Running Head: CORRECTIONS TO PANKSEPP (2008)

Corrections to Panksepp (2008)

Lisa Feldman Barrett [1, 2]

and

Kristen A. Lindquist [1]

1. Boston College

2. Massachusetts General Hospital/Harvard Medical School

Address correspondence to:

Lisa Feldman Barrett
Boston College
Department of Psychology
427 McGuinn Hall
Chestnut Hill, MA 02167

Email: barretli@bc.edu
Phone: (617) 552-1541
Fax: (617) 552-0523

Debates about scientific ideas are valuable. They help clarify positions, bring new evidence to light, inspire discourse, and have the potential to move a field forward in the process. At the frontiers of science, nothing speeds scientific progress like the clash of competing viewpoints. A debate can only achieve its potential, however, if everyone is careful to present their own views consistently and represent their opponents' views accurately. In this brief report, we address inaccuracies in Panksepp's (2008) response to our 2007 paper entitled "*Of mice and men: Natural kinds in the mammalian brain*?" (that was, in turn, a response to Panksepp's earlier 2007 critique of the Barrett 2006 paper entitled "*Emotions as natural kinds*?"). While we recognize (and want to avoid) the potential for infinite regress, we think it is important to set the record straight on certain essential points, particularly those where our scholarship has been questioned. In this vein, we briefly outline and address Panksepp's criticisms below (using direct quotes where necessary).

1. *Panksepp (2008) writes "Barrett incorrectly suggests that Valenstein's intriguing findings contradict my position"* (p. 305). Panksepp's view is that stimulating specific brain areas will specifically and consistently produce specific emotions. Panksepp (2007) wrote "Can one evoke such emotion patterns using ESB, in homologous brain regions, across species? The answer is yes" (p. 286). In reviewing the experimental literature on electrical brain stimulation, it is clear that Valenstein's (1973) answer is "no." In summarizing the literature on electrical brain stimulation, Valenstein (p. 88) wrote, "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" (for the full quote, see

Barrett et al., 2007, p. 300). Electrically stimulating specific neurons in the brain produces variable results, both in humans and in nonhuman primates.

2. *Panksepp (2008) writes, "Barrett asserts that dopamine does not consistently mediate positive affect"* (p. 305). In Barrett et al (2007), we noted that dopamine is neither necessary nor specific to representing positive, rewarding stimuli. Nor does it specifically engender an experience of pleasant excitement that is associated with anticipation of a reward. There is mounting evidence (only a handful of which is cited in Barrett et al., 2007) demonstrating that the mesolimbic dopamine system is involved in directing attention to and modulating behavioral responses to a range of aversive, novel, as well as appetitive stimuli. The same neurons within the ventral striatum can code for aversive or appetitive stimuli, depending on the context (Reynolds & Berridge, 2008).

3. *Panksepp (2008) writes "Barrett suggests I advocate an 'orderly and progressive phylogentic [sic] scale' (p. 303). I adhere to no 19th century conceptions of linear, hierarchical evolution"* (p. 305). Panksepp's writings on emotion utilize MacLean's triune-brain concept. The triune brain concept presupposes an orderly and progressive phylogenetic scale. Even as late as 1993, MacLean depicts brain evolution as a "phylogenetic hierarchy" that looks like layers of an onion: a "reptilian brain" sheathed by a "paeleomammalian" formation, which itself is sheathed by a "neomammalian" formation (MacLean, 1993, p. 67). We point out in Barrett et al. (2007) that most contemporary neuroscientists have foregone this older model because it does not adequately capture the realities of brain evolution (e.g., Striedter, 2002, 2005).

4. *Panksepp (2008) writes "Barrett suggests I advocate modular views of brain organization. I do not"* (p. 305). In the many review papers that he has written over the past twenty years, Panksepp has repeatedly subscribed to the idea that certain kinds of emotion correspond to architecturally separable subcortical circuits that can function without cortical input. Each circuit, once triggered, is said to cause a reflexive, stereotyped pattern of feeling, bodily state, and action. This idea is consistent with both psychological and biological descriptions of modularity (see Shilling, 2002 for descriptions of modularity across scientific disciplines). This being said, Panksepp does not deny the possibility that his putative emotion circuits can be influenced via projections from the cortex, and we never meant to suggest otherwise.

5a. *Panksepp (2008) claims that he did not cite Denton (2006) as evidence for his approach to emotion.* In the last draft we saw of Panksepp's 2007 commentary before our own paper went to press, Panksepp cited Denton (2006) several times as evidence for basic emotion systems in brain. For example on page 4 of that draft, Panksepp wrote "Here I argue that Barrett needs to consider *causal* evidence for various basic emotional systems derived from direct brain stimulation experiments in a variety of mammals, including humans (Denton 2006; Panksepp, 1982)" and on page 8 he wrote "The cross-species affective neuroscience view provides abundant evidence 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, 2000a)." On page 17, he also referred to Denton (2006) as providing evidence about "basic motivational or *homeostatic affects* (Denton, 2006)" (italics in the original) and on page 36 he wrote "See Denton, 2006, p. 7 who

has provided abundant information for the localization of basic homeostatic affects in subcortical regions of the human brain." Any apparent errors we made regarding Panksepp's reference to Denton are the result final editorial changes to the Panksepp paper that we were not privy to at the time our paper went to print.

5b. *Panksepp then goes on to make a completely different point, in saying that we "ignored evidence for multiple, subcortically engendered affective processes,"* (p. 306). Barrett et al. (2007) did not ignore evidence. We simply showed that the existing data routinely cited by Panksepp as evidence for his views are not sufficient to make confident claims about the existence of "multiple, subcortically engendered affective processes," by which he means basic emotion circuits.

6a. *Panksepp (2008) states that we "reassert that brain stimulation-evoked emotional states are just behaviors and nothing more"* (p. 306). In Barrett et al. 2007, we made the broader philosophical point that linking a brain area to a specific behavior is not, in and of itself, evidence that the area produces a phenomenological experience.

6b. *Panksepp (2008) then states that we sidestepped "a massive database showing that animals uniformly approach and avoid such subcortical brain stimulations,"* (p. 306). To our knowledge, we have never claimed that deep brain stimulation fails to elicit approach and avoidance states. Rather, in Barrett et al. (2007), we argued that given the pattern of research findings, such states are better considered as evidence of a general affect response than of specific emotions per se (because, in our scientific vocabulary, the words "affect" and "emotion" refer to different psychological phenomena). Furthermore, we noted that deep brain stimulation does not elicit specific behaviors with the degree of consistency that Panksepp repeatedly claims.

6c. *Panksepp (2008) goes on to say that we ignored the fact that deep brain stimulation of "subcortical circuits" in humans "yields diverse affects consistent with animal behavioral observations"* (p. 306). We did not claim that the human data disagreed with the non-human animal data. Rather, we pointed out that neither the human nor the non-human animal data support Panksepp's claim (2007) that deep brain stimulation provides clear evidence for the natural kind view of view of emotion.

6d. *Finally, Panksepp (2008) cites a recent paper (Blomstedt et al., 2008) as evidence that deep brain stimulation evokes "sadness/depression" in humans* (p. 306). Blomstedt et al. (2008) do indeed report evidence that stimulation of the subthalamic nucleus (STN) in the basal ganglia elicits crying and reports of acute depression in a single individual. This finding is neither specific nor consistent enough across studies to claim that this area causes depression, however. STN stimulation is widely used to increase mobility in Parkinson's disease (e.g., Limousin et al, 1995; Bejjani et al., 2000) and only a few published studies cite evidence of depression-like behavior upon STN stimulation. Even within these reports, only 5-10% of patients included actually report depression or show depression-like behavior upon stimulation (see Bejjanni et al. 1999, Doshi et al., 2002). Moreover, several other studies (Kulichevsky et al., 2002, Romito et al., 2002) have found evidence for mania upon stimulation of the STN in patients with Parkinson's disease, drawing into question the idea that the STN is specifically linked to depression or even to unpleasant affect.

7. *In the appendix of his paper, Panksepp wrote "Barrett et al. (2007) distorted my empirical contributions and theoretical positions in their extended appendix,"* (p. 307). The appendix that appears in Barrett et al. (2007) was meant to be a review of the

empirical literature that Panksepp often makes reference to as support for his model of emotion. It was not meant as a review of his own specific empirical contributions. We explained as much when we introduced the appendix on page 299 of our paper, saying "…many of the causal experiments cited as evidence for the existence of modular brain circuits for emotion are open to alternative explanations," (Barrett et al., 2007). Nonetheless, Panksepp (2008) makes the serious claim that our literature review was incorrect on a number of counts. In Table 1, we provide an accounting these claims. Many of the criticisms offered by Panksepp (2008) are a matter of opinion or point of view, and have already been extensively addressed in Barrett et al. (2007). In Table 1, then, we address only those points where Panksepp claims we made a factual error.

Conclusions

Panksepp (2008) was right about two things. First, in *Emotions as natural kinds*? (Barrett, 2006) and *Of mice and men: Natural kinds in the mammalian brain*? (Barrett et al. 2007), we did not lay out our own model of emotion in any detail, nor did we propose a neurobiological program to test it. As we explained in Barrett et al. (2007) (and repeat here), our goal in these papers was to examine the validity of natural kind approaches to emotion, and not to craft a soapbox from which to broadcast our own view. Our model is laid out in detail across a series of peer-reviewed papers published from 2006 onward (Barrett, 2006b, in press; Barrett & Bliss-Moreau, in press; Barrett, Mesquita, Ochsner, & Gross, 2007; Barrett, Lindquist, & Gendron, 2007; Barrett, Ochsner, & Gross, 2007; Duncan & Barrett, 2007; Lindquist & Barrett, 2008).

Second, for the past twenty years, Panksepp has indeed offered "testable strategies for decoding how various emotional affects are engendered in mammalian brains" (p. 306, Panksepp, 2008). But offering a strategy, no matter how compelling it is, is not equivalent to offering solid data to support your hypotheses. In our 2007 paper, we outline the reasons why, in our view, Panksepp has not yet delivered the evidence necessary to treat his model as fact.

In the end, Panksepp believes that we have not fully appreciated his model of emotion. We do, in fact, very much appreciate his views. We just don't agree with them. And, even though we do not agree with them, we do not question Panksepp's contribution to the field of emotion.

In the end, however, it does not matter how well a scientist tells a story, or how many times that story is repeated in print. Eventually, the data point scientists in the right direction. And so far, the diversity of empirical evidence, along with work on brain evolution, seem to indicate that emotions are not natural kinds.

References

Barrett, L.F. (2006a). Emotions as natural kinds? *Perspectives on Psychological Science, 1,* 28–58.

Barrett, L. F. (2006b). Solving the emotion paradox: Categorization and the experience of emotion. *Personality and Social Psychology Review, 10,* 20-46.

Barrett, L. F. (in press). The future of psychology: Forward into the past. *Perspectives in Psychological Science.*

Barrett, L.F. & Bliss-Moreau, E. (in press). Affect as a psychological primitive. *Advances in Experimental Social Psychology.*

Barrett, L.F., Lindquist, K.A., Bliss-Moreau, E., Duncan, S., Gendron, M., Mize, J., & Brennan, L. (2007). Of mice and men: Natural kinds of emotions in the mammalian brain? A response to Panksepp & Izard. *Perspectives on Psychological Science, 2,* 297–312.

Barrett, L. F., Lindquist, K., & Gendron, M. (2007). Language as a context for emotion perception. *Trends in Cognitive Sciences. 11,* 327-332.

Barrett, L. F., Mesquita, B., Ochsner, K. N., & Gross, J. J. (2007). The experience of emotion. *Annual Review of Psychology*, *58,* 373-403.

Bejjani, B., Damier, P., Arnulf, I., Thivard, L., Bonnet, A., Dormont, D., et al, (1999). Transient acute depression induced by high-frequency deep-brain stimulation. *New England Journal of Medicine, 340,* 1476-1480.

Bejjani, B., Dormont, D., Pidoux, B., Yelnik, J., Damier, P., Arnulf, I., et al. (2000). Bilateral subthalamic stimulation for Parkinson's disease by using three-dimensional stereotactic magnetic resonance imaging and electrophysiological guidance. *Journal of Neurosurgery, 92,* 615–625.

Berridge, K. C., & Robinson, T. E. (2003). Parsing reward. *Trends in Neurosciences, 9*, 507–513.

Blomstedt, P., Hariz, M.I., Lees, A., Silberstein, P. Limousin, P., Yelnik, J. & Agid, Y. (2008). Case report: Acute severe depression induced by intraoperative stimulation of the substantia nigra: A case report. *Parkinsonism and Related Disorders*, *14*, 253–256.

Blumberg, M.S., & Sokoloff, G. (2001). Do infant rats cry? *Psychological Review, 108,* 83–95.

Denton, D. (2006). *The primordial emotions: The dawning of consciousness*. New York: Oxford University Press.

Doshi, P.K., Chhaya, N, and Bhatt, H.M. (2002). Depression leading to attempted suicide after bilateral subthalamic nucleus stimulation for Parkinson's Disease. *Movement Disorders,* 5, 1084–1100.

Duncan, S., & Barrett, L. F. (2007). Affect as a form of cognition: A neurobiological analysis. *Cognition and Emotion 21,* 1184-1211.

Gordon, N.S., Kollack-Walker, S., Akil, H., & Panksepp, J. (2002). Expression of c-fos gene activation during rough and tumble play in juvenile rats. *Brain Research Bulletin, 57*, 651–659.

Kulisevsky, J., Berthier, M. L. Gironell, A., Pascual-Sedano, B., Molet, J. and Parés, P. (2002). Mania following deep brain stimulation for Parkinson's disease. *Neurology*, *59*, 1421-1424

Limousin, P., Pollack, P., Benazzouz, A., Hoffman, D., Le-Bas, J., Broussolle, E., Perrett, J., & Benabid, J. (1995). Effect on parkinsonian signs and symptoms of bilateral subthalamic nucleus stimulation. *The Lancet, 345,* 91-95.

Lindquist, K.A., & Barrett, L.F. (2008). Constructing emotion: The experience of fear as a conceptual act. *Psychological Science, 19,* 898-903.

MacLean, P. D. (1993). Cerebral evolution of emotion. In M. Lewis, & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 67–83). New York: Guilford Press

Mos, J., Lammers, J.H.C.M., van der Poel, A.M., Bermond, B., Meelis, W., & Kruk, M.R. (1983). Effects of midbrain central gray lesions on spontaneous and electrically induced aggression in the rat. *Aggressive Behavior, 9*, 133–155.

Panksepp, J. (1982). Toward a general psychobiological theory of emotions. *The Behavioral and Brain Sciences*, 5, 407-467.

Panksepp, J. (1986). The neurochemistry of behavior. *Annual Review of Psychology, 37,* 77-107.

Panksepp, J. (1998). *Affective neuroscience. The foundations of human and animal emotions.* New York: Oxford University Press.

Panksepp, J. (2000a). Affective consciousness and the instinctual motor system: the neural sources of sadness and joy. In: Ellis R, Newton N, (Eds.), *The Caldron of Consciousness: Motivation, Affect and Self-organization, Advances in Consciousness Research.* (pp. 27-54) Amsterdam: John Benjamins Publishing Company.

Panksepp, J. (2000b). Emotions as natural kinds within the mammalian brain. In M. Lewis & J. Haviland (Eds.), *Handbook of emotions* (2nd ed., pp. 87–107). New York: Guilford Press.

Panksepp, J. (2003). Can anthropomorphic analyses of separation cries in other animals inform us about the emotional nature of social loss in humans?: Comment on Blumberg and Sokoloff (2001). *Psychological Review, 110,* 376–388.

Panksepp, J. (2005). Affective consciousness: Core emotional feelings in animals and humans. *Consciousness and Cognition 14,* 30–80.

Panksepp, J. (2007). Neurologizing the psychology of affects how appraisal-based constructivism and basic emotion theory can coexist. *Perspectives on Psychological Science, 2,* 281-296.

Panksepp, J. (2008). Cognitive conceptualism—Where have all the affects gone?: Additional corrections for Barrett et al. (2007). *Perspectives in Psychological Science, 3,* 305-308.

Panksepp, J., Newman, J.D., & Insel, T.R. (1992). Critical conceptual issues in the analysis of separation distress systems of the brain. In K.T. Strongman (Ed.), *International review of studies on emotion* (Vol. 2, pp. 51–72). Hoboken, NJ: John Wiley & Sons.

Reynolds, S.M., & Berridge, K.C. (2008). Emotional environments retune the valence of appetitive versus fearful functions in nucleus accumbens. *Nature Neuroscience*, 11(4), 423-425.

Romito, L.M,., Raja, M., Daniele, A, Contarino, MF, Bentivoglio, A.R, Barbier, A, Scerrati, M, & Albanese, A. (2002). Transient Mania with Hypersexuality After Surgery for High-Frequency Stimulation of the Subthalamic Nucleus in Parkinson's Disease. *Movement Disorders, 17,* 1371-1374.

Schilling, M.A. 2002. Modularity in multiple disciplines. In Garud, R., Langlois, R., & Kumaraswamy, A. (Eds), *Managing in the Modular Age: Architectures, Networks and Organizations* (pp. 203-214). Oxford, England: Blackwell Publishers.

Siviy, S.M., & Panksepp, J. (1985). Sensory modulation of juvenile play in rats. *Developmental Psychobiology, 20*, 39–55.

Striedter, G.F. (2002). Brain homology and function: An uneasy alliance. *Brain Research Bulletin, 57*, 239–242.

Striedter, G.F. (2005). *Principles of brain evolution.* Sutherland, MA: Sinauer Associates.

Valenstein, E.S. (1973). *Brain control.* New York: Wiley.

Table 1

Correcting Additional Errors in Panksepp (2008)

1. "*Contrary to Barrett's claim, I have never made the ventral periaqueductal grey matter a definitive part of the PLAY network and cFos protein translation, though perhaps not transcription, is elevated in the dorsomedial thalamic parafascicular complex of rats following play*" (p. 307). In 2000, Panksepp wrote "The most definitive brain evidence is that the parafasicular area integrates somatosensory signals that instigate play, and that essential components probably lie in the PAG – probably the ventrorostral areas…" (Panksepp, 2000, p. 149). In Barrett et al. (2007), we noted that the parafasciular area (PFA) and dorsomedial nucleus of the thalamus (DMT) are not *specifically* necessary for realizing play behaviors (although they might routinely play a role) and are therefore not necessary for a play circuit. Electrolytic lesions of the PFA and DMT partially, but not completely, reduced play behavior (e.g., Siviy & Panksepp, 1985). Furthermore, electrolytic 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 realizing 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 ventral PAG of juvenile rats after they engaged in play behavior (Gordon, Kollack-Walker, Akil, & Panksepp, 2002).
2. *"Barrett claims that 'it is not clear whether the circuitry for attack behaviors is the same in cats and rats (let alone humans)' (p. 311). In fact, the brainstem regions from which affective attack/defensive behaviors can be generated are essentially identical in all mammals that have been studied"* (p. 308). In Barrett et al. (2007) we noted that while 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). Such findings indicate that the circuitry for attack behaviors is (to some extent) different in rats and cats. Furthermore, as we noted in our 2007 paper, connectivity between the cortex and brainstem nuclei is highly elaborated in humans (when compared to rats and even when compared to other primates, indicating that the circuitry for any behavior may be different (to some extent) across species.

3. *"Our work was never based on the infant rat model, for reasons detailed elsewhere (Panksepp, Newman, & Insel, 1992)"* (p. 308).

In Barrett et al. (2007), we reviewed the infant rat literature in our review because this is the broadest and most well-established literature on infant animal vocalizations. Although Panksepp's own research focused on guinea pigs and chicks, he cites research with rat pups as evidence for the so-called distress calls that emanate from his putative panic circuit (see chapter 14 in Panksepp, 1998). He also cites such evidence in his 2003 critique of Blumberg and Sokoloff (2001), stating: *"*…manipulations have been done with nest and maternal cues, which do effectively modulate USVs in ways that strongly suggest these vocalizations reflect social processes in infant rats …and they suggest that the infant rat USV model does have considerable potential for understanding certain social dynamics that may be relevant for understanding anxiety-type emotional processes." (Panksepp, 2003, p. 308).

4. "As noted above, the SEEKING system has never been linked only to dopaminergic circuitry" (p. 308).

In numerous papers, Panksepp makes reference to the role of dopamine in seeking behavior. For example, in a 1986 paper he wrote: "In summary, the judgment of this writer is that the general function of DA activity in appetitive behavior is to promote the expression of motivational excitement and anticipatory eagerness—the heightened enegerization of animals searching for and expecting rewards…" (p. 91) Nearly a decade later, Panksepp reiterated: "I now conceptualize this psycho-ethological pattern of behaviors as arising from the SEEKING/Expectancy system—an energizing, hedonically positive functional system of the brain…which has been further developed into a dopamine-centered "wanting" or "incentive salience" model by Berridge and Robinson (2003)" (Panksepp, 2005, p. 46).

5. *"I never restricted my analysis of the FEAR system to freezing." "I have often noted that the amygdala is not an optimal location to get such effects"* (p. 308).

We did not claim that Panksepp restricted his analysis of fear to freezing, although (as we pointed out in Barrett et al., 2007), he does cite research on the neurobiology of freezing behavior as evidence for his putative fear circuit, including his own unpublished data (see chapter11, Panksepp, 1998). As with many behavioral neuroscience models of fear, the amygdala plays a central role Panksepp's putative fear circuit.