*, and *happiness* are either discussed as complex reflexes that are automatically triggered by stimuli that organisms are prepared to respond to (the basic-emotion approach) or as responses that result from a meaningful interpretation of the situation (the appraisal approach). This distinction is sometimes phrased as a distinction between evolutionary and social constructivist causes of emotion. Although the two models clearly differ in some ways, Barrett (2006a) discussed how both perspectives share one core idea: Certain emotions are presumed to be natural kinds or phenomena that have firm boundaries in nature that exist independent of perception. The natural-kind model of emotion was popular in the early 20th century (Allport, 1924; MacDougall, 1908/1926) and was revived by Tompkins (1962, 1963) and Arnold (1960) after a long hiatus. It has defined the boundaries for emotion research ever since. Some theorists characterize natural kinds of emotion by analogy (i.e., the individual instances that we call by the same name, such as *anger*, are presumed to look the same or to share a distinctive collection of properties that co-occur). Other theorists characterize natural kinds of emotion by homology (i.e., the instances of an emotion kind, such as anger, are presumed to derive from the same causal mechanism).

Barrett (2006a) pointed out that whether defined by analogy or homology, empirical evidence from human experience, behavior, facial movements, psychophysiology, and cognitive neuroscience is steadily accumulating to disconfirm the natural-kind model of emotion. Not all instances of an emotion (e.g., what people call *fear*) look alike, feel alike, or have the same neurophysiological signature (i.e., they are not analogous). As a result, the natural-kind model cannot explain the considerable variability of emotional life that has been observed within individuals over time, across individuals from the same culture, and of course, across cultures. Even rats display behavioral flexibility that is context dependent. In the natural-kind model, such heterogeneity in emotional life is either treated as error or is explained by processes added to the model post hoc (e.g., display rules). To understand what emotions are and how they work, however, scientists must understand and model this variability, not explain it away. Furthermore, homologous emotion circuits of the sort presumed by the natural-kind model are unlikely to exist given what is known about the evolution of the human brain. When compared with other mammals, the human brain has seen a rapid expansion in the isocortical aspects of affective circuitry along with increasingly dense reciprocal projections to subcortical areas (some of which have evolved in concert with the cortical changes).[1] Together with the pronounced interspecies differences that exist in cognition and

behavior, these changes suggest that the human brain may function very differently when compared with nonprimate, mammalian species such as rats, calling into question the existence of strong emotion homologies. As a result, animal models yield necessary and important insights that must be incorporated into any model of emotion, but they have not (and probably cannot) give a sufficient account of the events people call *fear*, *anger*, or *sadness*.

In this issue of *Perspectives on Psychological Science*, Panksepp and Izard have written complementary but largely nonoverlapping critiques of Barrett (2006a). Izard offers a fine conceptual analysis of the basic-emotion approach and again reviews some evidence that is consistent with the hypothesis that basic emotions are natural kinds. He largely fails, however, to address any of disconfirming evidence summarized by Barrett (2006a). Panksepp, on the other hand, claims that the evidence reviewed by Barrett cannot be used to disconfirm the natural-kind view because it is correlational rather than causal. He claims that a "massive" amount of causal evidence exists to support the hypothesis that anger, sadness, fear, and a few others are natural kinds in what he calls "the mammalian brain" (Panksepp, 2007, p. 286).

The most general response to both critiques, but especially to Izard's, is that individual studies may be consistent with the natural-kind view of emotion, even when the combined body of evidence disconfirms it. The field needs a model of emotion that can account for all the published data. Izard cites individual studies that indeed provide support for the natural-kind view, but he fails to acknowledge the many studies that are incongruent with this view (discussed in Barrett, 2006a). Obviously, incidental support for the natural-kind view does not invalidate our analysis, which was based on this much larger and comprehensive body of evidence.

In the remainder of this article, we outline our specific disagreements with three main criticisms of Barrett (2006a) that appear in both commentaries (Izard, 2007; Panksepp, 2007). First, we argue that the evidence offered in Barrett (2006a) was not merely correlational, although correlational studies do enable an evaluation of the natural-kind model of emotion that is complementary to the evidence provided by so-called causal studies. Second, we argue that, despite suggestions to the contrary, there is no conclusive evidence for the existence of "at least seven prototype emotional systems in the mammalian brain" (Panksepp, 2007, p. 286) and, accordingly, that Barrett (2006a) did not fail to review evidence that was crucial to the argument that discrete emotions are natural kinds. Finally, we end the article by elaborating on the conceptual act model that was briefly mentioned in Barrett (2006a). The main thrust of Barrett (2006a) was to offer the suggestion that the current paradigm, grounded in natural-kind assumptions, has outlived its usefulness. Of course, reviewers want to see the old paradigm replaced by (at the very least) the outlines of a new one, but Barrett (2006a) was not meant as a complete discussion. The

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[1]The term *isocortex* is used instead of the more popular *neocortex* because there are very few new areas of cortex. Most areas of cortex have homologues in amphibian and other vertebrate brains, suggesting that the mammalian isocortex was not added like icing onto an already baked cake. Instead, it was radically transformed from a series of precursors (Striedter, 2005).

conceptual act model is set out in more detail in several recent publications (Barrett, 2006b, 2006c; Barrett & Lindquist, in press; Barrett, Lindquist, & Gendron, 2007; Barrett, Mesquita, Ochsner, & Gross, 2007; Barrett, Ochsner, & Gross, 2007; Duncan & Barrett, in press; Lindquist & Barrett, in press). Nonetheless, Panksepp and Izard critique the conceptual act model from their reading of Barrett (2006a), and so we address their criticisms. In the final section of the article, we share some thoughts on how to move the scientific study of emotion beyond the debate over whether or not emotions are natural kinds.

CORRELATIONS VERSUS EXPERIMENTS FOR DEDUCING CAUSATION

Panksepp argues (and Izard also briefly notes) that Barrett (2006a) reviewed mainly correlational evidence that is, in and of itself, not sufficiently robust to provide a strong test of the natural-kind model. We disagree with this claim for three reasons. First, many of the findings reviewed by Barrett (2006a) were in fact derived from experiments in which a person's mental state was manipulated and measures of subjective experience, facial muscle movement, the body, and/or brain activity were taken. Psychological manipulations (such as having people relive prior experiences of emotion, having them watch movies, and the like) are as experimentally valid for the purposes of inferring cause as are electrical or chemical stimulation or lesioning of a brain. These latter methods change existing and potential brain states more directly, to be sure, but manipulating the mental, by definition, also means manipulating the brain. Either way, it is possible to observe the consequences of such manipulations and determine whether they match predictions.

Second, Panksepp and Izard are correct that correlational studies cannot determine whether modular brain circuits cause (in Panksepp's terms) anger, sadness, and fear, and so they cannot verify the natural-kind model of emotion when kinds are defined by homology (or common cause, such as a brain circuit). But as Barrett (2006a) points out, natural kinds of emotion can also be defined by analogy (i.e., all instances of anger are presumed to look the same). Correlations are extremely useful and, in fact, are necessary for determining whether emotions are natural-kind categories when kinds are defined by analogy (for a discussion, see Barrett, 2006a).

Third, and perhaps most important, even experimental studies (even using rats) cannot conclusively determine cause; they can only suggest cause with a degree of certainty that is linked to the possibility of ruling out alternative explanations for the pattern of observed results. In an experiment using statistics based on least-squares-estimation procedures, rejecting the null hypothesis means that observations (following some manipulation) occur so infrequently in the population in which the null hypothesis is true, that it is more reasonable to assume that the observations came from some other population. But one can never accept the alternative hypothesis as true. The probability that the hypothesis of interest is true increases with experimental control (allowing alternative explanations for the observed findings to be dismissed). As we discuss in the next section (with examples in the Appendix), many of the causal experiments cited as evidence for the existence of modular brain circuits for emotion are open to alternative explanations.

CAUSAL EVIDENCE FOR SEVEN PROTOTYPE EMOTIONAL SYSTEMS IN THE MAMMALIAN BRAIN

We agree with Panksepp and Izard that all behaviors referred to as emotional are caused by neural activity and that all mental events, including the psychological events we call by the names *anger*, *sadness*, and *fear* (plus a few others), are instantiated by brain states. What is far from clear, however, is that these brains states are entailed by (or implemented in) fixed, architecturally and chemically distinct circuits such as those presumed by the natural-kind model of emotion (see Izard, 2007; Panksepp, 2007). In this section, we show (albeit briefly) that the evidence for coherently organized neural circuits for emotional behaviors in the mammalian brain is more suggestive than convincing.

First, we very briefly discuss whether the existing corpus of neuroscience research on nonhuman animals provides conclusive evidence for the existence of architecturally and chemically distinct subcortical circuits for seven classes of behavior that are each diagnostic of a discrete emotion. Second, we examine whether electrically stimulating the brain areas of each system reliably and consistently produce distinct behaviors in all mammals, as well as distinct feeling states in humans. Third, we consider how such subcortical circuits, if they indeed exist, should be understood in a human brain that is characterized by dense interconnectivity with paralimbic and prefrontal areas that exist in a more limited form in nonhuman mammals. Finally, we consider whether it is scientifically viable to think of circuits for behaviors of any sort (freezing, attacking, etc.) as evidence for the existence of natural kinds of emotions.

Core Emotional Systems in the Mammalian Brain?

In numerous articles published over the past decade, including his critique of Barrett (2006a), Panksepp has claimed that there is strong evidence for the existence of seven architecturally and/or chemically distinct circuits in the subcortex of the mammalian brain, each of which produces a constellation of behaviors (e.g., grooming, retrieving pups, and nursing) associated with a distinct experiential state (e.g., love) and constitutes the circuitry for a discrete or basic emotion (e.g., CARE). Izard also claims seven, but his list is a bit different (for a discussion, see Ortony & Turner, 1990). Our review of the literature leads us to challenge this claim; for the sake of brevity, we will focus on Panksepp's model (although our points apply to Izard's model as well).

In our view, Panksepp's hypothesized systems fall into three general categories. Some behaviors referred to as emotional (i.e., those associated with the putative PLAY circuit) do not yet

correspond to a well-worked-out circuit, or the behaviors are sufficiently diverse that they do not constitute a single psychological category, let alone a biological one (i.e., the putative RAGE circuit). Other behaviors (i.e., those associated with the putative PANIC, SEEKING, and FEAR circuits) are indeed instantiated by functionally identifiable neural circuits, but it is not clear that the behaviors themselves are emotional in nature (meaning that there is evidence to indicate that the behaviors reflect psychological categories other than sadness, expectancy, and fear). And still other behaviors (i.e., those associated with the putative CARE and LUST circuits) are instantiated by specific neural circuitry, but whether or not these behavioral circuits actually invoke phenomenologically distinct feeling states and therefore constitute circuitry for natural kinds of emotion is an altogether different issue. We provide examples to support these observations in the Appendix.

Electrical Stimulation of the Mammalian Brain
According to Panksepp, electrical stimulation studies provide the most direct evidence for the existence of natural kinds of emotion. In his 1998 book, he writes:

> Because of learning and the rapid development of behavioral habits, one can never capture innate emotional dynamics in their pure form, except perhaps when they are aroused artificially by direct stimulation of brain areas where those operating systems are most concentrated. I will refer to such experiments in subsequent chapters as one of the main lines of evidence to support the existence and provisional localization of emotional operating systems. It is now well established that one can reliably evoke several distinct emotional patterns in all mammals during electrical stimulation of homologous subcortical regions. . . .
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> (Panksepp, 1998, p. 26)

Direct electrical stimulation of the brain delivers electrical current of different intensities and duration to specific brain sites via surgically implanted electrodes (which are placed with a combination of imaging and precise stereotactic landmarking procedures). Changes in behavior, subjective experience, and neuronal firing elsewhere in the brain can then be observed. These experiments would, indeed, seem to have the greatest potential for providing direct "causal" evidence for the existence of natural kinds of emotion, because they typically do not permanently alter brain tissue (which can lead to a functional reorganization of brain circuitry). In this issue, Panksepp writes, "Can one evoke such emotion patterns using ESB [electrical stimulation of the brain] in homologous brain regions across species? The answer is yes . . . Do humans have such systems? They do" (Panksepp, 2007, p. 286). To bolster this claim, Panksepp cites Heath (1996) and his own review chapter (Panksepp, 1985).

We were unable to find a bibliographic record of the Heath (1996) book, but Panksepp (1985) cites nine primary sources, including Sem-Jacobson (1968) and Halgren, Walter, Cherlow,

and Crandall (1978), as well as ten or so secondary sources as apparently showing evidence that "emotive behavior patterns indicative of such emotional states can be elicited by localized electrical stimulation of transhypothalamic neural pathways and their higher and lower projection areas in lower mammals and . . . in human brain stimulation studies." (Panksepp, 1985, p. 272). Our review of the literature, however, suggests a rather different conclusion: The results of brain stimulation studies are consistent with the idea that affect (either pleasant or unpleasant) can be (but is not always) elicited from stimulating loci in the brain, and it is an inference to claim anything more (either because the studies in question measured experience in general affective terms such as relaxation, alertness, euphoria, depression, confusion, or because they failed to measure responses that would allow for more specific conclusions over and above those related to valence or arousal).

Sem-Jacobson (1968), for example, detailed that patients reported several categories of mood changes (relaxed; happy; euphoric; anxious, tense, or sad; irritable or mildly depressed; strongly irritable; depressed, angry, afraid, or scared; sudden emotional outbursts in either a positive or negative direction; ambivalence; and satiation responses in which stimulation produced such a positive feeling that additional stimulation in that area was experienced as unpleasant). These mood-related changes were not completely reliable and did not clearly configure into architecturally distinct circuits. In certain cases, electrical stimulation produced emotional behavior (such as laughing) in the absence of experience (such as pleasure).

We were also able to locate dozens of additional papers spanning several decades of electrical stimulation experiments, many of which are summarized by the renowned neuroscientist Elliot Valenstein (Valenstein, 1973; see also Valenstein, Cox, & Kakolewski, 1970). Evoked behavioral and experiential changes (that people would call emotional) can and do happen but not each and every time a specific brain area is stimulated. On the contrary, variability is the norm. Valenstein states:

> The impression exists that if electrodes are placed in a specific part of the brain, a particular behavior can inevitably be evoked. Those who have participated in this research know that this is definitely not the case. In a large percentage of cases, animals do not display any specific behavior in response to stimulation, even though great care may have been exerted to position the brain electrodes with as much precision as possible. Even in rats, where the behavior is more stereotyped than in monkeys and man, brain stimulation produces very variable results. (p. 88)

He then goes on to say the following:

> The experimental data clearly indicate that electrodes that seem to be in the same brain locus in different animals often evoke different behavior, and electrodes located at very different brain sites may evoke the same behavior in a given animal. (p. 89)

And finally, he suggests:

> It is unrealistic to think that the same stimulation would invariably evoke the same response. Part of the problem is that even among researchers who should know better, there is a tendency to think of the nervous system within too static a framework. It is not realistic to conceive of all nerve cells responding without variation to the same stimulus and being arranged without variation to convey impulses in a fixed direction and sequence. (p. 112)

Valenstein (1973) describes study after study demonstrating that the behaviors and experiences elicited from electrical stimulation are strongly influenced by the context in which the stimulation took place and by the preexisting temperament of the stimulated animal (pp. 86–114). For example, the behavior displayed in response to the same brain stimulation depends on the relative social rank of the animals present in the context (Ploog, 1970). Amygdala stimulation is associated with increases in aggressive behavior, but only in individuals who were observed to be violent before the experiment (Kim & Umbach, 1973), and is associated with reports of fear, but primarily in patients who seemed apprehensive about the stimulation procedure per se (Van Buren, 1961).

Furthermore, it is not clear that stimulating a specific area necessarily and sufficiently instantiates an experiential state. Valenstein (1973, p. 91) describes how a rat will eat pellets with hypothalamic stimulation but will not eat the pellets ground up into a mash (even though a hungry rat will eat either; Valenstein, Cox, & Kakolewski, 1968b). If pellets are not available, the animals may drink or even gnaw wood, and these alternative behaviors will continue even when pellets are returned to the test chamber (Valenstein, Cox, & Kakolewski, 1968a; see also Valenstein et al., 1970). These studies indicate that electrical stimulation of the hypothalamus alone does not necessarily produce a specific motivational state (such as hunger) and that the behavior elicited may be maintained by something other than the presumed state.

Later studies tend to support Valenstein's conclusions. A very well-controlled electrical stimulation of the temporal lobe (including the amygdala) in humans gave absolutely no evidence of clear brain-locus/experience relationships (Halgren et al., 1978). Of the 3,495 stimulations that were performed on 36 patients, only 267 elicited a mental response of any sort (35 reports of emotional experience reported as anger, fear, tension, or nervousness were observed across 8 patients). Mental responses were highly variable within participants across time and across participants. Stimulation of a given anatomical site produced different experiences in different patients, and stimulation at different sites produced the same mental content. As a consequence, there was little evidence that any mental contents were evoked by activation of a discrete and focal neuronal system. Halgren et al. state:

> There is no apparent tendency for any category of mental phenomena to be evoked more easily from any particular site, once the general tendency for all mental phenomena to be more readily evoked by anterior structures as compared to more posterior structures in taken into account. (p. 97)

Furthermore, Halgren et al. (1978) found that the type of experience was related to the personality of the patient rather than the precise location of the electrodes, which is very consistent with Valenstein's (1973) suggestion that "pre-existing temperament of the organism stimulated may be a better predictor of which behavior is obtained than the precise location of the stimulating electrode," (Halgren et al., 1978, p. 84).

Valenstein concludes:

> If studies with relatively homogenous, inbred animals suggested that there is a great amount of uncontrolled variability in the behavior produced by brain stimulation, we should expect an even greater source of unpredictability in the case of primates and especially humans. (p. 92)

This conclusion seems ever more apparent when you consider the striking evolutionary changes that have occurred in the primate brain over the past 65 million years or so, which we briefly review in the next section.[2]

The Human Brain

For the sake of argument, let's say that with more attention to anatomical details and with better experimental designs, scientists will discover circuits in subcortex of the rat brain for all fixed action patterns, much as they have for freezing or licking rat pups. Is it reasonable to expect these circuits to be similarly constituted in the human brain? Panksepp believes so. He writes, "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." (Panksepp, 2007, p. 286). There is some truth to this statement but only up to a point. There is no question that a complete scientific treatment of the psychological events called *emotion* requires an understanding of those processes that are species general and innate in the human brain (although something can be innate without being species general). We have great respect for what can be learned about behavior, core affect, and motivational states from the study of nonhuman animals. Yet, the idea of "core emotional systems" in the mammalian brain stem makes little sense to us on both logical and neuroanatomical grounds.

To believe in the existence of architecturally distinct emotion circuits as "genetically prescribed tools" (Panksepp, 2007, p. 290) comes close to the sort of preformation theory that Darwin's theory of evolution argued against (for a discussion of the irony of preformation theories in evolutionary biology, see

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[2]According to Tavare, Marshall, Will, Soligo, and Martin (2002), the first primates existed over 65 million years ago.

Lewontin, 2000; see also Hodos & Campbell, 1969). Even if strict structural homologies do exist (e.g., at the level of the brain stem), they need not function in exactly the same manner in both human and other animal brains (cf. Striedter, 2002). This is, in part, because a strictly hierarchical view of the brain, such as the triune brain concept adopted by Panksepp, is largely incorrect, as is the general idea of an orderly and progressive phylogenetic scale. The neocortex (more properly called isocortex; cf. Striedter, 2005) is not a crown that sits atop a more ancient and preserved subcortex like icing on an already baked cake (also see Footnote 1). As a consequence, the rat brain is a not a blueprint for understanding the mammalian brain, which itself is a "highly generalized abstraction" (Striedter, 2005, p. 91).

In his book *The Principles of Brain Evolution*, Striedter observes that brains are like companies—they must reorganize as they increase in size in order to stay functional (2005, p. 127). As a consequence, there is a tension between emphasizing evolutionary continuities and homologies on the one hand and species differences on the other. If you search for similarities in mammalian brains, you can certainly find them. But if you search for differences, you can find them as well. The general conclusion is that species differences in brain size, structure, and organization (including connectivity between areas) are of functional consequence and therefore should not be dismissed. This is particularly important when considering the human brain, which has undergone considerable reorganization when compared with other mammals (such as rats), when compared with the first primates, and even when compared with our closest biological relative, the chimpanzee.

There are three observations about brain evolution (summarized from Striedter, 2005) that should be considered when evaluating the hypothesis of core emotional systems (as conceived by Panksepp) or basic emotions (as conceived by Izard). First, size matters. Striedter and others cited in his book speculate that absolute brain size has huge functional consequences that were probably selected. Living in large social groups is the major adaptive advantage of humans. Larger brains allowed for more sophisticated social interaction that is involved with, among other things, foraging and storing food and negotiating conflict and aggression with conspecifics. This hypothesis is supported by the observation that both isocortical size and the size of the basolateral nucleus of the amygdala correlate positively with social group size (Barton & Aggleton, 2000; for a discussion, see Barrett, 2006a). Larger human brains also have many changes that allow for language, which then allowed major and accelerated changes in behavior as well as a multitude of mental states (for a discussion of the role of language in emotion, see Barrett, 2006b; Barrett, Lindquist, & Gendron, 2007). Much of the increase in human brain size is accounted for by rapid growth after birth. Most of the change in size is due to the addition of white matter (see also Schoenemann, Sheehan, & Glotzer, 2005; for a review, see Schoenemann, 2006; but see Schenker, Desgouttes, & Semendeferi, 2005), meaning that

connectivity between areas within the human brain can be molded by experience or epigenetic influences. In fact, there are large individual differences in both cortical volumes and connectivity, the functional significance of which (especially for emotion) are only beginning to be explored.

Not surprisingly, the second observation is that connectivity matters. Differences in the presence and magnitude of neuronal connectivity are crucially important to the behavioral differences that can be observed between different species such as rats and humans. Of particular importance is the observation that the human isocortex projects directly to and throughout the brain stem and spinal cord, especially to the ventral horn where motor neurons are (rats, in comparison, have fewer such projections). For example, the periaqueductal gray (PAG), which receives inputs from the hypothalamus, also receives direct inputs from areas of the prefrontal cortex in primates, including humans (An, Bandler, Ongur, & Price, 1998; Ongur & Price, 2000). The ventromedial prefrontal cortex (vmPFC; including subgenual and pregenual portions of the anterior cingulate cortex [ACC] and the medial surface of the most posterior parts of Brodmann Area [BA] 10) projects directly to the hypothalamus and brain stem in primates (Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003; Ongur & Price, 2000), and the areas within the entire orbital frontal sector project indirectly to the hypothalamus and brain stem via the amygdala and striatum (Amaral, Price, Pitkänen, & Carmichael, 1992; Carmichael & Price, 1995; Ghashghaei & Barbas, 2002; McDonald, 1998; Ongur & Price, 2000; Stefanacci & Amaral, 2002). As a result, humans (and other great apes) have greater direct and indirect cortical control over the subcortex and spinal cord than do rats, allowing greater autonomic and behavioral diversity and flexibility (and a decreased chance of fixed action patterns).[3]

Third, coordination matters. Regions that are anatomically interconnected evolve in concert, suggesting a functional connection that does not necessarily respect the cortex–subcortex distinction. For example, the basolateral nucleus of the amygdala and the prefrontal cortex covary in volume together across primate species, over and above what might be expected for body size (Barton & Aggleton, 2000; Barton, Aggleton, & Grenyer, 2003). Relative to what would be expected for a primate brain of human size, parts of the human orbitofrontal cortex (OFC; specifically, BA 13) are slightly smaller than expected, whereas the medial sector of the prefrontal cortex, including the ventral and dorsal medial prefrontal cortex (specifically, BA 10) and ACC, is slightly larger (Schenker et al., 2005; Semendeferi, Armstrong, Schleicher, Zilles, & Van Hoesen, 1998, 2001). The functional significance of these changes is not well understood, although presumably they would be affective in nature, given the role that these cortical areas play in producing affective

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[3]Furthermore, the fact that the olfactory bulb is smaller and less complex in humans (in comparison with rats) suggests that components of the limbic system became functionally uncoupled from one another as primates evolved.

behavior and autonomic function (Ongur, Ferry, & Price, 2003; Ongur & Price, 2000). Nonetheless, it has been suggested that richer connectivity between cortical areas, rather than increases in cortical volumes per se, accounts for the diversity and flexibility of human behavior (cf. Rilling, 2006; Semendeferi, Lul, Schenker, & Damasio, 2002), and rats certainly do not show this degree of interconnectivity.[4]

Taken together, all three observations suggest that cortical areas play an intrinsic role in the production of behavior and mental states in humans to a greater extent than is true in rats. As the isocortex became larger and more interconnected with other brain regions, it became more capable of influencing the activity in those regions. This does not necessarily mean that cognition regulates emotion, or even that cortex regulates emotion (as we discuss below). Instead, it means that the cortex has become intrinsically important to normally functioning behavior in humans. Given these changes, can we be certain that the putative "core emotional systems," if they do exist in the rat brain, function similarly in the human brain? The answer is probably no. And if the answer is no, then animal models represent a viable epistemological strategy for understanding something about human emotion life, but only up to a certain point. We are not arguing that animal models of behavior are valueless. On the contrary, they give us part of the story for understanding human emotional life.

To be clear, we are not arguing that emotions live in the cortex. Rather, we are emphasizing that great apes (including humans) have dense interconnectivity between subcortical and cortical areas and among cortical areas, the functional consequences of which should not be ignored. Damage to the subcortex is not only disastrous to emotional life, as Panksepp points out (2007, p. 290), it is disastrous to conscious life more generally. What Panksepp overlooks is that cortical damage is more disastrous for human psychological function than for any other mammalian species (except perhaps other great apes; for a discussion, see Striedter, 2005). This observation does not mean that functions have been transferred to the cortex (in fact, such functional encephalization is unlikely; again, see Striedter, 2005). Rather, it suggests that the cortex is necessarily part of a larger, distributed circuitry (i.e., part of the neural reference space) that produces behaviors and mental states (we come back to this point again in the final section of the article).

We believe that relying on the triune brain concept, or on any hierarchical brain concept that is grounded in the idea of an orderly and progressive phylogenetic scale as Panksepp and Izard do, can result in conceptual confusions in psychological theorizing. The amygdala is not the seat of emotion (nor the seat of fear, nor even the seat of affect per se), and the cortex is not the seat of conceptual processing (as claimed by Izard, 2007, p. 269). Cortical brain areas (such as the OFC or vmPFC) may regulate neural activity in the amygdala, but this does not mean that cognition or conceptual processing regulates emotions that live in the more primitive and "animalistic" part of the human brain.

From Emotional Behaviors to Emotions

Even if scientists someday discover that emotional behaviors are produced by architecturally or chemically distinct circuits (as opposed to flexible neuronal assemblies) that are hard coded into the subcortex of mammalian brain at birth (with minimal shaping by prior experience), and even if those circuits function in exactly the same way in a human (or great ape) brain as in other mammals (which is unlikely), would this constitute evidence that discrete emotions are produced by these circuits? That is, would this constitute evidence that anger, sadness, fear, and the like are each natural kinds (where instances of each kind are defined by homology or a common cause such as said brain circuits)? Panksepp (and presumably Izard) answer yes, but we would answer no. A circuit that produces a behavior is just that—it is not a circuit that produces a broad and complex psychological category.[5] To presume so is either a form of ontological reductionism (by redefining the psychological events we call *emotion* as the mere performance of certain actions) or anthropomorphism (by presuming that nonhuman mammals experience complex states with mental content that empirically is associated with some kind of theory of mind).

Both assumptions are errors of great consequence in the science of emotion. Rats freeze when they hear a tone paired with a foot shock, so they are presumed to be in a state of fear (instead of surprise, anger, a general state of alarm, or merely a state that is conducive to reducing uncertainty) and undergoing "fear learning" (as opposed to learning that a particular array of sensory information predicts threat or some negative outcome; for a similar discussion of this point; see Kagan, in press). Scientists presume that a map of the neural circuitry of freezing behavior will yield a neural mechanism for fear that is largely preserved in humans, and a decade of neuroimaging studies have focused on locating a homologous neural circuit in the human brain. As Barrett (2006a) points out, however, freezing may be an innate, fixed action pattern (in some mammals) and may be part of the Western script for fear, but evidence regarding

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[4]There are additional changes that are surely of functional significance (discussed in Striedter, 2005). Primates have a greater number of premotor cortical areas, as well as areas of somatosensory cortex and parts of lateral prefrontal cortex and BA 39/40 in inferior parietal cortex (extending to the precuneus) that have no obvious homologues in nonprimates and most primates. Noncortical structures may have been simplified because some of their old functions were shifted to or subsumed by the cortex (although the idea of functional neocorticalization as a general principle in brain evolution is not likely correct). Even broadly conserved neurotransmitter systems exhibit variation. And of course, we have not even considered the possibility of evolutionary changes in cytoarchitecture, neuronal type or shape, and membrane functionality (or firing properties).

[5]In fact, it may be incorrect to refer to a physical action such as freezing as a "behavior" in the sense that even behaviors are complex psychological events that are perceived in the actions of others (e.g., Gilbert, 1998). Freezing may be better thought of as a fixed action pattern, but we leave this discussion for another time.

the circuitry that produces freezing behavior does not constitute evidence for a module for fear, unless you are willing (a priori) to define fear in narrow, behavioral terms. To do so would miss 95% of the instances that constitute the category of fear in humans.

THE CONCEPTUAL ACT MODEL IN BRIEF

Thus far, we have suggested that Barrett (2006a) did not focus on irrelevant correlational evidence that questions the natural-kind status of emotion while ignoring crucial causal evidence for the existence of natural kinds of emotion. In this section, we discuss Panksepp's and Izard's misconceptions of the conceptual act model, discussed briefly in Barrett (2006a) as an alternative to the natural-kind view.

The conceptual act model was fashioned as a solution to the emotion paradox (Barrett, 2006b): Studies that measure emotion by relying on human perception (subjective reports of feelings or judgments of other people's faces and bodies) typically produce consistent evidence for the categories that in English we call *anger*, *sadness*, and *fear*; but instrument-based measures of the brain, face, and body (what scientists might call objective measures) do not. Our solution is that emotion categories live at the level of human perception. Emotions are contents, not systems, in the brain.

The conceptual act model hypothesizes that the events called *anger*, *sadness*, or *fear* are emergent psychological events constructed from two more basic psychological processes: a psychologically primitive and biologically basic mammalian system that produces some variation on positive or negative states (called *core affect*) and a human conceptual system for emotion (i.e., what people know about emotion). Contrary to Izard's claim, the conceptual act model does not hypothesize that affective and conceptual processing proceeds in a linear sequence. In fact, using constraint-satisfaction logic, we have argued just the opposite (Barrett, Ochsner, & Gross, 2007).

According to Panksepp (2007), the conceptual act model is an "attributional–dimensional constructivist view of human emotions" that is "largely theoretical conjecture rather than a conclusion derived from robust neuroscientific data" (p. 281). Panksepp is correct that our model constitutes a set of hypotheses, rather than a theory populated by firm conclusions derived from experimental evidence. We believe this new model accounts for the existing empirical evidence better than does the natural-kind view, but of course, it awaits direct empirical test (as we have stated on many occasions).

According to Izard, the conceptual act model conflates the distinction between basic emotions (the "pure" emotions) and emotion schemas. But we have not conflated the two—we are actively questioning whether one (the psychological events people call by emotion words) can exist as we typically conceive of it without the other (conceptual knowledge for emotion). An analogy can be found in the experience of color. Color is a continuous spectrum of light at different wavelengths, but we see color and experience it in discrete categories. The embodiment

constraints that influence both which parts of the spectrum are seen and how the sensory information is transduced are not sufficient to explain why color is experienced and communicated categorically (i.e., why certain sets of wavelengths are experienced as qualitatively different and others as qualitatively similar). For that, you need the conceptual structure that is afforded by language (Davidoff, 2001; Steels & Belpaeme, 2005). We are suggesting something similar with respect to affect and the conceptual system for emotion.

Because people's ability to assimilate new ideas into an existing framework is so much more powerful than is the ability to accommodate that framework to new ideas, it is easy to misconstrue the conceptual act model using distinctions that are well-established in the emotion literature. A careful read of papers that discuss the model (Barrett, 2006b, 2006c; Barrett & Lindquist, in press; Barrett, Lindquist, & Gendron, 2007; Barrett, Mesquita, et al., 2007; Barrett, Ochsner, & Gross, 2007; Duncan & Barrett, in press; Lindquist & Barrett, in press) will reveal, however, that these distinctions do not apply to the conceptual act model.

The model is not attributional because the main psychological mechanism for transforming affect into an emotional episode is categorization (which can produce the mental contents called "attributions"; said another way, an attribution is not a process, it is a state). Furthermore, instead of assuming that people categorize an already existing affective reality after the fact, affective and conceptual processing are thought to shape one another via constraint satisfaction to produce an emergent phenomenon: an experience of anger, or an experience of someone else (even a rat) as angry.

The model is not strictly dimensional because it integrates both dimensional and categorical perspectives. The dimensional aspect can be found in the suggestion that all emotional events, at their core, are based in a psychologically primitive kind of affective response to events in the world as positive or negative, helpful or harmful (although the neural states that instantiate a pleasant or unpleasant affective state may be numerous and varied). The categorical aspect can be found in the suggestion that people automatically and effortlessly categorize the ebb and flow of core affect using conceptual knowledge for emotion. To categorize something is to render it meaningful: to determine what it is, why it is, and what to do with it. We can then make reasonable inferences about that thing, predict how to best act on it, and communicate our experience of the thing to others. In the conceptualization of emotion, categorizing core affect as anger (or as any other emotion) performs a kind of figure-ground segregation, so that the experience of an emotion will pop out as a separate event from the ebb and flow in ongoing core affect (in which core affect is associated with the direction and urgency of initial behavioral responses). In doing so, people divide ongoing changes in core affect into discrete, intensional, and meaningful experiences.

The conceptual act model is also not a social-constructivist model. In fact, the conceptual act model avoids the hopeless

distinction between evolution and social construction by suggesting that core affect and conceptualization processes are themselves given by nature (in that humans are born with the ability to have simple affective responses and quickly acquire perceptual categories that develop into a conceptual system that provides the grounding for perception), although the content that they represent is learned and may vary across individuals and cultures. The conceptual act model is not intuitive, but it is parsimonious: It relies on two well-established psychological processes with clear grounding in neuroscience to explain what emotions are and how they work.

There are elements of the conceptual act model that are consistent with certain points laid out by Panksepp and Izard. First, dimensional and categorical models can coexist. Second, any model of emotion must consider both species-general and species-specific processes. Third, evolution has shaped the psychological events we refer to as emotion in important ways. The first two points are obvious from the description above, but the last perhaps deserves special emphasis. The conceptual act model is grounded in an evolutionary approach. The evolutionary legacy to the newborn is not a set of modular emotion circuits that are hardwired into the subcortical features of the mammalian brain but may be, instead, a set of mechanisms that compute core affect and allow affective learning, as well as those that allow conceptual learning and categorization. The ability to categorize confers adaptive advantage, and so it is likely biologically preserved, even if the specific categories are not. The specific categories are more likely culture-sensitive solutions to common problems that derive from our major adaptive advantage as a species: living in complex social groups.

As a solution to the emotion paradox, the conceptual act model leaves scientists with the ironic suggestion that the natural-kind model is grounded in human experience. People experience fear and see it in others, so they assume there must be a literal (modular) neural circuit for fear in the mammalian brain. The conceptual act model is not a form of anthropocentrism (as claimed by Panksepp, 2007, p. 284); it is an argument against it. Our perceptions of the world do not reveal to us how the world works. To presume otherwise is an "error of arbitrary aggregation" (Lewontin, 2000) or "naive realism."

FINAL POINTS FOR CONSIDERATION

Before turning to our conclusions, we'd like to make a final set of observations that we hope will clarify future discussions about the nature of emotion. First, we believe that it is important not to confuse affect and emotion as psychological concepts. Many of the findings cited by Panksepp and Izard can be read as more consistent with the existence of core affect (hedonic tone or arousal) as opposed to discrete emotional states, either because that is what scientists actually measured (regardless of the terms used) or because they failed to measure responses that would allow for more specific conclusions over and above those related to valence or arousal (for discussions, see Barrett, 2006a, and Barrett, Lindquist, & Gendron, 2007).

Second, we believe it is important be clear about whether a citation is a conceptual analysis or an empirical one. For example, Panksepp cites Denton (2006) as providing evidence that discrete emotions emerged early in brain evolution, but Denton's book is a conceptual analysis for the existence of "primordial emotions" (i.e., subjective mental states that accompany disruptions of homeostasis, such as thirst, hunger, and the like). Denton does not cite any evolutionary evidence about anger, sadness, fear, or other so-called basic emotions and, in fact, provides no experimental evidence about emotions over and above an imaging study that has already been published (i.e., Damasio et al. 2000).

Third, we believe it is important to take care in ascribing your own view to others. For example, Izard cites Edelman (2006) as claiming that basic emotion expressions and feelings are produced by subcortical mechanisms, but our read of Edelman is somewhat different. Edelman argues that there are value systems (related to reward and punishment) that help to select from the many neural representations that are competing with one another at any given moment in time to instantiate a mental state (Edelman, 1987). Furthermore, in all of his work, Edelman argues against a strict representational model of the brain that is implied in the basic-emotion approach (i.e., that there is one neural representation for one mental content).

Fourth, we believe it is important to avoid making claims that have been already disconfirmed by published scientific evidence. Children do not easily recognize a variety of distinct emotional states in others (e.g., Russell & Widen, 2002). Congenitally blind infants (Fraiberg, 1977), children (Roch-Levecq, 2006), and adults (Galati, Schere, & Ricci-Bitti, 1997) produce only a limited number of the predicted facial behaviors when portraying emotion and almost never produce an entire configuration of facial action units—but then neither do sighted people (again, see Galati et al., 1997). Infants categorize faces with different perceptual features (e.g., closed vs. toothy smiles) as distinct even when they belong to the same emotion category (Caron, Caron, & Myers, 1985), and no studies can rule out the alternative explanation that infants are categorizing faces on the basis of the valence, intensity, or novelty of the facial configurations.[6] The capacity to discriminate among discrete feeling states is not invariant (Barrett, 1998, 2004; Feldman, 1995). Positron emission tomography and functional magnetic resonance imaging are equally suitable for studying emotion in the

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[6]For example, infants look longer at fear (or anger or sad) caricatures following habituation to happy caricatures, but this may reflect their ability to distinguish between faces of different valence (e.g., Bornstein & Arteberry, 2003). Similarly, infants look longer at a sad face following habituation to angry faces (or vice versa), but infants may be categorizing the faces in terms of arousal (e.g., Flom & Bahrick, 2007, Experiment 3). Many studies find that infants tend to show biased attention for fear faces (e.g., Bornstein & Arteberry, 2003), but this is likely driven by the fact that infants rarely see people making these facial configurations.

human brain, particularly when imaging brain stem and mid-brain areas (Wager et al., in press).

Fifth, we believe it is important to remember that, in the end, scientists stand on the shoulders of those who have come before, even when their scientific positions don't agree. Panksepp and Izard, along with others who hold to a natural-kind view of emotion, have made important and lasting contributions to the scientific study of emotion, and this will remain the case even if the model proves false. They fashioned a set of hypotheses that has guided the field for almost half a century and has produced much of the scientific evidence that can now be used to evaluate the model (cf. Barrett, 2006a). The idea that emotions are real biological entities rescued the topic of emotion from the ashes of behaviorism, inspiring the scientific study of emotion for several decades (cf. Ekman, 1992) and introducing experimental procedures that provided improved control over those that had been previously used. In his writings, Panksepp has drawn scientific attention to the fact that the events that people call "emotions" are contentful states and, perhaps more than anyone else, has emphasized the idea that nonhuman animals have feelings that give them some moral standing. Izard's research has demonstrated the important relational and regulatory consequences that come from perceiving emotion in others. Research by Ekman and others has shown that facial behaviors are unlikely to be emblems that are entirely culture specific and that there is some agreement in perception across cultures (even if, in the end, this agreement reflects something other than innate production mechanisms; cf. Barrett, 2006b). Research on appraisal models has contributed important observations about the contents that constitute emotional experience (cf. Barrett, Mesquita, et al., 2007) and describe the psychological situations within which emotions (as psychological events) take place (Ortony, Clore, & Collins, 1988), even if appraisals do not themselves constitute the processes that cause emotion. The evidence that emotion is important to categorization (Niedenthal, Halberstadt, & Innes-Ker, 1999), to risk assessments and other forms of decision making (Lerner, Gonzalez, Small, & Fischhoff, 2003; Lerner & Keltner, 2000, 2001; Lerner, Small, & Lowenstein, 2004), and to attitudes about out-group members or the ease of persuasion (DeSteno, Dasgupta, Bartlett, & Cajdric, 2004; DeSteno, Petty, Rucker, Wegener, & Braverman, 2004) is real and must be explained, even if emotions, as psychological events, are not natural kinds.

Finally, it is possible to build a theory of emotion (or of any other psychological event) that is grounded in nature without being a nativist. Every human thought, feeling, and behavior must be causally reduced to the firing of neurons in the human brain. Prior experience and learning are encoded in the human brain; even a strict constructivist approach must therefore have some grounding in nature. Yet a neuroscience approach to emotion need not make the modular assumption that distinct brain regions or circuits are dedicated to instantiating instances of psychological categories such as anger, sadness, and fear.

Rather, it might be more productive to work with the assumption that emotional phenomena are instantiated in widely distributed, interacting networks. So instead of asking "Where is the brain locus of anger?", we might ask "What are the networks that participate in the brain states that we experience as anger, or in seeing someone as angry?". In this regard, the concept of a "neural reference space" (Edelman & Tononi, 2000) is useful. A neural reference space is the neuronal workspace that implements the brain states that correspond to a class of mental contents (e.g., anger). Different brain states are implemented by flexible neuronal assemblies, so that a given neuron need not participate in every brain state within a class (e.g., in every instance of anger) or in the exact same mental state (e.g., the exact same instance of anger) at two points in time. According to Edelman's neural Darwinism view (1987; Edelman & Tononi, 2000), groups of neurons compete to instantiate a mental content at a given point in time, and only one is selected to do so. According to Spivey (2007), the human brain is never in a discrete mental state but rather can be described by a fuzzy logic that allows many different states at once (each with some probability). It may be that there are different networks within the neural reference space for emotion that are differentially recruited for constituting different mental contents (e.g., anger vs. sadness vs. fear), or it may be that the space is entirely flexible. Either way, the question becomes one of functional selectivity for affect and emotion rather than functional specialization per se.

CONCLUSION

Barrett (2006a) points out that the field of emotion research finds itself in what Greenwald (Greenwald & Ronis, 1981) calls a "disconfirmation dilemma." For every study that reports evidence that is consistent with the natural-kind view, there is more than one study that does not. Even taking into consideration measurement error, the natural-kind model does not account for the majority of the data. It is possible to come up with reasons for why scientists don't see the expected results in any single measure of emotion, the pattern of findings across studies is clear. Like Izard, some might argue that the natural-kind model doesn't account for all the evidence but that it still accounts for some. And this is true. And like Panksepp, some might argue that all we need are better designs, better methods, and better measures. And again, this might be true. It all comes down to how disconfirming evidence is considered.

Classical Newtonian physics also fits our experience of the physical world. If you push something, it speeds up. If you drop something, it falls down. Newtonian equations work to describe the physical world in the majority of cases. It was the small number of cases in which these equations did not work that ignited a paradigm shift that forced scientists to rewrite the laws of physics, first with the special and general theories of relativity (to describe the movement of large bodies like stars) and then with quantum mechanics (to describe the movement of small

bodies like electrons). So, the question for emotion researchers is this: How do we want to treat the disconfirming evidence in our field, which, unlike physics, is found in considerably more than a few isolated cases? Maybe it is time to take other hypotheses seriously. Barrett (2006a) was advocating that the science of emotion do just that.

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APPENDIX

PLAY/Joy

At the present time, there is very little evidence that "rough and tumble play" behaviors are associated with a coherent, functional PLAY circuit. "Rough and tumble play" is a term Panksepp uses to describe a set of action patterns common among juvenile rats, including pouncing on a partner's back (dorsal contacts), instances in which one animal ends up on its back with the other animal on top (pinning), running towards or away from a partner (darts), running over a partner (crossovers), and roughly pulling at a partner's fur (grooming; Panksepp, 1998). Panksepp has hypothesized that the PLAY circuit is constituted by specific neuroanatomical regions (parafasicular area [PFA], the ventral periaqueductal gray [vPAG], and dorsomedial thalamus [DMT]), neurotransmitter systems (norepinephrine and dopamine), and neuropeptides (endogenous opioids). No studies to date have specifically demonstrated that the proposed neuroanatomical areas, neurotransmitter systems, or neuropeptides are necessary to rough and tumble play, nor have any studies addressed how these components might interact to form a functional circuit. Electrolytic lesions in the PFA and DMT partially, but not completely, reduced play behavior (e.g., Siviy & Panksepp, 1985); these lesions also damage axons that happen to pass through a particular region, so that it remains unclear whether play behaviors are facilitated by the PFA and DMT specifically or by the adjacent areas that pass axons through these regions. More recent investigations suggest that these neural regions are unlikely candidates for instantiating play behavior. For example, an in-situ hybridization study that measured *c-fos* mRNA levels (as a measure of neural activity) found no increase in *c-fos* in either the DMT or vPAG of juvenile rats after they engaged in play behavior (Gordon, Kollack-Walker, Akil, & Panksepp, 2002).

RAGE/Anger

Scientists often classify types of aggressive behavior in terms of the provoking stimulus or the stimulus towards which the behavior is directed. For example, defensive aggression occurs when an animal attacks a predator, maternal aggression occurs when a mother attacks an intruder who threatens her babies, territorial aggression occurs when one animal enters the space of another, etc. (for a discussion, see Moyer, 1976, referenced in

Panksepp, 1998). More fine-grained behavioral descriptions can be grouped into the larger categories of defensive attack (when an animal vocalizes, bears teeth or claws, and bites to keep another animal from attacking), offensive attack (when an animal bites or delivers blows to the body of another animal to establish dominance or gain a resource such as territory or access to a female), play fighting, and predation, and these larger categories of attack behaviors (e.g., offensive and defensive) seem to be mediated by separable (but somewhat overlapping) neurobiology (Blanchard & Blanchard, 2003). Furthermore, much of the research on the neural circuitry of attack behaviors has been carried out in cats (e.g., for reviews, see Blanchard & Blanchard, 2003; Siegel & Shaikh, 1997), and it is not clear whether the circuitry for attack behaviors is the same in cats and rats (let alone humans).[7] Nonetheless, some generalities can be made (which by no means capture all the detail in this area of research), in that the hypothalamus, the dorsomedial tegmentum/midbrain, and the central grey/PAG areas do seem to participate in the instantiation in attack behaviors (e.g., Mos, Kruk, van der Pol, & Meelis, 1982; Mos et al., 1983; Roberts & Nagel, 1996). However, these generalities are not sufficient to describe how different types of attack behaviors are instantiated.

PANIC/Distress

Infant rats produce 40-kHz vocalizations when separated from their mothers, but it is not clear that the brain areas supporting 40-kHz vocalizations constitute a PANIC circuit or that the infant animals are "crying" and experiencing psychological distress when they produce these behaviors, as claimed by Panksepp (1998, 2000, 2005).[8] Experimental evidence indicates that the ACC; the ventral septum; bed nucleus of the stria terminalis (BNST); dorsal preoptic area; DMT; and the dorsolateral, lateral, and caudoventral aspects of the PAG, along with other downstream brain stem regions that are involved in excitation of the thorax, constitute a pathway that supports a range of vocalizations in mammals, not just those that are negative (see Dujardin & Jurgens, 2005, 2006; Jürgens, 2002). Moreover,

40-kHz vocalizations might not even express psychological distress or serve the communicative function of calling the rat mother for help (for a discussion, see Blumberg & Sokoloff, 2001; but see also Hofer, 2002). Experimental evidence also suggests the alternative hypothesis that 40-KHz vocalizations are actually the byproduct of a more basic biomechanical process (thermoregulation) that is called into play when a pup is physically separated from its mother (e.g., Blumberg, Sokoloff, & Kent, 2000; Blumberg, Sokoloff, Kirby, & Kent, 2000; Kirby & Blumberg, 1998).

SEEKING/Expectancy

It is now well-known that appetitive behaviors are supported by the mesolimbic dopamine system. This pathway begins in the ventral tegmental area, which projects to the ventral striatum (including the nucleus accumbens [NAcc]). The ventral striatum in turn projects information to the lateral hypothalamus (Groenewegen, Wright, Beijur, & Voorn, 1999) and midbrain and brain stem structures that influence autonomic and endocrine function (Parvizi & Damasio, 2001); to other areas of the basal ganglia that are associated with voluntary motor actions (Graybiel, 1998); to the central nucleus of amygdala, which directs attention (Holland & Gallagher, 1999); and to the ventromedial prefrontal cortex, which is thought to influence value-guided choice behavior (Ongur & Price, 2000). This system is not specific for positive, rewarding stimuli, however, nor does it necessarily engender an experience of pleasant excitement that is associated with anticipation of a reward, as suggested by Panksepp (1998, 2000). The mesolimbic dopamine system is involved in directing attention to and modulating behavioral responses to a range of aversive, novel, and appetitive stimuli (Grillner, Hellgren, Menard, & Saitoh, 2005). The firing rate of individual neurons in the NAcc, for example, increases when animals taste both unpleasant (quinine) and pleasant (sucrose) liquids (Roitman, Wheeler, & Carelli, 2005). Both behavioral approach and withdrawal are facilitated via electrical stimulation of the rostral and caudal shells of the NAcc (Reynolds & Berridge, 2001, 2002, 2003), and approach behaviors become dopamine independent with overtraining (Choi, Balsam, & Horvitz, 2005). Findings such as these have led some researchers to suggest that the mesolimbic dopamine system appears to be involved in gating attention to novel, salient, or unexpected environmental events that require an effortful (usually behavioral) response (e.g., Berridge & Robinson, 1998; Horvitz, 2000, 2002; Salamone, Correa, Farrar, & Mingote, 2007; Salamone, Correa, Mingote, & Weber, 2005; Schultz, Apicella, & Ljungberg, 1993; Wise, 2005). This view is supported by research demonstrating that dopamine neurons increase their firing rates when surprising or unexpected appetitive events are presented (McCullough & Salamone, 1992) but do not increase their firing rates when appetitive events are predictable (Mirenowicz & Schultz, 1994). Although

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[7]For example, although PAG lesions block attack behaviors as a result of hypothalamic stimulation in cats, the same lesions only mildly and temporarily decrease the presence of attack behaviors in rats (Mos et al., 1983). These findings suggest that after PAG lesioning has occurred, alternative neuronal assemblies may come online to instantiate attack behaviors in rats, but not in cats.

[8]According to Panksepp (1998), "distress/panic" calls are only made by infant rats and occur following social isolation (they are essentially "crying" for one's mother). According to others (e.g., Blumberg & Alperts, 1990; Kehoe, 1988), the range for these types of calls is anywhere from 30 to 50 kHz. Adult rats produce vocalizations in the range of 20 to 70 kHz (Sewell, 1967) and produce 22-kHz vocalizations not only in unpleasant, highly arousing situations like attack by predators, foot shock, or social defeat by another animal (see Borta, Wöhr, & Schwarting, 2006) but also during copulation (Barfield, Auerbach, Geyer, & McIntosh 1979). The frequency of vocalizations that an animal produces is in part determined by the animal's body size (in general, the larger an animal, the lower the frequency they are capable of producing; see Blumberg & Sokoloff, 2001). Thus, animals may not be voluntarily producing different frequencies to communicate different things, or if they are, they're doing so within a fixed ultrasonic range.

we would not rule out the possibility that this circuit is involved in a basic affective or motivational state, we do question the scientific validity of referring to this state as a basic or discrete emotion.

FEAR/Anxiety

Literally hundreds of studies have been conducted to examine the circuitry in the rat brain that produces the fixed action patterns that occur in threatening situations (what scientists often call fear behaviors). Rats often (but not always) freeze in response to a potential threat (like a predator or foot shock), and it is clear that the circuitry to support this behavior includes the amygdala, BNST, dorsolateral PAG, and vPAG (e.g., Fendt, Siegl, & Steiniger-Brach, 2005; LeDoux, 2000; Vianna, Landeira-Fernandez, & Brandao, 2004). It is far from clear, however, that freezing is actually indicative of the discrete emotion that people refer to as fear. The evidence is also consistent with the interpretation that freezing is an alert behavioral stance that allows an organism to martial all its attentional and sensory resources to quickly learn more about a stimulus when its predictive value is uncertain. In this view, the amygdala is not the brain locus of fear, nor is it the locus of negative affect, nor is its primary function affective per se. Rather, the amygdala might be thought of as a structure that tags a sensory representation when its predictive value is uncertain and creates a behavioral stance that prioritizes additional processing to allow the organism to better learn whether this sensory pattern (i.e., the stimulus) predicts a threat or a reward. This interpretation is not only consistent with the neuroscience work on classical conditioning (in our view mistakenly called "fear" conditioning), but it is also consistent with the research showing that the amygdala is selectively engaged by novel stimuli (e.g., Wright et al., 2003; Wright, Wedig, Williams, Rauch, & Albert, 2006) and that, in humans, the amygdala habituates quickly to a stimulus once its predictive value is known (e.g., Fischer et al., 2003).

CARE/Nurturance and Love, and LUST/Sexuality

Maternal behaviors such as pup retrieval, grooming, nest building, and nursing are clearly supported by neural circuitry involving the medial preoptic area (MPOA), the ventral BNST, the VTA, the NAcc, the ventral pallidum, the anterior hypothalamic nucleus in the preoptic area of the hypothalamus, the PAG, and the habenula in the pineal body (for reviews, see Numan, 2006, 2007; Numan & Insel, 2003). The MPOA and adjacent BNST are perhaps the most integral aspects of the circuitry, as they serve as gating mechanisms that allow normally pup-avoidant females to suppress neophobic responses (that would cause them to eat their pups) and express maternal behavior (see Lonstein & Morell, 2007; Numan, 2006, 2007).

Circuitry specific to fixed action patterns associated with sexual motivation like copulation (in male rats) and lordosis (in female rats) includes brain regions that detect and process sexual stimuli (such as the medial nucleus of the amygdala and BNST), which in turn project to central, hormonally-mediated regions of control. In males, MPOA is considered a central integrative site for the regulation of male sexual behavior (Dominguez & Hull, 2005). The ventromedial hypothalamus serves a similar role in females (Pfaff & Sakamura, 1979a, 1979b). Both the MPOA and the ventromedial hypothalamus project to the PAG, which in turn projects to motor regions controlling ejaculation or lordosis (e.g., Marson, 2004; Marson & Murphy, 2006). Dorsomedial/lateral PAG appears to be involved in male ejaculation, whereas ventrolateral/lateral PAG appears to support lordosis (see De Vries & Simerly, 2002; Marson & Murphy, 2006; Murphy & Hoffman, 2001).

Although maternal and sexual behaviors are caused by well-mapped brain circuits, there is little conclusive scientific evidence at present that nonhuman mammals feel anything specific beyond a basic affective or motivational state (pleasant or unpleasant, aroused or sleepy, or states that alternatively can be characterized as approach or avoidance) during the expression of these behaviors. Females sometimes show conditioned place preference for areas where they previously copulated (although this only occurs when females control or "pace" the sexual stimulation they receive; see Paredes & Vasquez, 1999, for a review) and dams will perform operant behaviors to retrieve their own pups (e.g., Lee, Clancy, & Fleming, 2000), but this is not, in and of itself, evidence that rats are experiencing lust or love.