The Brain Basis of Emotion: A Meta-analytic Review

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Dissertation
by
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submitted in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy

May, 2010

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Abstract

Researchers have wondered how the brain creates emotions since the early days of psychological science. With the advent of neuroimaging techniques in the early 1990's and a surge of studies in affective neuroscience in recent years, scientists are now poised to answer this question. In this paper, I present the most up-to-date and statistically advanced meta-analytic summary of the human neuroimaging literature on emotion. I compare the locationist approach (i.e., that emotion categories consistently and specifically correspond to distinct brain regions) with the psychological construction approach (i.e., that emotions are constructed of more general brain networks not specific to emotions) to better understand the brain basis of emotion. I begin by outlining the set of brain regions consistently activated across all studies of emotion experience and perception. I next report findings from two sets of analyses probing the brain basis of discrete emotion categories. The first types of analysis demonstrates the brain regions that are consistently associated with the experience and perception of anger, disgust, fear, happiness and sadness across studies. The second type of analysis demonstrates the mental states (e.g., emotion experience or perception, cognitive load, locus of attention, mental response to methods, etc.) that are consistently associated with activity in given brain locations across studies. Overall, there was little evidence that discrete emotion categories can be localized consistently and specifically to individual brain regions. Instead, I found evidence that is consistent with a psychological construction approach to the mind: a set of common processes corresponding

to interacting brain networks constitute emotion experience and perception across a range of emotion categories.

> "…of two things concerning the emotions, one must be true. Either separate and special centres, affected to them alone, are their brain-seat, or else they correspond to processes occurring in the motor and sensory centres already assigned…"
>
> (James, 1890, p. 473)

At the turn of the 19th century, William James asked how emotions were created by the brain. In this paper, I statistically summarize the last two decades of neuroimaging research on emotion in an attempt to answer this question. I examine the utility of two different brain-based approaches to understanding emotion. The *locationist* account assumes that all mental states belonging to the same category (e.g., "fear") are produced by brain activity that is consistently and specifically associated with an architecturally defined locale (e.g., the amygdala), and that mental states belonging to different categories (e.g., "anger," "sadness," and "disgust") are associated with brain activity in different locales (e.g., the orbitofrontal cortex, the anterior cingulate cortex, the insula). A *psychological constructionist* account, in contrast, assumes that all mental states (regardless of which emotion category they belong to) are realized by interacting brain networks that are not functionally specific to any emotion category or even to the category "emotion" itself. These brain networks correspond to more basic functions or ingredients of the mind, such as processing sensory information from the body (experienced as core affect), making meaning of sensory signals, language and executive control. In this paper, I use a meta-analysis of the human neuroimaging literature on emotion to compare locationist and constructionist approaches to the brain basis of emotion. Although other theoretical models dealing with the nature of emotion exist (such as social constructionist and appraisal approaches), they have yet to

inspire neuroscientific investigations and so I do not review them here. I begin the paper with a brief theoretical summary of the locationist and psychological constructionist approaches and outline the specific hypotheses being evaluated. Next, I review how each approach has fared with previous meta-analyses of the neuroimaging literature on emotion. Finally, I present the present meta-analytic findings summarizing all neuroimaging studies on the experience and perception of "anger," "fear," "disgust," "happiness" and "sadness" published between 1993 and the end of 2007. This is the first paper using a new and improved statistical method to explicitly evaluate the brain basis of discrete emotions. I specifically evaluate the locationist approach by examining whether discrete emotion categories consistently and specifically activate certain brain regions, and discuss the relevance of these findings to a psychological construction approach. I next review other brain regions found to be consistently activated across emotion categories, bearing on the constructionist approach. In the process, I also report whether methodological variables influence the meta-analytic findings in any significant way. I close the paper by locating the results in the broader science of emotion, and discuss how this meta-analytic approach places scientists one step closer to understanding the complexities of mind-brain correspondence.

The Brain Basis of Emotion

Locationist Accounts of Emotion

The locationist account of emotion is most clearly associated with the basic emotion approach. Although basic accounts of emotion date back to the beginnings of Western civilization (e.g., Hippocrates' conception of the four humors), most scientists locate their modern beginnings with Darwin's (1972) *The Expression of Emotions in Man and Animals.* In

this book, Darwin used commonsense, essentialized ideas about emotion in a teleological fashion to support his claims about natural selection. In actuality, Darwin was not trying to fromulate a theory of emotion. His book was written in response to Charles Bell's (1802) *Anatomy and Physiology of Expression*, which claimed that humans have divinely created muscles to express their feelings (for discussion, see Gendron & Barrett, 2009; Russell, 1994). Nonetheless, early scientists found Darwin's ideas inspiring and codified them into a set of scientific hypotheses that today ground the modern "basic emotion" approach (e.g., McDougall, 1908; Allport, 1924; for a discussion see Gendron & Barrett, 2009). One of Darwin's lasting legacies in the field of emotion, in fact, is the assumption that specific expressions derive from ancient nervous system mechanisms shared with our mammalian cousins. This idea, along with the idea suggested somewhat earlier by Spencer (1855) that responses belonging to different emotion categories are consistently and specifically caused by distinct brain regions, set the stage for later locationist accounts of emotion, including those put forth by Tomkins (1962/1963), Ekman (1972), and Panksepp (1998). Nearly a century later, Ekman (1999) reiterated these ideas, stating:

> "It is necessary to posit emotion-specific central nervous system (CNS) activity in my account of basic emotions. The distinctive features of each emotion, including the changes not just in expression but in memories, imagery, expectations and other cognitive activities, could not occur without central nervous system organization and direction. There must be unique physiological patterns for each emotion..."

Early work in the field of medicine and behavioral neuroscience corroborated early philosophical and psychological locationist efforts. In 1921, Cannon famously proposed that the brain basis of emotion resided in the thalamus (based on investigations of cat brains and human cadavers). Papez (1937), Yakovlev (1948), and MacLean (1949) all expanded upon this finding to suggest that emotions derived from the primitive subcortical "limbic system."

According to Papez, the limbic system included the hypothalamus, thalamus, parahippocampal gyrus, and cingulate. Yakovlev (1948) expanded Papez's model to include orbitofrontal cortex, the insula, anterior temporal lobes, and amygdala. MacLean (1949) added the hippocampus, hippocampal gyrus, amygdala, pituitary gland, basal ganglia and midbrain and assimilated the "limbic system" into his concept of the "triune brain." The triune brain relies on hierarchical conceptions of brain organization and function. For instance, homeostasis and motor function is thought to derive from the "reptilian" brainstem and cerebellum. Emotions are thought to derive from the limbic system, which presumably emerged in during the evolution of early mammals. Higher-order functions (i.e., cognition), including the ability to regulate the more basic functions are thought to have emerged with the evolutionarily "new" neocortex. Although it is now recognized that the triune brain concept does not best characterize brain evolution (see Streider, 2005), the idea that emotions derive from the subcortex (and are regulated by the cortex) is still reflected in some locationist accounts. Panksepp (1998; 2007), for instance, posits that emotions like anger, fear, lust, distress, love, joy, and expectancy derive from discrete subcortical networks in the mammalian brain. (For a recent discussion of the empirical standing of this model, see Barrett, Lindquist et al. 2007; Panksepp, 2007).

Although differing in specifics, locationist accounts of emotion share the fundamental assumption that the category "emotion" and individual categories such as "anger," "disgust," "fear," "happiness," and "sadness" (and perhaps a few others) are categories that respect brain anatomy (i.e., natural kind categories; see Barrett, 2006a for a discussion). Over the past two decades, the cognitive neuroscience literature has made vigorous efforts to identify the consistent and specific brain basis for discrete categories of

emotion (see Calder, 2003). These efforts have been inspired, in large part, by behavioral neuroscience research in animals that has carefully mapped the circuitry for particular action patterns (e.g., freezing, attack, vocalizations). Table 1 lists the specific locationist hypotheses. I focus only on the hypotheses that have been the target of intense scientific inquiry, namely the amygdala-fear, insula-disgust, OFC-anger, and ACC-sadness hypotheses.[1] Important empirical findings ground each hypothesis.

The Amygdala-Fear Hypothesis

The amygdala is a small, almond shaped structure deep within the temporal lobe (see Figure 1, inset 1). The first animal work to suggest a role for the amygdala (and the temporal lobe more generally) in emotion found that monkeys increased exploratory behavior and were less cautious of novelty following temporal lobectomies (Kluver & Bucy, 1937). The hypothesis that the amygdala is the brain locus of fear was most clearly popularized by behavioral neuroscience work showing that the amygdala plays a role in the blood pressure and heart rate changes involved in "fear learning" (i.e., classical conditioning; when rats

[1] I do not discuss a locationist hypothesis of happiness because a clear locationist hypothesis has yet to emerge in the literature. Recent meta-analytic evidence has linked happiness to the dorsomedial prefrontal cortex/dorsal anterior cingulate cortex (DMPFC/dACC) (Murphy et al. 2003) and basal ganglia (Phan et al. 2002) (other meta-analyses link happiness to a number of different brain regions but for the sake of brevity, I do not innumerate those brain regions here; see Fusar-Poli, et al. 2008; Vytal & Hamann, in press). One recent hypothesis has linked the ventral striatum to happiness (Kringelbach & Berridge, 2009). This hypothesis derives from findings in rats and humans linking the ventral striatum to reward and hedonic pleasure (see Berridge & Kringelbach, 2008 for a review). It remains unclear from the evidence that the ventral striatum is specific to hedonic pleasure, however, never mind happiness. Accumulating evidence suggests that the mesolimbic dopamine system (of which the ventral striatum is a part, is involved in directing attention to and modulating behavioral responses to a range of aversive, novel, and appetitive stimuli (for a review, see Grillner, Hellgren, Menard, & Saitoh, 2005). The mesolimbic dopamine system is also involved in gating attention to novel, salient, or unexpected environmental events that require an effortful 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). Even the nucleus accumbens, which has been consistently linked to pleasant hedonic states (see Kringelbach & Berridge, 2009) has aspects that are involved in avoidance-related behaviors (Reynolds & Berridge, 2002; 2003). The cells of the nucleus accumbens cell have a flexible, context-specific mapping such that those cells which respond to aversive cues in a stressful environment also respond to appetitive cues in a safe environment (Reynolds & Berridge, 2008).

freeze in response to a tone that has been previously paired with electric shock; LeDoux et al. 1983; 1985; 1990; for reviews see Fanselow & Poulous, 2005; Fendt & Fanselow, 1999; LeDoux, 2007; Ohman, 2009) or "fear potentiated startle" (when rats show an enhanced startle response to said tone; Davis, 1992; Hitchcock & Davis, 1986; 1987; see Davis et al. 2008; Fendt & Fanselow, 1999). The amygdala-fear hypothesis was further strengthened by evidence that humans show increased amygdala activity during aversive learning (LaBar et al. 1998). Individuals with amygdala lesions (LaBar et al. 1995) or atrophy (Bechara et al. 1995) show impaired skin conductance responses to neutral tones that were previously paired with noxious noise blasts (i.e., impaired "fear learning") and have difficulty perceiving fear in voices (Brierley et al. 2004; Scott et al. 1997, but see Adolphs & Tranel, 1999; Anderson & Phelps, 1998), bodies (Sprengelmeyer et al. 1999; but see Atkinson et al. 2007), and faces (e.g., Adolphs et al. 1994; 1995; 1999; although see Adolphs et al. 2005 for an alternate account, also discussed in the *Amygdala-Salience Hypothesis* below). Finally, the amygdala is implicated in psychopathology involving the experience of fear and anxiety (for a review see Damsa et al. 2009; for a meta-analytic review, see Etkin & Wager, 2007).

The Insula- Disgust Hypothesis

The insula is an extension of the claustrum, a section of ancient cortex, and is generally involved in representing sensory cues from the body (Craig, 2002, 2009) (see Figure 1, inset 2). The granular posterior section of the insula contains a somatotopic map of the body (e.g., Brooks et al. 2005; Hua et al. 2005). The agranular anterior aspect of the insula is important for representing sensory information from the viscera. It is paralimbic cortex and contains primary cortical regions for representing taste and smell. The dysgranular anterior

aspect is evolutionarily more recent (and perhaps unique in humans), and is involved in interoception and the representation of conscious feelings (see Craig, 2002, 2009).

The insula – disgust hypothesis originates in the belief that disgust evolved from a primitive food rejection reflex (Rozin et al. 2000) or aversion to disease-threat (e.g., Curtis et al. 2004) (with the assumption that both involve cues from the body). The insula's role in disgust has not been investigated using animal models to date. The empirical evidence that supports the insula-disgust hypothesis instead derives from research with humans (e.g., Jabbi et al. 2008; Wicker et al. 2003; see Calder et al. 2001; Calder, 2003 for reviews). Individuals with damage to the insula and basal ganglia have difficulty perceiving disgust in facial and vocal caricatures (Adolphs et al. 2003; Calder et al. 2000). Such individuals also report experiencing less disgust in response to scenarios about body products, envelope violation, and contamination (which typically evoke disgust in people with intact insulas) (Calder et al. 2000). Individuals with Huntington's and Parkinson's disease (neurodegenerative diseases affecting the insula and basal ganglia) show diminished experiences of disgust to foul smelling odors (Mitchell et al. 2005) and have difficulty perceiving disgust in the faces of others (e.g., Sprenglemeyer et al. 1996; 1998; Suzuki et al. 2006; Kipps et al. 2007; see Calder et al. 2001; Sprengelmeyer, 2007 for reviews). (Although of note, other studies have found a decrement in emotion perception more generally, e.g., Milders et al. 2003).

The Orbitofrontal Cortex-Anger Hypothesis

The orbitofrontal cortex (OFC) includes both paralimbic, granular, and dysgranular cortex in Brodmann's areas 47/12, 11, 13, 14, and 10. (see Öngür et al. 2003). The lateral OFC (lOFC) consists of areas 47/12, 11l, 13l and 13m (see Figure 1, inset 3; Figure 2; purple areas). At its posterior border, lOFC is contiguous with the anterior insula and extends

laterally to the frontal operculum. The lOFC has connections to the secondary association areas for all sensory modalities (e.g., Carmichael & Price, 1995a; see Kringelbach & Rolls, 2004) and to limbic areas including the cortical aspects of the amygdala (e.g., basolateral complex; e.g., Carmichael & Price, 1995b). The medial OFC (mOFC), sometimes called ventromedial prefrontal cortex (vmPFC), consists of areas 10m, 10p, 14r and 11m (Öngür et al., 2003) (see Figure 2; blue areas). The mOFC has robust reciprocal connections to all limbic areas (including many nuclei within the amygdala and the ventral striatum), as well as to the hypothalamus, midbrain, brainstem and spinal cord areas that are involved in viceromotor control (e.g., Carmichael & Price, 1995a; Eblen & Graybiel, 1995; Ongür & Price, 1998; 2000; Rempel-Clower & Barbas, 1998; see Kringelbach & Rolls, 2004 for a review).

Primary support for the OFC-anger hypothesis derives from prior meta-analytic reviews of the neuroimaging literature that found consistent OFC activation across studies of anger (Murphy et al. 2003; Vytal & Hamann, in press). These findings are consistent with the electroencephalography (EEG) literature linking electrical activity in left prefrontal cortex to anger and approach motivation. For instance, activity in left prefrontal cortex is associated with the experience of anger in response to an insult (Harmon-Jones & Sigelman, 2001), with an increased tendency to retaliate towards another person following an insult (by allocating him a dose of unpleasant hot sauce in a putative taste test; Harmon-Jones & Sigelman, 2001), and with high levels of self-reported trait anger and aggression (Harmon-Jones & Allen, 1998).

Lesion studies in animals and humans have also been cited as evidence for an OFC-anger hypothesis (see Murphy et al. 2003). It remains unclear that these studies offer strong

support for a locationist hypothesis of anger, however. Most studies find that monkeys are more aggressive towards humans (Raleigh et al. 1979) and are more likely to threaten other monkeys (Deets et al. 1970; Machado & Bachevalier, 2006) following OFC lesions, implying that the OFC is not necessary to (i.e., does not generate) aggression. Only several studies find that lesions to lOFC and mOFC reduce aggressive behavior in monkeys (towards humans; Butter & Snyder, 1972; Kamback, 1973; and other monkeys, Raleigh et al. 1979). The findings in rats mirror the majority of the monkey findings. Although aggressive behavior is associated with increased activity in rat ventral forebrain (including the OFC) (Ferris et al. 2008; Halász et al. 2006), lesions to large swaths of the OFC increase the tendency that a rat will aggress against other rats that enter its home cage (e.g., de Bruin et al. 1983).

This pattern of findings is also reflected in studies of humans lesion patients and humans with pathology targeting the OFC. A large literature links vmPFC (including mOFC) damage to socially aberrant behavior (e.g., Eslinger & Damasio, 1985; Saver & Damasio, 1991). Individuals with vmPFC lesions become frustrated more easily and engage in more verbal (but not physical) aggression than do neurologically intact healthy control subjects (Grafman et al. 1996). Psychopathy and antisocial disorder are marked by increased aggression and correspond to structural (e.g., Raine et al. 2000) and functional (e.g., Harenski et al. 2009; Glenn et al. 2009) changes to mOFC (for a recent meta-analysis, see Yang & Raine, 2009). Fewer studies have linked lOFC to aggressive behavior, but one study found that individuals with borderline personality disorder who have lowered baseline lOFC (BA 47) activity are more likely to aggress against others (Goyer et al. 1994).

Together, the lesion and human pathology findings call into question the idea that the OFC is the brain seat (i.e., generates) aggression/anger. Should the OFC be consistently and specifically implicated in anger, then these findings would instead imply that it plays a role in the regulation or gating of behavioral responses associated with anger. One goal of this meta-analytic review will thus be to test this provisional OFC-anger account.

The ACC-Sadness Hypothesis

The anterior cingulate cortex (ACC) is an area of agranular cortex that wraps around the corpus callosum along the medial wall at the front of the brain. The ACC consists of a ventral surface (vACC; including BAs 24a, b, 25 and 32) that is divided into subgenual and pregenual portions, as well as a dorsal surface (dACC; including BAs 24a', b', c' and 32') (Bush et al. 2000; Paus, 2001) (see Figure 1, inset 4 and Figure 3). The vACC controls autonomic responses via connections with the amygdala, PAG, brainstem motor nuclei and the spinal cord (Devinsky et al. 1995; Vogt et al. 1992). Electrical stimulation of the vACC in human and non-human primates activates a range of autonomic functions including cardiovascular, respiratory, digestive, thermoregulatory, and endocrine systems (see Devinksy et al. 1995 for a review). The dACC, on the other hand, has connections to executive attention and motor control regions such as the dorsolateral and ventrolateral prefrontal cortex and supplementary motor area (e.g., Barbas & Pandya, 1989; see Devinsky et al. 1995 for a review).

The ACC-sadness hypothesis, like the OFC-anger hypothesis, derives primary support from prior meta-analyses of the neuroimaging literature (e.g., Murphy et al. 2003; Phan et al. 2002). Little research in animal links the ACC to sadness. Panksepp (1998, 2007) lists the ACC as part of the mammalian brain circuit for "distress" due to its role in

producing vocalizations in infant animals that have been removed from the nest (although the specificity of this relationship is in question; see Blumberg & Sokoloff, 2001). Some support for an ACC-sadness hypothesis derives from the human lesion literature. Individuals with dACC lesions (including dorsomedial prefrontal cortex, BA 9) are hypersensitive and more likely to cry at sad events than are individuals with intact dACCs (Hornak et al. 2003). Perhaps the most-cited evidence for an ACC-sadness hypothesis stems from studies of depression. Clinical depression is marked by structural and functional changes in vACC (see Gotlib & Hamilton, 2008 for a review), although clinical depression admittedly involves many symptoms above and beyond the experience of sadness (see Coyne, 1984). Furthermore, the vACC is hyper-activated during mania (see Fountoulakis et al. 2008), calling into question the specificity of this structure's role in sadness or even negative affect. In reference to these clinical findings, it is interesting that electrical stimulation of the vACC (BA 25) relieves intractable depression by reducing feelings of apathy and anhedonia, normalizing sleep disturbances and decreasing gross motor impairments (e.g., Mayberg et al. 2005). One goal of this meta-analytic review will thus be to test this provisional ACC-sadness account.

Psychological Constructionist Accounts of Emotion

Psychological constructionist accounts of emotion date back to the beginning of psychological writings on emotion (Wundt, 1897; James, 1884). These psychological construction accounts, as well as those that followed, all share the basic assumption that emotions are psychical compounds constructed out of more basic psychological ingredients that are not themselves specific to emotion. One primary ingredient in all psychological construction models of emotion is some form of information from the body. James (1884)

emphasized the importance of raw sensory processing of somatic, visceral, vascular and motor cues from the body as the basic building block of emotion (see also Duffy, 1957; Mandler, 1975, 1990; and Schachter & Singer, 1962 who referred to this ingredient as "arousal"). Wundt (1897) focused on the mental representation of those internal cues, which he called "affect" (see also Barrett 2006b; Barrett & Bliss-Moreau, 2009; Harlow & Stagner, 1932; Hunt, 1941; Russell, 2003). Psychological construction accounts do not simply reduce emotion to affect, however (as is often claimed in summaries of so-called "dimensional models of emotion"; e.g., Keltner et al. 2003). Typically, these models include a second ingredient, which is a process by which internal sensory or affective states are made meaningful (i.e., as related to or caused by the external surroundings). It has been proposed that this meaning analysis is the result of ideas (Wundt, 1897), social referencing (Schachter & Singer, 1962), attribution (Russell, 2003), or categorization (Barrett, 2006a, b; Barrett, Lindquist, & Gendron, 2007). This meaning analysis of the body is assumed to proceed automatically with little sense of agency or effort. As discussed below, the most recent psychological constructionist model proposed two additional ingredients that are important to emotion: language for emotion (Barrett, Lindquist, & Gendron, 2007) and executive attention (Barrett, 2009a; Barrett, Tugade, & Engle, 2004).

In some models, psychological ingredients combine in stages (e.g., Wundt, 1897/1998; Schachter & Singer, 1962; Russell, 2003). In other models they combine and constrain one another like ingredients in a recipe, influencing and shaping one another in real time (Barrett, Lindquist et al. 2007). It is hypothesized that this process is smoothly managed smoothly by executive attention (Barrett, 2009a; Barrett, Tugade, & Engle, 2004). *Emergent* psychological construction models view emotions as more than the sum of their

parts (e.g., Wundt, 1897). *Elemental* psychological construction models, on the other hand, ontologically reduce emotion to their more basic psychological ingredients (e.g., Duffy, 1957; James, 1884; Russell, 2003). In all psychological construction models, the ingredients that constitute the psychological states that people colloquially refer to as "emotion" also constitute other mental states that people refer to as "cognitions" (e.g., thoughts, beliefs, and memories; Duncan & Barrett, 2007).

William James framed the question of brain-emotion correspondences when he wrote "A science of the relations of mind and brain must show how the elementary ingredients of the former correspond to the elementary functions of the latter." (1890/1998, p. 28). For the most part, however, psychological construction models of emotion have not included specific hypotheses about brain-emotion correspondences. Over the past several years, my lab has developed a psychological construction model that outlines a specific set of hypotheses for mapping brain states to psychological categories such as "emotion," "anger," "sadness," "fear," "disgust", and "happiness" (Barrett, 2006a, b, 2009a, b; Barrett, Lindquist, Bliss-Moreau, Duncan, Gendron et al. 2007; Barrett, Lindquist, & Gendron, 2007; Barrett, Mesquita, Oschner & Gross, 2007; Gendron & Barrett, 2009; Lindquist & Barrett, 2008). Our model assumes that complex psychological categories are not natural kind categories that are respected by brain anatomy (or that can be distinguished from one another by a correlated set of measurable features such as facial behaviors, peripheral physiology, etc.; for empirical reviews, see Barrett, 2006a; Barrett, Lindquist et al., 2007; Barrett & Wager, 2006). Instead, we hypothesize that each psychological category (e.g., "emotion") corresponds to a functional "neural reference space." According to Gerald Edelman (1989) who coined the

term, a "neural reference space," refers to the neuronal workspace that implements the collection of brain states corresponding to a class of mental events.

Taking inspiration from connectionist and network approaches to the brain (e.g., Büchel & Friston, 2001; Fuster, 2006; Mesulam, 1998; Raichle & Snyder, 2007; Seeley et al, 2007), we hypothesize that the neural reference space for discrete emotion is populated by a number of distributed networks that constitute the fundamental building blocks of all mental states. They are the constituents out of which all mental states, such as "emotions," "cognitions," and "perceptions" emerge. Each functional network is thought to correspond to a basic psychological ingredient that serves a more general psychological function in the brain. Like ingredients in a recipe, their weighting and contribution is predicted to vary across instances of each emotion category.

According to our psychological construction model, there are at least four basic psychological ingredients that help construct emotions (and all mental states, for that matter). Although I only discuss four ingredients here, I do not assume that this list is complete or exhaustive. Similar to all other psychological construction views, the first hypothesized ingredient is core affect. Core affect is a neurophysiological state associated with changes in the core of the body. Core affect can be experienced as subjective feelings of pleasure or displeasure with some degree of arousal, although it need not be (Barrett & Bliss-Moreau, 2009; Russell, 2003; Russell & Barrett, 1999). Changes in core affect are a homeostatic barometer – the body's way of signaling what is valuable and what is not in given the context at a particular point in time. If sensory stimulation from the outside world tells you what is in the environment, then core affect tells you what to do about it. Core affect is at the core of every emotional experience in people across the world (Mesquita,

2003). The concept of core affect is similar to Damasio's concept of the "core self" (Damasio, 1999), Craig's hypothesis that interoceptive sensations form the core of consciousness (Craig, 2009), and Cabanac's hypothesis that affect is a common currency that determines the value of things in the world (Cabanac, 2002). The basic psychological processes that contribute to core affect and the brain regions that constitute these functional groups in the brain are discussed below.

The second ingredient proposed by our model of emotion is core association. This ingredient corresponds to what has been called the episodic memory network (e.g., Vincent et al. 2006), the prospective brain (Schacter et al. 2007) and the network involved in self-referential processing (see Mitchell, 2009), context-based predictions (Bar, 2009), and theory of mind (Saxe & Kanwisher, 2003). The regions in this network also compose a resting-state network called the "default network" (Raichle et al. 2001). I use the term core association (after Buckner and colleagues) here because this network associatively recombines prior experiences and is at the core of a number of mental processes including the construction of past experiences (as in episodic memory; see Buckner & Carroll, 2007), the construction of representations of the future (as in simulation; see Buckner & Carroll, 2007), and the categorization of exteroceptive sensations in the present (Bar, 2007). In emotion, this network is thought to make a prediction about what sensory input from the body means and what caused it (i.e., in categorizing it) (Barrett, 2006 b; 2009b). In so doing, these brain areas help to realize an emotional gestalt, or what Edelman calls "the remembered present" (cf. Edelman, 1989; see Barrett, Mesquita et al., 2007; Barrett, 2009b).

The third hypothesized ingredient is language. Language likely works in tandem with core association to flexibly make meaning of core affective states during emotional

experiences and perceptions (Barrett, Lindquist & Gendron, 2007). One possibility is that core association helps the brain make gist-level predictions about what a core affective state means (i.e., that negative, high arousal affect is about a stimulus in the world), whereas linguistic concepts are brought to bear in making more specific, fine-grained distinctions that produce emergent discrete emotional states (e.g., that negative, high arousal affect is about blocked goals; i.e., is an instance of anger).

The fourth hypothesized ingredient is executive attention. In our psychological construction view, controlled attention is employed in the process of categorizing affect to produce an "emotional gestalt." Executive control helps seamlessly negotiate which conceptual elements are activated and which are suppressed during categorization (see Barrett, Tugade, & Engle, 2004, for a discussion) (c.f. Barrett, 2009b).

In our psychological construction perspective, the functions of distinct brain areas are best understood in terms of which basic psychological ingredients they instantiate. Because the same brain region might help constitute a number of ingredients in the brain, a brain's function is best conceived of in the context of the other brain areas to which it is connected (either anatomically or because of the timing of neural firing). The same brain areas might be consistently activated across a range of emotion categories (and although it is beyond the scope of this paper, even in non-emotional states), meaning that they are not specific to any emotion category (or even to emotion per se). A brain region might be functionally selective for a given emotion in a given instance, however. Although a brain region would not be considered the locus of a particular emotion, it is possible that it plays an important function in that emotion category because supports one of the more basic ingredients that contribute to the emergent emotion.

I next present the psychological construction hypotheses for the particular brain areas identified in locationist hypotheses and link them to the ingredient(s) they are hypothesized to support (Table 2). Following this discussion, I outline a set of brain regions that are not predicted a priori by locationist accounts, but that play a key role in grounding psychological ingredients in our psychological constructionist account. Table 3 outlines the ingredients and their hypothesized neural networks.

Amygdala-Salience Hypothesis

From a psychological constructionist view, the amygdala is part of the distributed network for core affect. In particular, the amygdala is involved in signaling whether exteroceptive sensory information has motivational salience to an organism (for similar views see Adolps, 2008; 2009; Duncan & Barrett, 2007; Sander et al. 2003; Whalen, 1998; 2007). A stimulus is motivationally salient when the brain cannot easily predict its identity, its value in that context, the best response to deal with it, or the consequences of the response. Salient objects or events are affectively potent because they influence an organisms' body state in a way that can be experienced as core affect (i.e., feelings of pleasure or displeasure with some degree of arousal) (Barrett & Bliss-Moreau, 2009). When something is salient, the amygdala signals other parts of the brain to sustain processing in order to learn more about that thing or resolve ambiguity about a behavioral response. From a psychological construction point of view, "fearful" stimuli might fall into the class of uncertain and therefore salient stimuli, but the amygdala is not specific to the category "fear."

From an anatomical standpoint, the amygdala is well positioned to signal the presence of salient stimuli. It projects to all but 8 cortical sites in humans (Young et al. 1994), modulating the networks involved in processing every exteroceptive sensory modality

(vision, e.g., Freese & Amaral, 2006; see Amaral et al. 1992; audition, e.g., LeDoux et al. 1991; Romanski et al. 1993; olfaction, e.g., Carmichael et al. 1994; McDonald, 1998; touch, e.g., Romanski et al. 1993; taste; Halsell, 1992; Norgren, 1976). The amygdala also has connections to lOFC (e.g., Öngür & Price, 2000; see Pessoa, 2009), which itself projects to the sensory modalities. Finally, the amygdala also has strong reciprocal connections to the executive attention network involving the dACC (e.g., Öngür & Price, 2000; see Pessoa, 2009) and the core association network (including the hippocampus and entorhinal cortex; for a review, McGaugh, 2004). The result is that the amygdala influences both what is attended to in the moment and what is stored in long term memory.

Consistent with the amygdala-salience hypothesis, the amygdala is routinely implicated in orienting attention to salient stimuli (see Holland & Gallagher, 1999). The amygdala responds to stimuli that are experienced as subjectively arousing (e.g., Bradley et al. 2001; Weierich et al. 2010), intense (e.g., Bach et al. 2008) and emotionally "impactful" (e.g., Ewbank et al. 2009). Novel (Breiter et al., 1996; Schwartz et al., 2003; Weierich et al. 2010; Wilson & Rolls, 1993; Wright et al., 2003; Wright et al., 2006; Wright et al. 2008) and uncertain (Herry et al. 2007) stimuli robustly activate the amygdala and produce cardiovascular responses associated with affective changes (Mendes et al. 2007). Amygdala lesions disrupt normal responses to novelty and uncertainty in mammals (e.g. Bliss-Moreau et al. in press; Burns et al., 1996; Mason et al., 2006; Missilin & Ropartz, 1981; Nachman & Ashe, 1974; for reviews, see Knight and Grabowecky, 1999; Petrides, 2007). Finally, amygdala responses habituate rapidly, even in the continued presence of salient stimuli like fearful faces (Breiter et al. 1996; Büchel et al. 1999; Fischer et al. 2003; Whalen et al. 2004;

Wright et al. 2001). This fact calls into question the idea that the amygdala is necessary for all instances of fear perception or experience.

These findings explain the amgydala's role in "fear learning" without assuming that the amygdala is specific to fear. More likely, amygdala activity reflects orienting responses that occur when an organism attends to neutral and salient stimuli during learning. The amygdala contributes to the production of the skin conductance responses (SCRs) (Laine et al. 2009) used to index "fear learning." SCRs are known to covary with changes in attention (e.g., Blakeslee, 1979; Spinks et al. 1984), consistent with an orienting account. Furthermore, there is evidence that the amygdala-driven SCRs that occur during learning are linked more to changes in attention than anticipation of an aversive stimulus. Amygdala responses are associated with the SCRs that occur immediately following the onset of a conditioned stimulus (e.g., a tone), but not the SCRs that occur prior to the onset of the unconditioned stimulus (e.g., a shock) (Cheng et al. 2007). That the amygdala is involved in attention to salient stimuli during learning but not fear per se would explain why the amygdala is activated when organisms learn that a neutral stimulus predicts reward (e.g., Paton et al. 2006; for a review see Murray, 2007). Indeed, the amygdala is implicated in a host of mammalian social behaviors (e.g., male and female sexual behavior, maternal behavior, aggression; see Newman, 1999), probably because social stimuli are, by default, salient.

Lastly, the amygdala-salience hypothesis helps to explain why the category "fear" is not always linked to amygdala response. A number of behaviors that rats perform in threatening contexts, such as avoiding the location of the threat (e.g., Vazdarjanova & McGaugh, 1998) and defensive treading (where rats kick their bedding in the direction of the threat; Reynolds & Berridge, 2002, 2003, 2008) are not amygdala-dependent (e.g., Kopchia et

al. 1992; Vazdarjanova & McGaugh, 1998). Although the amygdala is required to learn that a tone predicts a shock, it is not required to retain this association. Monkeys still startle at a tone that previously acquired aversive value even following bilateral amygdala removal (e.g., Antoniadis et al. 2009). In humans, the amygdala is activated more so by tasks requiring a person to learn the predictive value of a fearful face than by tasks where fearful faces are passively viewed (Hooker et al. 2006). Furthermore, the amygdala shows minimal activation to fearful faces with averted gazes, although such faces signal imminent danger (Adams et al. 2003). Instead, the amygdala routinely shows increased activation (although it is short lived; e.g., Fischer et al. 2003) to fearful faces with a forward gaze (where the target person is looking straight at the perceiver), presumably because these expressions are rare and difficult to interpret (Whalen et al. 2001). Even an individual with bilateral amygdala damage is capable of perceiving fear in posed expressions once she is explicitly directed to look at the eyes of target faces (Adolphs et al. 2005). She can also accurately perceive fear in faces under subliminal and rapid presentation latencies (e.g., Tsuchiya et al. 2009) and in bodies posed to depict fear (Atkinson et al. 2007).

The Anterior Insula-Interoception Hypothesis

From a psychological constructionist view, the insula is not the brain locus of disgust, but plays a key role in representing core affective states. The insula is key node in a network for receiving and representing feedback from the body. To the extent that brain states corresponding to disgust represent a stimulus' consequence for the body as a key element, the anterior insula should be activated in disgust. Indeed, a key ingredient in the mental states referred to as "disgust" is likely a representation of how an object will affect

the viscera. A psychological constructionist approach would not hypothesize that the insula is specific to disgust, however.

From an anatomical perspective (see Craig, 2002, 2003, 2008, 2009), it is easy to see why the anterior insula holds a key position in representing core affective feelings. Posterior aspects and mid-insular cortex serve as primary and secondary sensory cortex for somatovisceral cues and in so doing represent interoceptive sensations from the body in a somatotopic manner (e.g., Brooks et al. 2005; Hua et al. 2005). The anterior insula is implicated in the re-representation (i.e., subjective awareness) of these sensations (e.g., Craig et al. 2000; see Craig, 2002; 2003; 2009 for reviews), and shows increased activation during awareness of body movement (e.g., Tsakiris et al. 2007), awareness of body sensations like gastric distention (e.g., Wang et al. 2008), and in orgasm (e.g., Ortigue et al. 2007). Furthermore, there is growing evidence that the insula is a hub in a large-scale resting state network involved in attention (Corbetta & Shulman, 2002; Corbetta et al. 2008), suggesting that body-based signals constitute a form of attention in the brain (see Duncan & Barrett, 2007 for a discussion of affect as attention).

The OFC- Context-based Behavior Hypothesis

The OFC plays a role in core affect as a key node in a network that integrate sensory information from the world and body to guide behavior in a context-specific, goal-dependent manner. Sensory information from the world tells an organism what is in its environment—sensory information from the body (experienced as core affect) tells the organism what to do about those things. From an anatomical standpoint, the OFC is well suited to guide behavior by integrating sensory information from the world and body. Except for the rhinal regions of the temporal lobes, the OFC is the most polymodal region

in the brain (cf. Kringelbach & Rolls, 2004; e.g., Barbas, 1988). Reciprocal connections between the OFC and gustatory, olfactory, somatosensory, auditory and visual modalities, along with visceral input from the insula and the thalamic nucleus, allow the OFC to unite diverse sources of sensory input (Rolls, 1999). The OFC has reciprocal connections to the amygdala (Carmichael & Price, 1996) and ACC (Van Hoesen et al. 1993), which form a source of attention in the brain (see sections on *The amygdala-salience hypothesis and The ACC-conflict hypothesis)*. The OFC also has reciprocal connections with other frontal areas such as BA 9 and 46 involved in executive control (Barbas & Pandya, 1989; Carmichael & Price, 1995a) and premotor areas involved in volitional behavior (Barbas & Pandya, 1989). These connections, along with outputs to the hypothalamus, PAG, and ventral striatum allow the OFC to guide motivation and behavior (for a review of the OFC and connections see Kringelbach & Rolls, 2004).

Findings linking the mOFC to decision-making (e.g., Bechara et al. 1996; 2000; Koenigs et al. 2007), associative learning (e.g., Rolls et al. 1994; 1996) and the valuation of objects (e.g., Chib et al. 2009) are consistent with an OFC-context-based behavior hypothesis. The inability to properly integrate exteroceptive and interoceptive information to guide behavior would explain why social behavior is altered in individuals with OFC lesions, even in the presence of preserved intelligence and executive function (e.g., Eslinger & Damasio, 1985; Saver & Damasio, 1991) (for a similar view see Damasio et al. 1990). This hypothesis also explains the evidence linking the OFC to anger and aggression (see section of the *OFC-Anger Hypothesis*). Failure to integrate exteroceptive and interoceptive information will result in behaviors that are not well-tuned to the context (e.g., dysregulated aggression). Indeed, one account suggests that psychopaths are aggressive not because the

OFC is the brain seat of anger, but because an inability to learn the changing value of stimuli causes them frustration (Blair, 2007).

The ACC-Conflict Hypothesis

According to a psychological constructionist view, the ACC helps compose core affect in the brain as part of a network for detecting and guiding behavior in the face of conflicting sensory inputs (for a discussion of the ACC's role in conflict monitoring see Botvinik, 2007; Bush et al. 2000). Conflict might stem from competition between different types of exteroceptive sensory representations (e.g., visual v. auditory representations; two different visual representations), or between exteroceptive sensory representations and interoceptive representations. One means of representing conflict in the external world is as core affective feelings (Botvinik, 2007; Hacjack & Foti, 2003).

Together, the subcomponents of the ACC are anatomically well-suited for computing conflict and responding to conflict. The dACC is known to play an executive role and uses exteroceptive sensory information (via thalamic projections; Barbas et al. 1991) and interoceptive sensory information (from the insula; Mesulam & Mufson, 1989), to direct attention and motor responses via connections to DLPFC, VLPFC and SMA (e.g., Barbas & Pandya, 1989; see Devinsky et al. 1995; Paus, 2001 for reviews). Tasks involving conflict include those where individuals are asked to inhibit a prepotent motor response (such as in the classic "go/no go" task; e.g., Braver et al. 2001; Durston et al. 2002), to process competing sensory stimulus arrays (such as in the classic "Stroop" task, e.g., Botvinick et al. 2001; Bunge et al. 2002), or tasks on which errors are made (Hajcack & Foti, 2008) (for reviews see Botvinick, 2007; Bush et al. 2000; Paus, 2001). One means by which the dACC might engage attention and influence behavior following the detection of conflict is via the

production of a core affective state. Consistent with this hypothesis, dACC activity correlates with cardiac control during cognitive tasks (Critchley et al. 2003). The dACC also likely accomplishes autonomic control via innervations to the vACC which in turn projects to the PAG, brainstem, and the spinal cord (e.g., Devinsky et al. 1995; Vogt et al. 1992). dACC might respond to conflict in "top-down" manner by engaging regions involved in cognitive control, whereas vACC might do so in a more "bottom-up" way by causing a shift in an organism's core affective state.

Other Areas in the Neural Reference Space for Discrete Emotion

DMPFC, MTL, and Retrosplenial cortex/PCC. As part of the ingredient of core association, I predict that the dorsomedial prefrontal cortex (DMPFC), medial temporal lobe (MTL), and retrosplenial cortex/posterior cingulate cortex will play some consistent role in constructing the present, in the form of momentary experiences and perceptions of emotion (Barrett, 2009a; Barrett & Lindquist, under review) (see Figure 4).

ATL and VLPFC. As part of the network for language (e.g., for a review and meta-analysis see Price, 2000 and Vigneau et al. 2006), I predict that the anterior temporal lobe (ATL) and the ventrolateral prefrontal cortex (VLPFC) will be part of the neural reference space for discrete emotion (see Figure 5). The ATL plays an integral role in the representation of conceptual knowledge (Pobric et al. 2007; Lambon Ralph et al, 2009; Rogers et al. 2004), including the representation of abstract social concepts (e.g., Ross & Olson, in press; Zahn et al. 2007; 2009). Consistent with our psychological constructionist hypothesis that language is integral to emotion (Barrett, 2006b; 2009a; Barrett, Lindquist & Gendron, 2007), patients with semantic dementia have focal atrophy to the ATL and exhibit deficits in emotion perception (Rosen et al. 2004) and empathy (Rankin et al. 2006). Areas

of the VLPFC (e.g., BA 44, 45, 46) are known to be involved in the goal-directed access to conceptual knowledge and selection amongst competing response representations (e.g., Schnur et al. 2009; Thompson-Schill, 1997; Badre & Wagner, 2007). There is also growing evidence that areas of VLPFC ground abstract category knowledge including feature-based information about categories (e.g., Freedman et al. 2002; see Miller et al. 2002). To the extent that language is brought to bear in the categorization of affective states during the construction of emotion experiences (Barrett, 2009a) these brain regions will be consistently activated at greater than chance levels and therefore appear as part of the neural reference space for discrete emotion.

VLPFC and DLPFC. As part of the network for executive function (see Miller & Cohen, 2001; see Wager & Smith, 2003 for a meta-analysis), I predict that areas of lateral prefrontal cortex, including VLPFC and dorsolateral prefrontal cortex (DLPFC), will be part of the neural reference space for discrete emotion (see Figure 6). VLPFC helps comprise this ingredient given its role in goal-directed retrieval of conceptual knowledge. Bilateral DLPFC is helps comprise this ingredient because it is implicated in working memory (for a meta-analysis see Wager & Smith, 2003) and in the goal-directed control of attention (see Miller, 2000). This type of goal-directed control of attention is seen in emotion regulation (e.g. Ochsner et al. 2004; Urry et al. 2006) but is hypothesized to be active across every moment of emotion as executive control modulates the activity of other psychological ingredients in emotion (Barrett, 2009b).

Prior Meta-Analyses of the Brain Basis of Emotion

Meta-analyses of the neuroimaging literature are useful for comparing the scientific support for both the locationist and psychological construction models of emotion for two

primary reasons. First, a meta-analysis allows researchers to summarize hundreds of empirical studies by statistical means; this is particularly beneficial give the high rate of false-positives and largely variable experimental and statistical methods used across individual neuroimaging studies (see Wager et al. 2007; Kober & Wager, in press). Not only are the results more reliable than the findings from any given study, but it is possible to statistically model the influence of these between-study methodological and statistical differences. Second, neuroimaging is probably the best way to test a psychological construction model, where the experienced content of a mental state (whether it is anger or fear, or a memory, or a belief) will never reveal the processes that produced or realized that state in the first place. It is possible to construct the experience of emotion in behavioral studies by manipulating core affect and emotion knowledge and measuring the resulting emotion experience (e.g., Lindquist & Barrett, 2008). Yet with imaging, researchers can directly unmask the ingredients involved in constructing an experience, even if they are not accessible to conscious awareness.

Despite the obvious promise of meta-analysis for resolving questions about the brain basis of emotion, prior attempts to summarize the growing mass of neuroimaging studies on emotion have not yet realized their full potential. At present, six published meta-analyses have assessed the neuroimaging literature on emotion: three assessing the locationist view (Fusar-Poli et al. 2008; Phan et al. 2002; Vytal & Hamann, in press), two assessing the psychological construction view (Kober et al., 2008; Wager et al., 2003), and one assessing both (Murphy et al. 2003). One examined the brain basis of emotion perception (Fusar-Poli et al. 2008) and the remaining five looked at a mixture of both experience and perception studies (Kober et al. 2008; Murphy et al. 2003; Phan et al. 2002; Vytal & Hamann, in press;

Wager et al. 2003). The results are largely inconsistent across meta-analyses, in large part due to variations in the number and nature of studies sampled and in the statistical methods used. See Tables 4 and 5 for a summary of the relevant findings.

Testing Locationist Hypotheses

To verify a locationist account of brain-emotion correspondence, it is necessary to show that a brain area is consistent for each discrete emotion category (for discussion see Barrett & Wager, 2006; Schienle & Schäfer, 2009; Vytal & Hamann, in press). *Consistency* refers to the fact that a given brain region shows increased activity for every instance of a discrete emotion category (e.g., the amygdala shows increased activity each and every time a person experiences fear). The meta-analyses examining the locationist approach to the brain basis of emotion were largely inconclusive about the consistency with which brain regions were activated for discrete emotion categories. All meta-analyses found evidence that the amygdala is consistently active in studies of fear (although the degree of consistency were less than might be expected; see Barrett & Wager, 2006). As Table 4 indicates, this is where clear consensus across the meta-analyses ends.

In addition to demonstrating consistency, it is also necessary to show that a brain area is specific to a given discrete emotion category (for discussion see Barrett & Wager, 2006; Vytal & Hamann, in press). *Specificity* refers to the fact that a given brain region is active for instances of one (and only one) emotion (e.g., the amygdala does not show increased activity when the person is experiencing something other than fear). As summarized in Table 3, all four meta-analyses testing locationist hypotheses failed to find clear evidence for specificity in brain-emotion correspondence. First, there was lack of specificity across meta-analyses. For example, Murphy et al. (2003) found that the insula was

activated in the category "disgust" but Vytal and Hamann (in press) found it was also activated in "happiness," and "fear," and Fusar-Poli et al. (2008) found it was activated in "anger." In addition, there was lack of specificity within a single meta-analysis. For example, Murphy et al. (2003) found that the dACC was active in both "sadness" and "happiness." Vytal and Hamann (in press) found that the left amygdala was consistently activated in "happiness," "anger," "fear," and "disgust." (For additional overlapping findings, see Table 2 in Vytal & Hamann, in press and Table 3 in Fusar-Poli et al. 2008). Although Vytal and Hamann (in press) and Fusar-Poli et al. (2008) considered their findings in support of a locationist approach, this lack of specificity in the brain areas associated with discrete emotion categories is actually more consistent with a psychological construction view.

Although any meta-analysis is a herculean effort to be applauded, all four of the existing meta-analyses testing locationist hypotheses of emotion suffer important methodological drawbacks. These drawbacks very likely contributed to the inconsistency in their findings. First, these meta-analytic summaries did not account for the fact that results from imaging studies have a nested structure (i.e., specific peak activations are nested within specific contrasts are nested within specific papers). Published studies typically report a number of contrasts (e.g., anger vs. neutral, happy vs. neutral) and multiple locations of peak activation are reported for each contrast. All four meta-analyses treated the peaks within study contrasts as independent data points (when in fact peaks from the same study contrast are not independent of one another). Because individual imaging studies vary in the number of peak activations that they report for each contrast (in part based on the processing and thresholding decisions made during data analysis), small differences in the studies included in

a meta-analysis can produce large differences in the final results, making it difficult to observe consensus across meta-analyses. A number of factors influence how many peaks are reported by an individual study, including the sample size (i.e., power to find significant results), the authors' pre-processing and statistical thresholding decisions, and whether a fixed or random effects analysis was used. For example, Damasio et al. (2000) reported 15 peaks in a single sad v. neutral contrast and influenced the results over two times as much as Phillips et al. (1998b), who reported 6 peaks in a single sad v. neutral contrast. Ignoring the nested data structure allows random error to creep into the analyses, rendering results more variable, and ultimately making it even harder to find consensus across the meta-analyses. To the extent that meta-analyses have different databases, they will contain differing degrees of random noise that will distort their findings. Vytal and Hamann's (in press) database contained more than double the number of studies in Murphy et al. (2003) and a little less than double the studies in Phan et al. (2002), which also means that it contains additional (i.e., non-overlapping) random error.

Second, all four meta-analyses allowed individual peaks from both fixed and random effects analyses to contribute equally in the final empirical summary, despite the fact that those resulting from a random effects analysis are more generalizable to the population. This can also add random error to meta-analytic findings. For example, using a fixed effects analysis on data from 7 participants, Beauregard et al. (1998) reported 13 peaks from a sad v. neutral contrast. Phillips et al (1998b) reported 7 peaks from a random effects analysis of data from 8 participants. Although Phillips et al.'s (1998b) findings are, by definition, more predictive of the population, the Beauregard et al. (1998) findings would weigh more heavily in the final empirical summary because they reported almost twice as many peaks.

Testing Psychological Construction Hypotheses

Two of the three previous meta-analyses assessed a psychological construction approach to emotion in that they evaluated the brain basis of only one ingredient of this approach: core affect. Both Murphy et al. (2003) and Wager et al. (2003) compared the brain basis of different psychological models of affect (i.e., positive v. negative and approach v. avoidance). These analyses suffer from the same limitations as those meta-analyses testing the locationist emotion hypotheses and produced no consistent or specific results (summarized in Table 5).

To overcome the limitations of the prior work, the Barrett and Wager laboratories began a collaborative meta-analysis project on the brain basis of emotion in 2005. To date, we have published two chapters and one paper on the functional networks existing in the neural reference space for emotion (i.e., the brain regions consistently involved in realizing all varieties of emotional and affective states; Barrett, Mesquita, Oschner, & Gross, 2007; Kober et al. 2008; Wager et al. 2008). This neural reference space was derived from neuroimaging studies of discrete emotion and affective experience and perception published between 1990 and 2005. Our meta-analysis capitalized on the hierarchical structure of neurimaging data (peaks nested within contrasts nested within studies), thereby correcting some of the statistical limitations present in other meta-analytic studies (see section entitled *Multilevel Peak Kernel Density Analysis;* also see Kober et al. 2008; Wager et al. 2007; 2008; Kober & Wager, in press). We also included a measure of quality control and weighted studies using random effects analysis and those with larger sample sizes more heavily in the statistical summaries.

Using multidimensional scaling, we found that the neural reference space for emotion could be parsed into six distributed functional groups supporting a psychological construction approach to emotion (Kober et al. 2008). The brain areas in two of the functional groups (core limbic and lateral paralimbic groups) are consistently part of the core affect network. Aspects of two functional groups (the medial posterior and medial PFC groups) are part of the core association network. Areas in the cognitive/motor control cluster are consistent with the ingredients of executive control and language. In addition, a visual processing functional cluster was also identified as part of the neural reference space for emotion. See Figure 7. In the present paper, I build and expand upon the initial efforts published in Kober et al. First, I updated our database to include papers from 2006 and 2007. This nearly doubled the number of studies in the database. Second, I sampled only those studies from the database investigating the experience or expression of discrete emotion. This allowed me to specifically examine whether discrete emotion categories can be localized to consistent and specific locales within the neural reference space (testing a locationist approach), or whether the space can be parsed into more basic psychological processes that are common across discrete emotion categories (testing a psychological construction approach).

A Meta-analysis of Brain-Emotion Correspondence

The present meta-analysis possesses several statistical advantages, each of which is outlined below. This meta-analysis is the first to explicitly use neuroimaging data to test locationist v. psychological constructionist views of emotion. Because a locationist account has strong empirical requirements (i.e., evidence for consistency and specificity in brain-emotion correspondence), it is easier to reject and therefore could suffer a disadvantage

when compared to the more flexible constructionist account. I made several analysis decisions to adjust for this fact.

First, I exclusively sampled studies that tested a locationist account (i.e., studies that explicitly search for the brain basis of discrete emotional states) which should therefore produce the best evidence of brain localization for different emotion categories. Second, I included only those studies that utilize subtraction methods. Subtraction methods reveal "islands" of brain activation that are maximally unique to the emotion category of interest and are thus more likely to support a locationist account than an account that hypothesizes the existence of distributed functional brain networks. Third, I only included those study contrasts with a neutral comparison condition, because subtractions using other emotion categories as a comparison condition (e.g., fear v. anger or fear v. happiness) could add noise to the sample and thus obscure evidence for a locationist account.

Finally, I performed a number of statistical analyses with the potential to yield evidence in favor of a locationist account. I asked whether discrete emotion categories were consistently associated with increased activation in any brain regions more so than what would be expected by chance. In addition to this stringent test, I also reported a more lenient test which assessed whether any discrete emotion category was consistently associated with increased activation in any brain region relatively more than another (even if all emotion categories were consistently associated with an absolute increase in activity in a given brain region). Furthermore, I reversed the direction of inference and asked whether, given consistent increase in activity within a brain region across studies, people were more likely to be experiencing any one mental state (emotion category, affective state, mental response to a method variable, etc.).

Method

The Database

The present database contains 656 experimental contrasts reported in 234 PET or fMRI studies. Studies were sampled from all PET and fMRI studies related to emotion and affect that were published from 1990-2007. Papers in the present meta-analysis include those analyzed in Phan et al. (2003), Murphy et al. (2003), Wager et al. (2008) and Kober et al. (2008). Wager et al. and Kober et al. utilized a database of studies sample from 1990-2005. For the present analysis, I sampled additional papers published between 2006-2007 from Medline, PsychInfo and Google Scholar. Only studies that used subtraction analyses and that reported peak activations were included. Contrasts reporting deactivations (e.g., neutral minus fear) were not included. Studies were disqualified from the database if the method of emotion induction included fear conditioning or the administration of pain. Studies assessing the neural correlates of learning, explicit memory, priming, error processing, emotion regulation/suppression, emotion anticipation and hunger or thirst (or some other bodily sensation; e.g., stomach distension) were not included because these phenomena were not deemed to be exclusively emotional. Study contrasts assessing patients, children (<18 years of age) or older adults (> 65 years of age) were not included in the database. Study contrasts examining drug treatment, certain genotypes/phenotypes, or study contrasts where participants were arbitrarily split into groups (e.g., chocolate cravers v. non-cravers) were not included. Finally, contrasts in which ambiguous or blended emotions were perceived and contrasts assessing romantic love were excluded from the database.

Once the database was compiled, I further restricted the present analysis to studies targeting the experience or perception of discrete emotion (240 contrasts of anger, sadness, fear, disgust, and happiness from 91 studies published between 1993 and 2007). Studies of emotion experience were those that induced feelings through a range of sensory modalities including vision (e.g., pictures), olfaction (e.g., odors), memory (e.g., autobiographical recall), and imagery (e.g., simulation of scenarios). Studies of emotion perception were those that asked participants to view faces or listen to voices with emotional content. I excluded contrasts assessing more general affective states (pleasure, displeasure or arousal) to achieve the clearest test of the locationist approach to emotion. Contrasts assessing emotional states such as amusement, surprise, or contempt were not included in the present analysis because there were too few in the literature to reliably assess. Only those contrasts that used a neutral comparison condition (e.g., fear experience minus neutral) were included so that activation associated with another emotion would not influence findings (e.g., fear experience minus sadness experience). Of the published meta-analyses, only Fusar-Poli et al. (2008) and Vytal and Hamann (in press) utilized this latter criterion in defining their database; failure to restrict the analysis to contrasts with a neutral reference condition is a drawback of most other meta-analyses.

Each study was coded for methodological variables like the induction method used (e.g., vision, audition, olfaction, imagery, recall), stimuli used (e.g., faces, voices, pictures, etc), sample size and gender break-down, and the type of statistics used (fixed v. random effects). Studies were also coded for a number of psychological variables such as the states assessed (e.g., affect v. emotion), the mode (experience v. perception), the presence of cognitive load (whether participants were asked to perform another task simultaneous to

emotion experience or perception), the object of participants' evaluation (whether a participant was directed to evaluate feelings or a stimulus) and the focus of attention (whether emotion was foregrounded in attention, i.e., the judgment explicitly involved attention on emotion, or whether emotion was backgrounded in attention i.e., the judgment did not explicitly involve attention on emotion). Each study was coded twice for reliability by a set of three raters (including K.L). All raters were in perfect agreement. Once studies were coded, codes and MNI or Talairach coordinates were entered into an Excel database for each experimental contrast that passed qualification criteria. See Table 6 for studies included in the present analysis and the Appendix for references.

Multilevel Kernel Density Analysis

The database was analyzed using the Multilevel Peak Kernel Density Analysis (MKDA; Wager et al. 2007; http://www.columbia.edu/cu/psychology/tor/). The MKDA has now been used in a number of published meta-analyses of the neuroimaging literature (Barrett et al. 2007; Wager et al. 2007a; Nee et al. 2007; Etkin & Wager, 2007; Wager et al. 2008; Kober et al. 2008). The MKDA possesses a number of statistical advantages that make it superior to prior meta-analytic techniques. Whereas other analysis strategies compute the proportion of peak activations at a given location for a given category (e.g., fear), the MKDA allows reported peaks to be nested within a contrast for a given study and treats study contrast (rather than individual peaks) as the level of analysis. So, for example, when Damasio et al. (2000) reported two peaks within a 10 mm radius of one another in the ACC for a sadness experience v. neutral contrast, , the MKDA approach would count one (rather than 2) activations at that location towards the final proportion estimate. As a consequence, the statistical dependency between peaks within a study contrast is accounted for, and no

single study can contribute disproportionately to the final results. The MKDA method also weights studies by sample size and the rigor of statistical methods used in the primary analysis as a means of quality control.

A summary of the MKDA method is presented in Figure 8. The MKDA begins by first creating a map plotting the peak coordinates reported in every contrast in the database. The MKDA then convolves each reported peak with a 10-mm 3-D kernel. The effect of this step is that any two peaks in a given contrast that are within 10-mm of one another get subsumed by the same kernel and do not count twice towards the final meta-analytic summary. The MKDA then computes the proportion of contrasts in the database that activate within 10-mm of each voxel in the entire brain. This is represented as a density map, or a map of proportions. To discern which proportions would be expected at a level greater than chance, a null distribution is computed using a Monte Carlo simulation (using 18,000 iterations). The map of proportions is then thresholded at $p<.05$ using a Family-wise error correction. The result is the neural reference space, the set of brain regions consistently activated across all studies of discrete emotion experience and perception.

Analysis Strategy

I used two types of analyses to ascertain whether regions within the neural reference space for discrete emotion correspond consistently and specifically to individual emotion categories. The first set of analyses asked which brain regions were most likely to be active when a person was experiencing or perceiving a given emotion (e.g., given that a person was experiencing fear, what brain regions were active?). First, for each emotion category, I assessed whether increased activation in any brain voxels was more likely than chance to be associated with a specific emotion category using a χ^2 test. All χ^2 tests were false discovery

rate (FDR) corrected for multiple comparisons (FDR $p < .05$). I refer to these as *absolute analyses*.

In addition, I asked the more lenient question of whether certain voxels were relatively more likely to show an increase in activation for one emotion category vs. the others (even if those voxels were more likely to show an increase in activation for more than one category relative to chance). I refer to these as *relative analyses*. A significant effect here would indicate that correspondence between an emotion category and brain region is a matter of degree, not kind (e.g., the amygdala might be more likely to show an increase in activation during the perception of all emotions but this likelihood increases by a greater degree during the perception of fear). This was accomplished by contrasting the likelihood of increased activation associated with one category (e.g., fear experience) vs. all others (e.g., anger experience, disgust experience, happy experience, sad experience). I report regions surviving either a height-based threshold corrected at $p < .05$ or an extent-based threshold corrected at $p < .001$. The height-based threshold is the most spatially specific cluster-extent-based threshold for significant consistency across studies in the MKDA analysis (i.e., those regions where the peak density is high enough that the null-hypothesis chances of finding a single significant voxel anywhere in the gray matter of the brain is $p < .05$). The extent-based threshold (where the number of contiguous voxels above $p<.001$ are compared with the number of contiguous voxels expected by chance; see Kober et al. 2008) is set at the most stringent level because these clusters are the most spatially specific.

In addition to asking "given that a person is in a particular mental state (e.g., the experience of anger), which voxels show a consistent and specific increase in activation?," I also asked "given a significant increase in activation at this location, can I predict what type

of mental state the participant is likely to be in?" (e.g., given a significant increase in activation in the amygdala, is the person more likely to be experiencing anger, perceiving fear, etc.?). I used a series of exploratory stepwise logistic regressions to ascertain which emotion categories and methodological variables best predicted increases in activation in particular brain regions. Effect-coded emotion and method variables were regressed on to binary activation vectors indicating whether a given contrast in the database (e.g., fear perception) activated within 10 mm of a given brain location. Clusters were selected from the neural reference space in an a priori fashion (using the height-threshold or a stringent extent-threshold, $p<.001$) based on the predictions of locationist and constructionist hypotheses. In each brain region, I examined whether significant increases in activation were best predicted by each emotion category by modality (e.g., anger experience, anger perception, etc.), more general valence (pleasantness v. unpleasantness) and arousal (high v. low arousal), as well as induction method (visual, auditory, imagery, recall, taste, olfaction), stimuli used (films, pictures, faces, music, voice, words, personal event, food, odor, sounds), level of cognitive demand (present or absent), focus of attention (foregrounding or backgrounding of emotional content), and object of evaluation (bodily feelings or external stimulus). All variables were analyzed as main effects. Activation vectors were extracted from regions of interest within the neural reference space for discrete emotion using a Matlab script written by Tor Wager (Meta_cluster_tools.m). All logistic regressions were performed in Stata 10 software using the Logistic Regression package developed by J. Scott Long (from www.indiana.edu/~jsloc/stata) [2]

[2] Step-wise regression has limitations because it only includes the variables that best predict activation in the resulting model. To address this issue, I also ran a set of hierarchical logistic regressions that specifically tested

Predictions

The strongest support for a locationist account in the present meta-analysis would be evidence that a given emotion category was consistently and specifically associated with increased activation in a given brain area. This evidence would be observed in the absolute analyses and logistic regressions. In the absolute analysis, evidence for a locationist account would be observed if one emotion category, but no others, was associated with increased activation in a certain brain region at levels greater than would be expected by chance alone (e.g., when a person is experiencing fear, an increase in amygdala activity that is greater than would be expected by chance is observed. When a person is experiencing anger, sadness, etc., an increase in amygdala activity is not observed). In the logistic regressions, evidence for a locationist account would be observed if an increase in activity in a given brain area was predicted by one emotion category and all other emotion categories negatively predicted an increase in activity there (e.g., an increase in amygdala activity is predicted by the experience of fear but the experience of anger, sadness, etc. predict that the amygdala will be not be activated).

The strongest support for a constructionist view in this meta-analysis would be evidence that emotion categories are consistently associated with activation in brain regions more generally related to core affect, core association, language, and executive attention. Emotion categories will not be specifically related to any one brain region, however. This pattern might be evidenced in the absolute analyses, relative analyses or the logistic regressions. In the absolute analysis, evidence for a psychological construction account would be observed if several emotion categories were associated with increased activation in

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locationist hypotheses of brain-emotion correspondence. The findings of these hierarchical logistic regressions were similar to the step-wise logistic regressions reported here, so I do not include them.

a given brain region at levels greater than would be expected by chance (e.g., when a person is experiencing fear, anger or sadness, an increase in amygdala activity that is greater than would be expected by chance is observed). I might also find that brain activation is relatively more associated with one of these emotion categories than the others in the relative analyses (e.g., when a person is experiencing fear, there is a significantly greater increase in amygdala activity than when a person is experiencing anger or sadness). This relative difference would imply that those mental states differentially recruit a certain psychological ingredient (e.g., the experience of fear involves the processing of salient information relatively more so than the experience of anger or sadness). Finally, evidence for a psychological construction account would be observed in the logistic regressions if an increase in activation in a given brain area was best predicted by several different emotion categories, methods variables or more general psychological variables (cognitive load, arousal, visual stimuli) (e.g., an increase in amygdala activity is predicted by the experience of fear and the experience of anger or by high arousal stimuli and visual stimuli more generally).

Results

The Neural Reference Space for Discrete Emotion

Even with the addition of new studies and different inclusion criteria (i.e., only discrete emotion contrasts with a neutral reference condition), the neural reference space for discrete emotion (presented in Figure 9) remained very similar to the one published in Wager et al. (2008) and Kober et al. (2008). Regions surviving the height-based and most stringent extent-based thresholds are presented in Table 7. Consistent with previous findings, the neural reference space included limbic and paralimbic areas involved in a core affect network (Barrett, Mesquita, et al. 2007; Barrett & Bliss-Moreau, 2009), areas within the core

association network (e.g., Buckner et al. 2007; Schacter et al. 2007; Bar, 2009), lateral prefrontal areas involved in language and executive control and exteroceptive sensory processing areas. Consistent with a psychological construction approach, these brain regions are implicated in many other psychological phenomena, including the representation of interoceptive states, memory, categorization and prospection, semantic retrieval and language processing, working memory, executive control, and sensation and perception. Figures depicting the neural reference spaces for the experience and perception of anger, disgust, fear, happiness and sadness are presented in Appendix B.

Testing Brain-Emotion Correspondence

Table 8 presents the brain regions that were consistently associated with a given discrete emotion category at levels that would be expected greater than chance (*absolute analyses*). Table 9 presents the brain regions that consistently showed greater activation for one discrete emotion category relative to all others (*relative analyses*). Table 10 presents the emotion experience and perception variables and method variables that were most likely to be associated with activation in a given brain area (*logistic regressions*). For each brain area, the overall model fit and significance are listed, as are the β, odds, and statistical significance of each variable's ability to predict activation at the brain locale. In the logistic regressions, the odds ratio is the likelihood that activation of a brain location is associated with a variable (e.g., that amygdala activity is associated with fear perception) divided by the probability that is not associated with a variable (e.g., that amygdala activity is not associated with fear perception). An odds ratio can also be represented as a percent increase (over the mean likelihood of any variable in the database activating there), which are referred to in Figures 10-18 (this is calculated by subtracting 1 from the odds ratio and multiplying by 100).

The amygdala- fear hypothesis. The amygdala was consistently, but not specifically, associated with the perception of fear. The perception of fear was associated with increased activation in bilateral amygdala (611 voxels) relatively more so than the perception of any other emotion (Table 9). Yet, the perception of fear was not associated with amygdala activation more than would be expected by chance alone, suggesting that increases in amygdala activation during the perception of fear are a matter of degree and not kind (i.e., the amygdala is not specific to fear). The experience of disgust, on the other hand, was consistently associated with increased activation in 111 voxels within the amygdala more so than would be expected by chance (Table 8). The experience of disgust was also relatively more likely to be associated with increased activation in 247 voxels of bilateral amygdala than were the experience of "anger," "fear," "happiness" and "sadness."

The amygdala not only showed a consistent increase in activation during the perception of fear and the experience of disgust in the MDKA, but these categories also predicted amygdala activation in the logistic regressions. See Figure 10 and Table 10. When a significant increase in L. amygdala activation was observed, participants were more likely to be experiencing disgust or perceiving fear than to be in any other mental state. Furthermore, during this increase in amygdala response, participants were unlikely to be experiencing fear or perceiving anger. The fact that other emotion states (e.g., the perception of disgust) did not positively or negatively predict increased L. amygdala activity indicates that the L. amygdala showed an increase in activity during some instances of these categories, but not others (e.g., the L. amygdala showed increased activity during some instances of perceiving disgust but during other instances it did not). When a significant increase in R. amygdala activation was observed, participants were most likely to be experiencing or perceiving any

emotion category that was high in arousal (e.g., the experience and perception of fear, anger and disgust).

By any set of criterion, then, the amygdala cannot be considered the brain locus of fear. Instead, the findings appear to be more consistent with the psychological construction hypothesis that the amygdala is involved in the brain response to salient stimuli. Activation within the R. amygdala was best predicted by arousal, a property of the psychological ingredient of core affect (Barrett & Bliss-Moreau, 2009; Russell & Barrett, 1999). Activation in the L. amygdala was not predicted by arousal more generally because the experience of fear and the perception of anger—two of the six types of contrasts containing high arousal emotional content in the database—consistently failed to be associated with increased activity in the L. amygdala. Yet, the fact that L. amygdala activity was associated with the experience of disgust and the perception of fear is still consistent with the idea that the amygdala responds to high arousal affect. Over a third (34.8%; 15/43) of the study contrasts in the database assessing disgust experience presented participants with images that were highly arousing (i.e., containing contamination, mutilated body parts, maggots, etc.) and novel (i.e., infrequently experienced in the industrialized world). Similarly, over ninety percent (92.9%; 53/57) of the study contrasts assessing fear perception in the database used fearful faces, which are experienced as highly arousing (e.g., Russell & Bullock, 1986) and are relatively novel and unfamiliar to college students (Whalen et al. 2001) (who are typically the participants in neuroimaging studies of these sort). Consistent with the psychological constructionist argument that the amygdala is implicated in the brain response to salient exteroceptive sensory stimuli, the L. amygdala was also less likely to be active when emotion experience was induced via recall of a personal event and mental imagery (see Figure 10).

The insula-disgust hypothesis. The anterior insula was consistently, but not specifically, associated with the perception of disgust. The experience of disgust was not associated with consistent activation of the anterior insula in either the absolute or relative analyses. The perception of disgust was consistently associated with increased activation in four voxels in the right anterior insula more so than would be expected by chance (R. a. ins) (Table 8). The perception of disgust was also relatively more associated with activation in 252 voxels in the R. a. ins. than were other emotions (Table 9). The experience of anger, on the other hand, was consistently associated with increased activation in one voxel of the left anterior insula (L. a. ins) more so than would be expected by chance. This effect was also observed relative to the experience of other emotions in 2 voxels of L. a. ins (Tables 8 and 9).

The L. a. ins not only showed a consistent increase in activation during the experience of anger in the MKDA, but this category also best predicted L. a. ins. activation in the logistic regressions (see Figure 11; Table 10). The cluster extracted from the height-based threshold actually contained some voxels from the lOFC (see Table 10 note) making these findings consistent with the OFC-anger hypothesis. To examine whether the experience of anger predicted activity in the insula specifically, emotion and method variables were regressed on a 193-voxel cluster in L. mid-insula (extracted from the extent-based neural reference space, $p<.001$). Consistent with the findings in L. a. ins, activation increases in this L. mid-insula cluster were also more likely to be associated with the experience of anger than any other variable (see Table 10). Finally, the variables predicting activation in R. a. ins were assessed. Consistent with a psychological construction account linking the insula to the representation of bodily states, increased activation in R. a. ins was most likely to occur in tasks where participants were asked to explicitly evaluate their feelings

(regardless of the emotion category experienced). Taken together, these findings suggest that the relationship between disgust perception and activation in the insula is asymmetrical: when a person is perceiving disgust, it is likely that their R. a. ins will be active. But when R. a. ins. activity is observed, it is not necessarily the case that a person will be perceiving disgust.

The present findings suggest that the a. ins is involved during the perception of disgust but not the experience of disgust, providing partial support for the insula-disgust hypothesis. These findings do not rule out a psychological construction account of insula function, however; disgust perception might preferentially activate the insula because people are more likely to simulate visceral states (like those associated with the gut and food rejection) when perceiving facial behaviors characterized by a wrinkled nose and curled lip (i.e., oral revulsion; Angyal, 1941 see Rozin, Haidt, & McCauley, 2008; e.g., von dem Hagen et al. 2009). Indeed, I found evidence consistent with this psychological construction account: increased activity in R. a. ins. was best predicted by a focus on feelings across all studies of emotion.

The meta-analytic findings also suggest that the insula might be important to the experience of anger, although they are not strong enough to form the basis of a new locationist hypothesis because only 1 voxel in the left a. ins was associated with the experience of anger more so than would be expected by chance. More likely, the experience of anger is associated with activation in a whole swath of cortex in the left frontal lobe (see findings below for the L. lOFC, VLPFC and DLPFC). This interpretation is consistent with electrophysiological findings that approach (vs. avoidance) motivation is relatively left-lateralized (Fox, 1991; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). It also fits

with characterizations of anger as an appetitive, approach-related emotion (Carver & Harmon-Jones, 2009) and findings that anger corresponds to electrical activity in left prefrontal cortex (e.g., Harmon-Jones & Sigelman, 2001).

The orbitofrontal cortex-anger hypothesis. The OFC was somewhat consistently, but not specifically, associated with the experience of anger. Neither the experience nor perception of anger consistently activated the OFC in the absolute or relative analyses. Instead, the experience of disgust was consistently associated with increased activity in 167 voxels in L. lOFC (BA 11) (both at levels significantly greater than would be expected by chance and to a significantly greater degree than any other emotion category; Tables 8 and 9). The perception of disgust was consistently associated with increased activation in 8 voxels in R. lOFC (BA 47) more so than would be expected by chance. The spatial extent of this effect increased when the perception of disgust was compared to the perception of other emotions (to cover a 37-voxel cluster extending into R. a. ins.).

The OFC not only showed a consistent increase in activation during the perception of disgust in the MDKA, but this category predicted OFC activation in the logistic regressions. When a significant increase in R. lOFC activation was observed, participants were most likely to be perceiving or experiencing disgust than to be in any other emotion state (see Figure 12 and Table 10 for findings in a separate cluster of R. lOFC). Consistent with prior findings linking the experience of anger broadly to left prefrontal cortex, increased activation in L. lOFC (BA 47) was more likely to occur during the experience of anger than any other emotion category. Finally, increased activation in clusters of L. and R. lOFC was predicted by auditory stimuli (i.e., sounds, voices), picture stimuli, and high arousal affect more generally, consistent with the psychological construction OFC-context-based behavior

hypothesis (in which the OFC integrates sensory information from the world and body to guide behavior).

Anterior cingulate cortex-sadness hypothesis. The meta-analytic evidence is not consistent with the hypothesis that the ACC is the brain basis of sadness. No single emotion category was associated with consistent activation in vACC or dACC at levels greater than would be expected by chance. This means that increased activation in the ACC occurred in all emotion categories. Two emotion categories were associated with activation in dACC relatively more than other emotion categories, however. The perception of sadness was consistently associated with increased activation in a single voxel in dACC (BA 32) to a greater degree than was the perception of other emotions (Table 9). The perception of fear was consistently associated with increased activation in 186 voxels in dACC (BA 24) to a greater degree than the perception of other emotions (Table 9). Consistent with a constructionist ACC-conflict hypothesis, the logistic regressions revealed that increased activation in two clusters in the dACC (BA 32) was more likely to occur during tasks containing cognitive load than during any other emotion state or in response to any other method variable. See Figure 13 and Table 10.

By any set of criterion, then, the ACC cannot be considered the brain locus of sadness. Instead, the findings appear to be more consistent with the psychological construction hypothesis that the ACC is involved in the brain response to conflicting sensory arrays. Although I found evidence that the perception of sadness was relatively more associated with a single voxel in the dACC than was the perception of other emotions, this is not sufficient support to conclude that the ACC is the brain basis of sadness. Instead, the findings are more consistent with the psychological construction hypothesis that the dACC

is involved in executive control processes during conflict monitoring and response. Consistent with this hypothesis, dACC was consistently associated with the perception of fearful faces, perhaps because they are unfamiliar, hard to understand (e.g., Whalen et al. 2001), and thus require executive attention to reduce ambiguity.

DMPFC, MTL and Retrosplenial cortex. Regions of the core association network such as DMPFC, MTL and retrosplenial cortex (Buckner et al. 2008) were part of the neural reference space for discrete emotion. My findings are the first to indicate that the core association network is integral in the experience and perception of discrete emotions, and are consistent with the hypothesis that prior episodic experience helps shape experienced or perceived affect into meaningful instances of emotion (Barrett, 2006 b; 2009a, b).

Areas in the core association network were associated with discrete emotion categories with differential consistency, in line with the constructionist view that the relative weighting of ingredients differs across mental states. The experience of sadness was associated with consistent activation in only 1 voxel in DMPFC (BA 9) (both significantly more than would be expected by chance and to a significantly greater degree than the experience of other emotions) (Tables 8 and 9). The experience of happiness was associated with consistent activation in a 324-voxel cluster in DMPFC (BA 9; abutting BA 32) and in a 379-voxel cluster in retrosplenial cortex (BA 30) relatively more than were other emotion categories (Table 9). Additionally, the logistic regression findings revealed that increased activation in a cluster of DMPFC (extending into dACC) was more likely to occur during the perception of disgust than any other variable (see Figure 14 and Table 10). The cluster of DMPFC associated with disgust perception in the logistic regressions overlapped with the clusters associated with the experience of happiness and sadness in the MKDA, suggesting

that the DMPFC is broadly implicated in a number of emotion categories. Consistent with the role of core association in simulating episodic experience (see Buckner et al. 2007), increases in activation in a separate cluster in DMPFC (BA 9) were more likely to occur during emotion inductions using recall and films than during any other emotion state or experimental method (see Table 10). The fact that films were associated with activation in the same cluster as recall suggests that inductions using film recruit a form of deep simulation that is involved in recollecting one's own experiences.

Several emotion categories were associated with consistent activation in the MTL. The experience of sadness was consistently associated with increased activation in 10 voxels in L. dorsal entorhinal cortex (EC) to a degree greater than would be expected by chance. The experience of sadness was also consistently associated with increased activation in broader cluster of EC (19 voxels) when compared to the experience of other emotions (Tables 8 and 9). The perception of sadness was consistently associated with increased activation in one voxel in the L. hippocampus more so than the perception of other emotions. The experience of disgust was consistently associated with increased activation in a 1-voxel cluster in left dorsal EC (BA 34) (both significantly greater than would be expected by chance and to a significantly greater degree than other emotion categories). The perception of anger was associated with increased activation in 23 voxels within R. dorsal EC (BA 34) to a degree significantly greater than chance. Relative to other emotions, this cluster expanded to 27 voxels. Finally, the perception of fear was consistently associated with increased activation in one voxel in R. dorsal EC more so than any other emotion category. This finding was corroborated by the logistic regressions. See Figure 15 and Table 10. Increased activation in bilateral EC and the L. hippocampus was more likely to be associated

with the perception of fear than any other variable. This finding is consistent with the constructionist hypothesis that the amygdala facilitates increased sensory processing and encoding of uncertain information in memory. In line with the constructionist hypothesis that the core association network supports categorization during the perception of all affective facial behaviors, I also found that increased activity in the R. hippocampus was likely to occur any time a person perceived emotion in the face or voice of another person.

Anterior temporal lobe and VLPFC. Areas in the distributed network for language like the ATL and VLPFC were part of the neural reference space for discrete emotion. The fact that the ATL and VLPFC were consistently activated across studies of emotion is consistent with the psychological construction hypothesis that language-based categories support the experience and perception of emotion (Barrett, 2006b, 2009a; Barrett, Lindquist et al. 2007). No single emotion category activated the ATL (either significantly more than expected by chance or significantly more than other emotion categories), suggesting that the ATL is ubiquitously active across all emotion categories. The perception of disgust was the only category to be associated with a consistent increase in activation in R. VLPFC (BA 44; 66 voxels) that was significantly greater than what would be expected by chance (Table 8). When compared to the perception of other emotions, this cluster expanded to 71 voxels (Table 9).

Consistent with the MKDA findings, increased activation in L. and R. VLPFC (BA 44) was more likely to be associated with the perception of disgust than any other emotion state or method variable in the logistic regressions. Increased activation in the L. ATL and L. VLPFC (BA 44) was more likely to occur during the experience of anger than any other emotion category, in accordance with the prior findings that the experience of anger recruits

left prefrontal cortex (see *The Insula-Disgust Hypothesis* and *The Anger-OFC Hypothesis*). See Figure 16 and Table 10.

Finally, the logistic regressions revealed that increased activation in L. VLPFC (BA 44) was likely to occur in tasks in which participants explicitly attended to emotional information (Figure 16). That the VLPFC would be active when people were aware of affective feelings or perceptions is consistent with the psychological construction hypothesis that language is brought to bear during the experience of emotion by making core affect meaningful (e.g., Lindquist & Barrett, 2008), and during the perception of emotion by making affective facial actions meaningful (e.g., Barrett & Kensinger, in press). Increased activation in L. VLPFC (BA 44) was also likely to occur when participants perceived face stimuli, providing additional support for the idea that language is brought to bear when perceiving emotion in faces (See Table 10 for other variables associated with increased activation in the L. VLPFC). Increased activation in the R. ATL occurred during tasks in which participants were asked to evaluate a stimulus, also suggesting this region's role in language and categorization more generally (Figure 17 and Table 10).

VLPFC and DLPFC. Areas in the distributed network for executive control such as VLPFC and DLPFC helped constitute the neural reference space for discrete emotion, consistent with the psychological construction hypothesis that executive control modulates how psychological ingredients influence one another in the emergence of emotion (Barrett, 2009a, b; Barrett, Tugade & Engel, 2004). The perception of anger was consistently associated with increased activation in 27 voxels within L. DLFPC (BA 9) and 137 voxels within R. DLPFC (BA 9) (both at levels significantly greater than would be expected by chance and to a significantly greater degree than in the perception of other emotions)

(Tables 8 and 9). This finding was replicated in the logistic regressions, where increased activation in L. BA 9 was more likely to occur during the perception of anger than during any other emotion state or method variable. The perception of anger was also more likely to be associated with increased activation in L. BA 46 than any other variable (see Table 10).

Consistent with the DLPFC's known role in executive function, the logistic regressions revealed that increased activation in R. DLPFC (BA 9) was most likely to occur in contrasts where participants were asked to explicitly evaluate stimuli (Figure 18 and Table 10). Increases in L. BA 9 and L. BA 46 were also particularly likely to occur in tasks where participants were asked to pay attention to emotional information (Figure 18 and Table 10).

Periacquiductal Grey (PAG). I did not have strong a priori hypotheses about the PAG's role in discrete emotion states. The PAG surrounds the cerebral aqueduct and is the location of ascending fibers of the spinothalamic and spinomesencephalamic tract. It is implicated in visceromotor control during goal-directed behaviors (see Kober et al. 2008 for a discussion) and so is generally implicated in instantiating core affective states. Consistent with the idea that the PAG is generally involved in instantiating core affect and hence involved across all emotional states, the PAG did not correspond to any single emotion category more than another (although the experience of sadness was associated with consistent activation in 1 voxel within ventral PAG see Tables 8 and 9). The logistic regressions demonstrated that increased activation in the PAG was particularly likely to occur during the experience or perception of any high arousal emotional state (see Table 10). Consistent with this finding, increased activation in a different cluster in the PAG was likely to occur during the experience of fear (see Table 10). All high arousal emotion categories in the database were

also unpleasant (e.g., fear, anger, disgust) so these findings are consistent with prior meta-analytic findings linking the PAG to unpleasant affect (Wager et al. 2008).

Visual Cortex. I did not predict a priori that brain regions involved in processing exteroceptive sensory information would help comprise the neural reference space for discrete emotion. Yet regions of visual cortex were some of the most frequent to appear in the MKDA findings (replicating several recent meta-analyses; e.g., Fusar-Poli et al. 2008; Kober et al. 2008; Vytal & Hamann, in press). Consistent activation of visual cortex was associated with unpleasant, highly arousing emotions, consistent with growing functional evidence that affect modulates vision (e.g., Damaraju, Huang, Barrett & Pessoa, 2009; Mickley Steinmetz & Kensinger, 2009; Padmala & Pessoa, 2008; Phelps et al. 2006) and anatomical tracer studies demonstrating connectivity between the amygdala, OFC, and various aspects of visual cortex (e.g., Barbas, 1995; Carmichael & Price, 1995b; Freese & Amaral, 2006; for reviews see Duncan & Barrett, 2007; Barrett & Bar, 2009). It is beyond the scope of this paper to discuss these findings in detail here, but in brief, I found that the experience of fear, the perception of anger, and the experience of disgust were consistently associated with activity in regions of visual cortex ranging from V2 to visual association cortex (BAs 37, 21) (see Tables 8 and 9). Such activation was not solely predicted by the stimuli used in the experimental studies, however, because emotion categories significantly predicted activation in visual cortex in the logistic regressions as well. See Table 10 for variables that activated the visual cortex and Lindquist, Wager, Bliss-Moreau, Kober & Barrett (in prep) for a discussion.

Conclusion

Over a century ago, William James argued that "sensational, associational, and motor elements are all that [the brain] need contain" to produce the variety of mental states that constitute the human mind (cf., p. 473, James, 1890/1998). James believed that emotions, thoughts, and memories were commonsense categories whose instances do not require special brain centers. James' view foreshadowed modern psychological constructionist views of the mind and the findings of this meta-analytic review, which are largely consistent with a constructionist approach. This meta-analytic review suggests that emotion categories are not natural kind categories respected by the architecture of the brain.

In keeping with James' predictions, this meta-analytic review did not find strong evidence for any locationist hypothesis of brain-emotion correspondence. In all instances where a brain region was consistently active during a discrete emotion category (e.g., the amygdala in fear perception), it was not specifically so, failing to support a key locationist assumption. Instead, brain regions were active across many emotion categories. Often, these regions were consistent with the psychological ingredients that we have written about and hypothesized in other papers (e.g., Barrett, 2009a; Barrett & Lindquist, under review). For instance, across emotion categories there was consistent activation in key nodes in networks involved in uncertainty detection, representation of bodily states, conflict monitoring and response, and using sensory information to make context-based decisions. These mechanisms together help to realize core affect. There was also consistent activation in the brain regions implicated in core association (simulation of prior episodic experiences), language (representation and retrieval of semantic concepts), and executive control (volitional attention and working memory) across emotion categories. The absolute association between emotion categories and activity in certain brain regions was minimal—in

most cases, activity in only 1-20 voxels associated with an emotion category more than would be expected by chance. Of course, there were sometimes relative differences in the degree to which brain regions were involved in realizing different categories of subjective experience or perception. These relative differences perhaps point to differences in the contents of these mental states (e.g., anger involves approach motivation; disgust perception involves the experience of bodily activation; fear perception involves detection of salient stimuli).

Kober et al. (2008) hinted at the existence of psychological ingredients in the construction of emotion, but the current paper is the first to show that ingredients like core association, language, and executive control are consistently activated in neuroimaging studies despite a range of methodological variables, not because of them. This important finding lends further support to our psychological constructionist view. For instance, consistent activation in the amygdala was predicted by high arousal affect. Consistent insula activation was predicted by a focus on feelings. Consistent OFC activation was predicted by exteroceptive auditory and visual information. Consistent dACC activation was predicted by cognitive load.

I also found that activation in visual cortex was predicted by the experience and perception of unpleasant, highly arousing emotions. This finding, along with the fact that visual cortex was one of the most frequently activated brain regions in the MKDA, might be grounds for adding another basic psychological ingredient (e.g., "exteroceptive sensory perception") to a psychological constructionist theoretical framework. Regions in left lateral prefrontal cortex were also consistently observed in anger experience, suggesting that a network for approach-related motivation might constitute another aspect of core affect.

Although it is possible that the left frontal lobe (including the anterior and mid-insula, VLPFC, DLPFC and OFC), is generally the brain seat of anger, it is unlikely that this heterogeneous group of brain regions would be the origin of a single psychological category.

Alternate Interpretations

Of course, there are alternate explanations for why this meta-analysis did not reveal strong evidence in support of a locationist framework. It is possible the level of psychological analysis used was too gross. The common sense categories of emotion that we colloquially use in English ("fear," "anger," "sadness," etc.) might be too heterogeneous to support scientific induction, and more subordinate level categories might better correspond to consistent and specific locations in the brain. For instance, it is possible that states of "anxiety" and "nervousness" have a clear correspondence in discrete brain regions but scientists have thus far been unable to discern this because these states are classified together under the superordinate category of "fear." While this is always a possibility (and is empirically testable, although not with the present database), the present findings suggest that it is unlikely that any complex emotion categories correspond well to specific locales of brain anatomy. If the English terms "fear," "anger" and "sadness" don't correspond to discrete brain circuitry, then it seems unlikely that English terms like "anxiety," "rage," and "despair" would. Put another way by neuroscientist Eliot Valenstein following his electrical stimulation studies of the human brain: "It would be very surprising indeed if the brain were organized into spatially discrete units that conform to our abstract categorizations of behavior" (Valenstein, 1973, p. 142-143).

It is also possible that I did not find strong support for locationist accounts because my level of neuroscientific analysis was too broad. Neuroimaging is only capable of

measuring gross blood flow to a given brain area during a psychological state. It thus remains possible that scientists might find functional specialization for emotion at a more basic level of analysis (e.g., at the level of cortical columns of neurons). Yet it is also possible that localizations for complex psychological categories would be even less likely at this more basic level of analysis. The same neurons very often participate in different neural assemblies producing different functions (referred to as *neural degeneracy* by Edelman, 1989; for a discussion of the mechanism by which this might occur, see Izhikevich et al. 2003). This makes strong locationist interpretations of brain function, even at the level of the cortical column, unlikely. If localization at the level of cortical columns exists, is likely to serve more basic mechanisms that help constitute numerous psychological processes (for a discussion, see Barrett, 2009b). This idea, too, is consistent with the psychological construction ontology of brain-emotion correspondence.

A final explanation for the present findings is that I failed to locate the discrete brain basis for emotions because what actually exist are discrete circuits for behaviors that correspond to specific discrete emotion categories. There is certainly well-documented evidence for the neural circuitry underlying specific actions like vocalizations (see Jürgens, 2009), maternal behavior (see Numan, 2007), freezing (see Fanselow & Poulos, 2005), startle (see Davis et al. 2008; Lang et al. 2000), and reward (see Berridge & Kringelbach, 2008; Shultz, 2006) (just to name a few). Yet, animals produce behaviors in a context-driven fashion, so many different behaviors can be associated with a given discrete emotion category. For instance, rats freeze in the face of a threat, but they also aggress, run away, or sometimes do nothing. Even if certain action patterns are more likely to occur in certain emotion categories, it would be philosophically and scientifically problematic to reduce the

range of human emotion to a set of fixed action patterns because a large number of human mental states would go unexplained (cf. Barrett, Lindquist et al. 2007). For instance, freezing might be more likely to occur in "fear" than in "sadness," or "happiness," but this does not mean that the neural circuitry for freezing is the neural circuitry for "fear." If researchers defined "fear" as only the tendency to freeze in the face of a predator, then times when a rat flees, attacks, kicks bedding at the predator or avoids an unknown corner of a maze would not be considered "fear." And times when humans avoid a dark alley, bungee jump, remember the events of September 11th, lock the door at night, or password protect their online bank accounts could not be considered part of the category "fear."Although these three alternate conventions for mapping the mind to brain are theoretically possible, they do not seem plausible based on the current literature.

Mapping Emotion to Brain

As neuroscientific methodologies progress, it will become increasingly imperative that scientists formulate a viable framework for mapping emotions to the brain. The present findings strongly suggest that a psychological construction view can satisfactorily describe the brain basis of emotion. Locationist views might be deeply entrenched in both our scientific and commonsense explanations of emotion, but they do not match the scientific evidence. In this regard, the neuroimaging findings are consistent with other objective measures used in the study of emotion which fail to consistently distinguish between discrete emotional states (for a review, see Barrett, 2006a; Barrett Lindquist et al., 2007). Nonetheless, locationist approaches to emotion have dominated research and theorizing about emotion in psychology over the last century for several reasons.

First, the idea that emotions are coded in discrete ways in our brain fit with our most deeply held beliefs about emotions. People experience emotions as discrete and bounded events. That we *experience* emotions as states that wash over us, occupying our consciousness, and commanding our bodies, is not in doubt. Nor is it debated that people automatically and effortlessly perceive emotions on the faces and bodies others, in animals, or in inanimate objects. But rarely does the experience of a phenomenon shed light on how it is caused. Few scientists in this post-Cartesian era would deny that emotions are "created" in the brain or that emotions correspond to observable brain activity. Yet, this does not mean that we have neural apparatus for triggering "anger" when we're offended or for detecting "fear" when someone moves their face a certain way. Of course, the fact that people take the contents of their experiences as evidence of the processes that produce them is not unique to emotion. Consider, for example, the science of taste. Because human beings can readily distinguish between sweetness, sourness, saltiness, and bitterness, researchers have long assumed that there are four basic tastes, with a fifth discovered later (see Lindemann et al. 2002)[3]. Over a century of research has followed suit, searching for the anatomical, neurochemical and neural correlates of these tastes (see Erickson, 2008). As it turns out, the evidence for these four basic tastes might not be as strong as scientists once thought (see Erickson, 2008).

The second reason that locationist views of emotion have dominated over psychological construction approaches is historical. As a scientific discipline, psychology began as an amalgam of philosophy, physiology and neurology. In particular (and perhaps not surprisingly), the methods of scientific induction used in early neurology have most

[3] Umami, a taste corresponding to foods rich in glutamate (e.g., fermented and aged foods), has recently been proposed as the fifth basic taste.

impacted modern-day neuroscience. This has not been without consequence for the science of emotion, however. Early neurological investigation of brain-mind correspondence derived hypotheses from double dissociations observed between mental states (e.g., emotions) and brain anatomy (e.g., the amygdala) following lesions. The result was locationist hypotheses of brain function: because a given brain region was damaged, a certain psychological function was impaired. I am not trying to argue that this research has not been essential to progress in neuroscience. Quite to the contrary—research in neurology during the 19th and 20th centuries literally formed the foundations of modern neuroscience (e.g., in the study of affect; e.g., Adolphs et al. 1994; Bechara et al. 1996; Harlow, 1848/1999; and memory; Corkin, 1965; 1984; Schacter et al. 1982) and has been invaluable in scientists' quest to understand how the brain creates the mind. Studies assessing patients with lesions (e.g., Clark et al. 2008; Khalsa et al. 2009; Koenigs et al. 2007) and neurodegenerative diseases (such as Alzheimers disease; e.g., deIopolyi et al. 2007; frontotemporal dementia; e.g., Chow et al. 2009; Rosen et al. 2004; and semantic dementia; e.g., Gorno-Tempini et al. 2004 to name a few) are still making great contributions to scientists' knowledge of brain function. The point is that the locationist logic used in lesion studies is not without consequence for how researchers theorize and even construct studies assessing brain-emotion correspondence.

Finally, researchers might assume that emotions are reified in the brain because of the tenets of classical measurement theory (for a discussion, see Barrett, 2006a). In classical measurement theory, a set of correlated, measurable outcomes are assumed to derive existence from an abstract, latent category that is not directly measurable (Bollen & Lennox, 1991). Using this logic, emotions are treated as latent variables that are supposed to give evidence of their existence in hypothesized correlations between facial expressions, feelings,

behaviors, and physiological patterns (Barrett, 2006a). In neuroscience, this translates into the assumption that there is a neural center from which these diagnostic emotion patterns issue. The idea that an emotion stems from a neural essence is also known in philosophy as the tendency to define emotions as a natural kinds by homology (see Barrett, 2006a). The idea that emotions produce correlated outcomes is known in philosophy as the tendency to define emotions as natural kinds by analogy (see Barrett, 2006a) and as it turns out, there is not good evidence for this assumption either. Emotion-specific patterns in emotion perception (for a review, Russell, 1994; Russell et al. 2003), psychophysiology (Cacioppo et al., 2000), electrical stimulation of the mammalian brain (Valenstein, 1973), or other measurement modalities used in the science of emotion are elusive (for a review, see Barrett, 2006a; Barrett, Lindquist et al., 2007; Mauss & Robinson, 2009).

Despite the field's emphasis on locationist views (that inspired most of the experiments used in this meta-analysis), the bulk of the empirical evidence is more consistent with the hypothesis that emotions consist of basic ingredients that map to broad-scale networks in the brain. As a consequence, to fully explore the power of a psychological construction approach in future research, researchers might combine traditional neuroimaging techniques with methods that make network-based assumptions (e.g., Multivoxel Pattern Analysis; Haxby et al. 2001; Multivariate Partial Least Squares Analysis; McIntosh et al. 1996) about brain function (see Schienle & Schafer, 2009 for additional analysis approaches).

Furthermore, the present findings suggest that researchers need to carefully consider the behavioral tasks that they ask participants to perform in neuroimaging studies. The findings that were most consistent with locationist accounts in the present meta-analysis

were found in studies of perception, not experience. In the study of emotion, there has been a tendency to make inferences about the universal production of emotion based on the ease and (somewhat bounded) universality in perceiving emotion (e.g., Ekman & Friesen, 1971). Indeed, many neuroimaging studies rely on the perception of faces to yield evidence for the brain basis of a category more generally (e.g., studying the brain basis of perceiving "fearful" faces will yield the more general brain basis of "fear"). Yet the present findings, along with prior meta-analytic findings from our project (Wager et al. 2008) suggest that the experience and perception of emotions recruit different aspects of the neural reference space for emotion in differing degrees. It is important not to assume that identifying the circuitry for perceiving an emotion necessarily implies anything about the circuitry for producing emotion when either designing studies or interpreting findings.

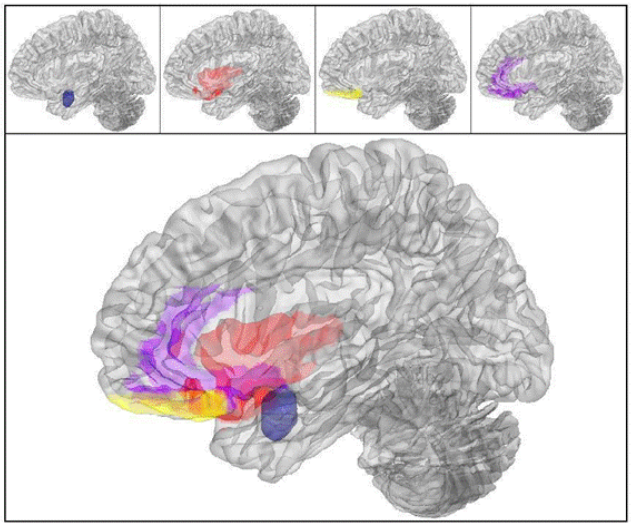
Unifying the Mind

If a psychological construction approach to the mind is right, then some of psychology's time honored distinctions become phenomenological ones, potentially impacting the scientific understanding of psychological phenomena ranging from decision-making, attention, visual perception, mental illness, and perhaps even consciousness more generally. If phenomenological categories like "anger," "sadness," and "fear" are not respected by the brain, then it is possible that phenomenological categories like "cognition" and "emotion" are not respected by the brain either (for a discussion, see Barrett, 2009b; Duncan & Barrett, 2007; see Pessoa 2008 for a similar view). According to a psychological construction view of the mind, "emotion" does not influence "cognition" during decision making as one pool ball exerts influence on another. Instead it suggests that core affect, core association, language, and executive control processes (and perhaps others) unite to form a

behavioral outcome. If this is the case, then scientists might not assume that "emotion" and "cognition" battle it out in the brain when a person makes the moral decision to sacrifice one life to save many (e.g., Greene et al. 2004), or that consumer decisions are predicated on competing affective and rational representations (e.g., Knutson et al. 2007). Instead, researchers might assume that affect and executive control are merely different sources of attention in the brain rather than processes that differ in kind (see Barrett, 2009b; Vuilleumier & Driver, 2007). Feeling and seeing might not be as distinct as typically assumed (Barrett & Bar, 2009; Duncan & Barrett, 2007). Even conceptions about "internal" v. "external" processing begin to break down when we take into account the fact that "internal" ingredients like affect and core association shape the very way in which exteroceptive sensory input is realized as sensations by the brain (Bar, 2009; Barrett & Bar, 2009). A psychological construction framework of the mind thus begins to break down the most steadfast assumptions of our commonsense categories. In so doing, it charts a different but exciting path forward for the science of the mind.

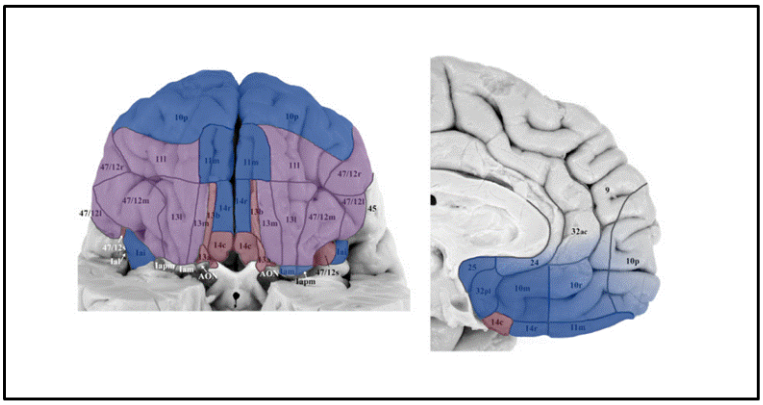
Figures

Figure 1. Brain areas in Locationist accounts of emotion



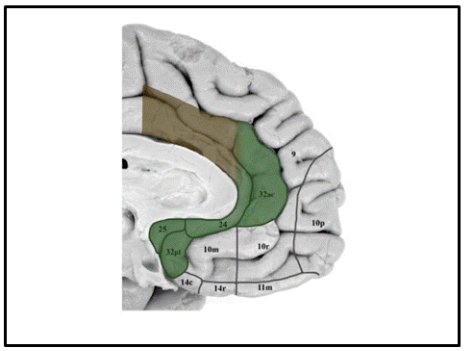
Regions include the amygdala (blue, inset 1), insula (red, inset 2), OFC (yellow, inset 3), and ACC (purple, inset 4).

Figure 2. The anatomy of the OFC



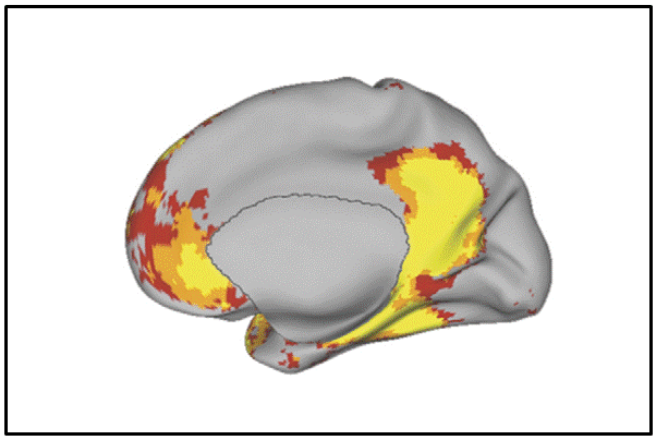
Lateral (purple) and medial (blue, pink) networks in the OFC (figure from Barrett & Bar, 2009; adapted from Ongur et al. 2003)

Figure 3. The anatomy of the ACC.



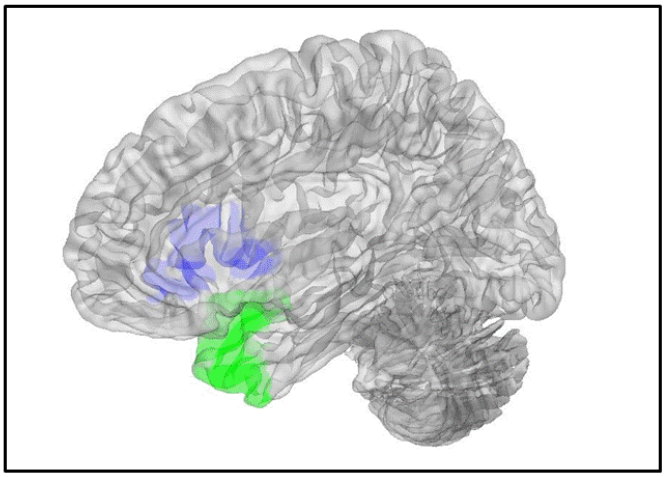
vACC (green) and dACC (brown) in the ACC (adapted from Bush et al. 2001)

Figure 4. The core association network



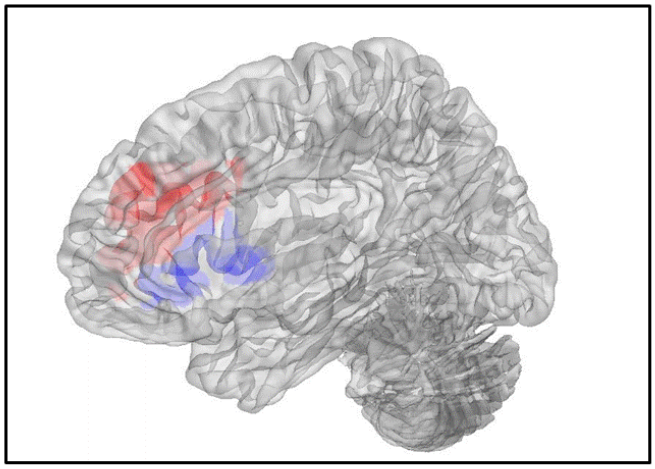
Areas involved in the core association network, including VMPFC, DMPFC, MTL, PCC/Retrosplenial cortex (adapted from Buckner & Carroll, 2007)

Figure 5. The language network



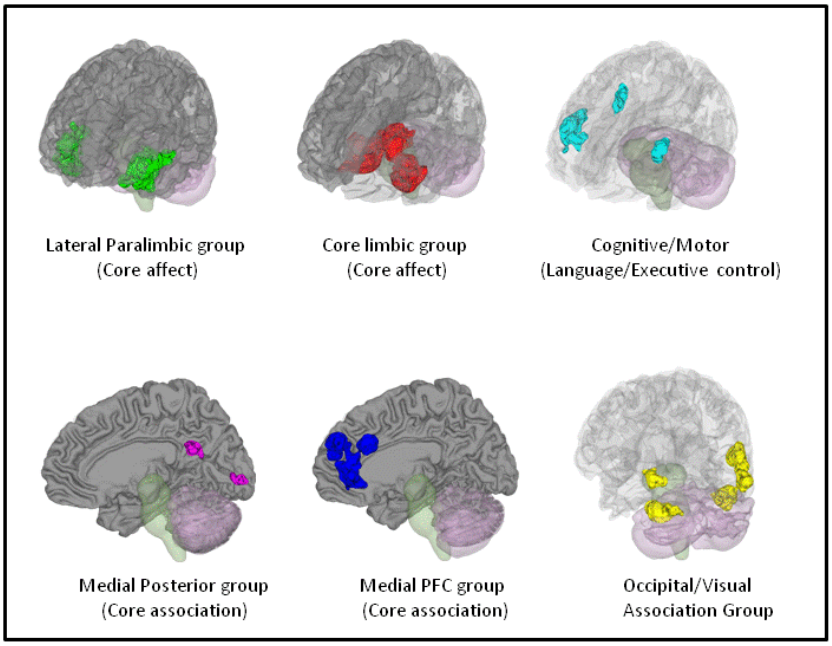
Areas involved in the language network, including ATL (green) and VLPFC (blue)

Figure 6. The executive attention network



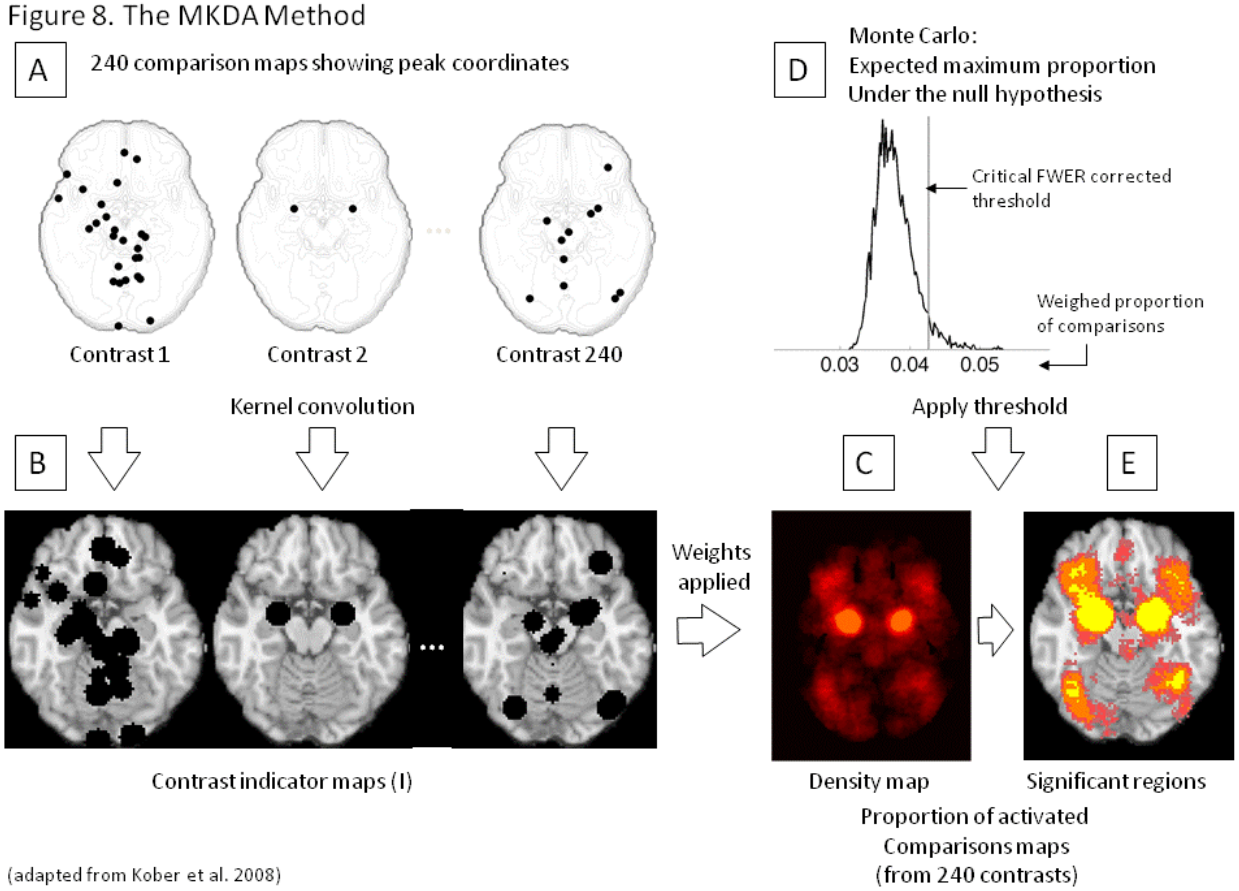
Areas involved in the executive attention network, including VLPFC (blue) and DLPFC (red)

Figure 7. Kober et al.'s (2008) Functional Networks



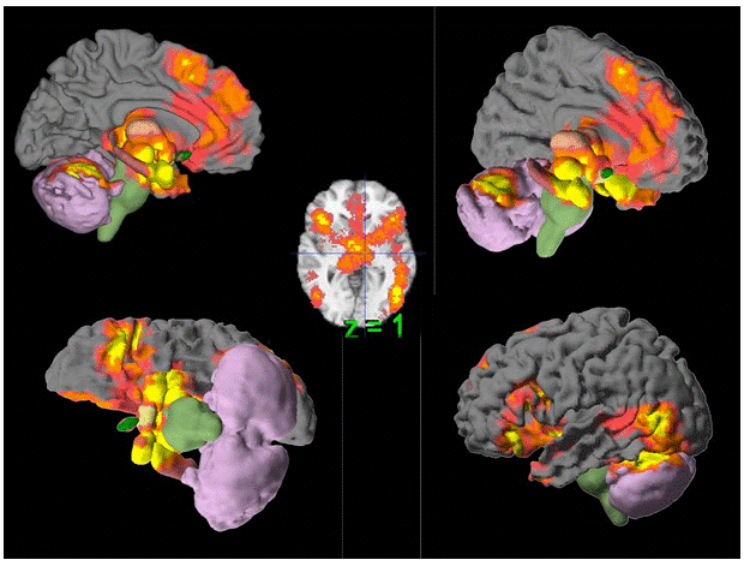
Areas from Kober et al.'s (2008) six functional networks are consistent with the ingredients hypothesized by a constructionist account. The Lateral paralimbic group (green) and core limbic group (red) are consistent with our hypothesized core affect network. The Medial Posterior group (purple) and Medial PFC group are consistent with our hypothesized core association network. The cognitive/Motor group (aqua) is consistent with the ingredients of language and executive attention.

Figure 8. The MKDA Method



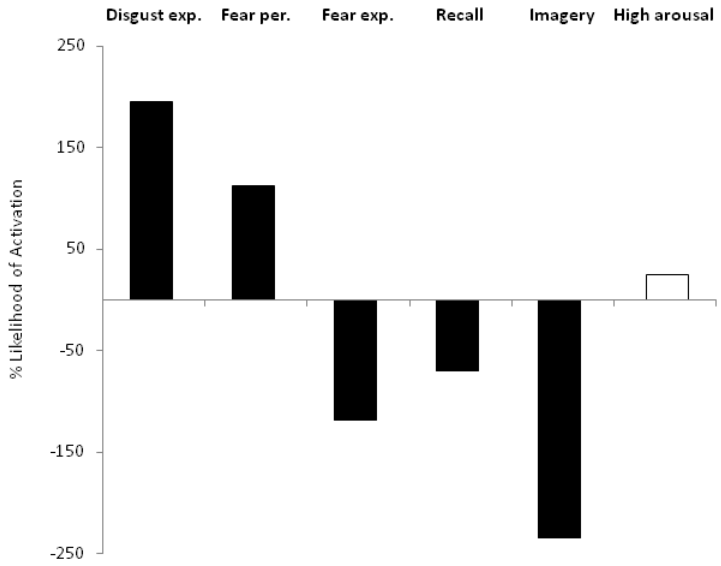
(adapted from Kober et al. 2008)

Figure 9. The Neural reference space for discrete emotions



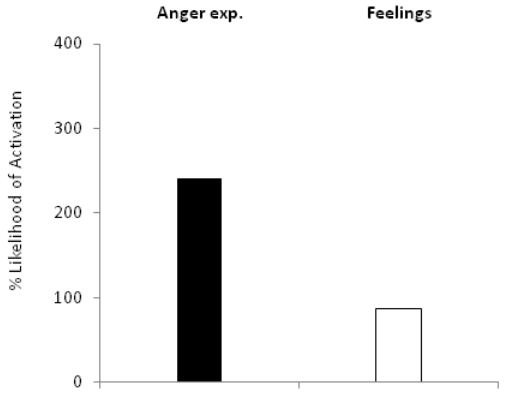
The neural reference space (coined by Edelman, 1989) is the set of brain regions consistently activated across all studies of emotion. Brain regions in yellow exceeded the height threshold ($p<.05$) and regions in orange exceeded the most stringent extent-based threshold ($p<.001$). Regions in pink and purple correspond to lesser extent-based thresholds and are not discussed in this paper.

Figure 10. Variables Associated with an Increase in Bilateral Amygdala Activation



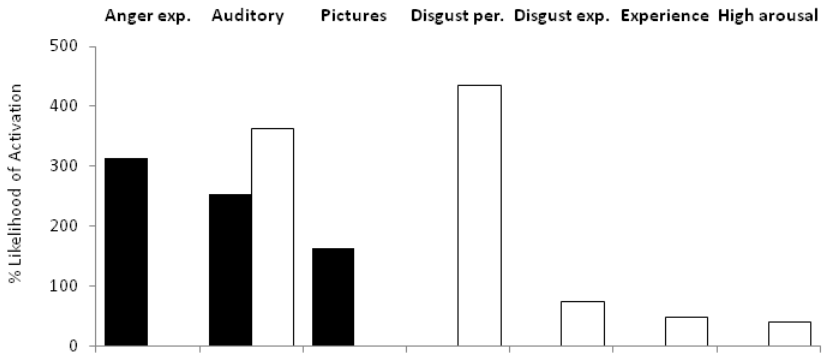
Percent likelihood that a variable was associated with increased activation in the L. amygdala (-20, -4, -16) in the logistic regressions is presented in black. Percent likelihood that a variable was associated with increased activation in the R. amygdala (22, -4, -16) is presented in white.

Figure 11. Variables Associated with an Increase in Bilateral Insula Activation



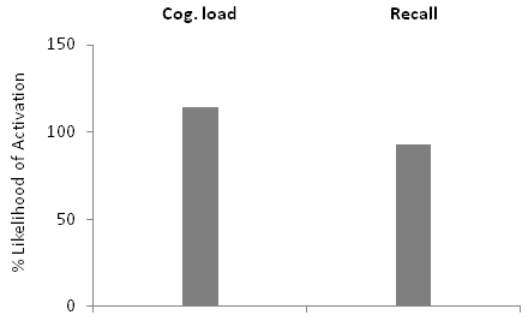
Percent likelihood that a variable was associated with increased activation in the L. ins. (-40, 26, -6) in the logistic regressions is presented in black. Percent likelihood that a variable was associated with increased activation in the R. ins. (48, 12, 0) is presented in white.

Figure 12. Variables Associated with an Increase in Bilateral OFC Activation



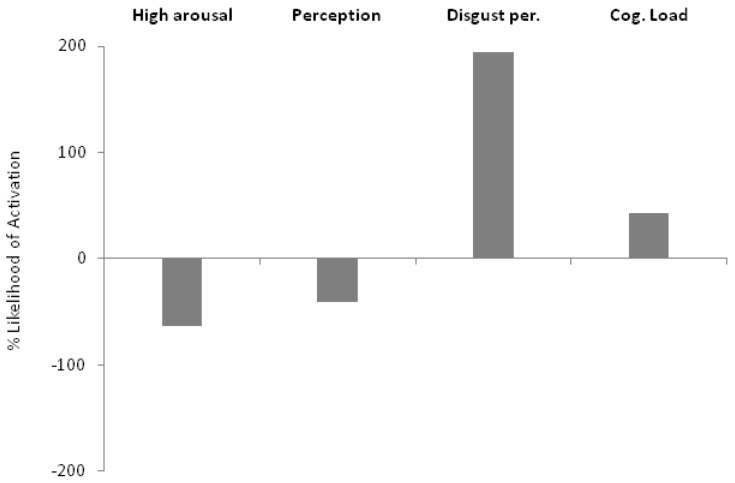
Percent likelihood that a variable was associated with increased activation in the L. OFC. (-42, 28, -8) in the logistic regressions is presented in black. Percent likelihood that a variable was associated with increased activation in the R. OFC. (42, 24, -4) is presented in white.

Figure 13. Variables Associated with an Increase in dACC Activation



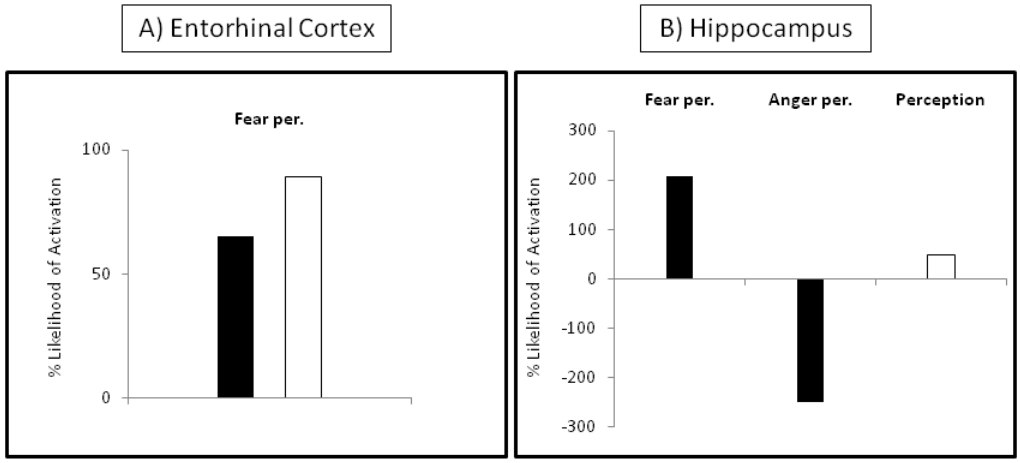
Percent likelihood that a variable was associated with increased activation in the dACC (3, 20, -2) in the logistic regressions.

Figure 14. Variables Associated with an Increase in DMPFC Activation



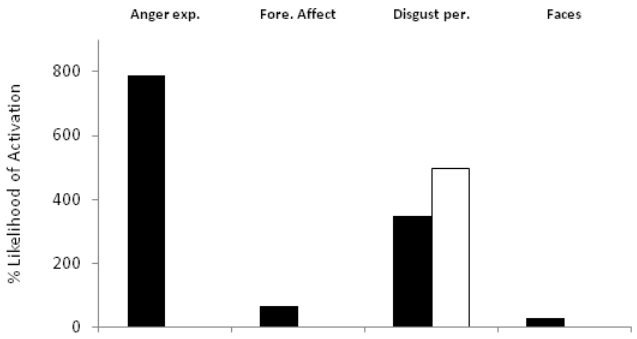
Percent likelihood that a variable was associated with increased activation in the DMPFC (extending into dACC; -6, 44, 24) in the logistic regressions.

Figure 15. Variables Associated with an Increase in Bilateral MTL Activation



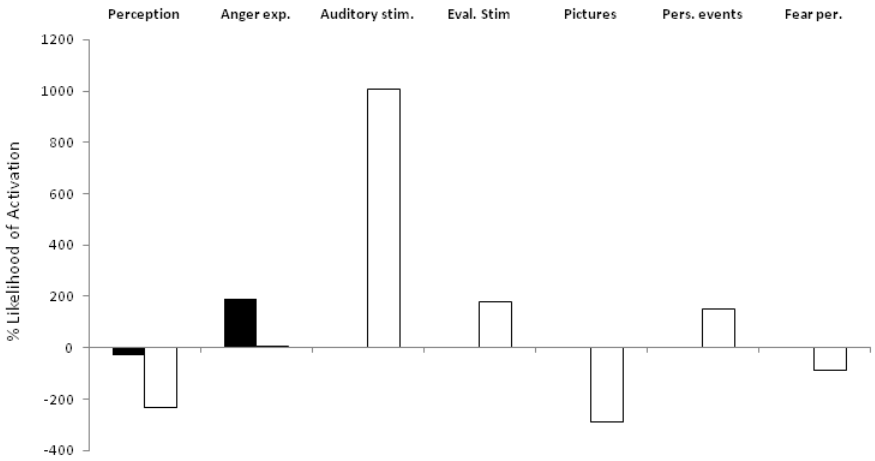
Percent likelihood that a variable was associated with increased activation in the L. entorhinal cortex (black; -12, 4, -18) and R. entorhinal cortex (white; 12, 24, -18) in the logistic regressions are presented in panel A. Percent likelihood that a variable was associated with increased activation in the L. hippomcapus (black;-32, -8, -20) and R. hippocampus (white; 30, -12, -16) are presented in panel B.

Figure 16. Variables Associated with an Increase in VLPFC Activation



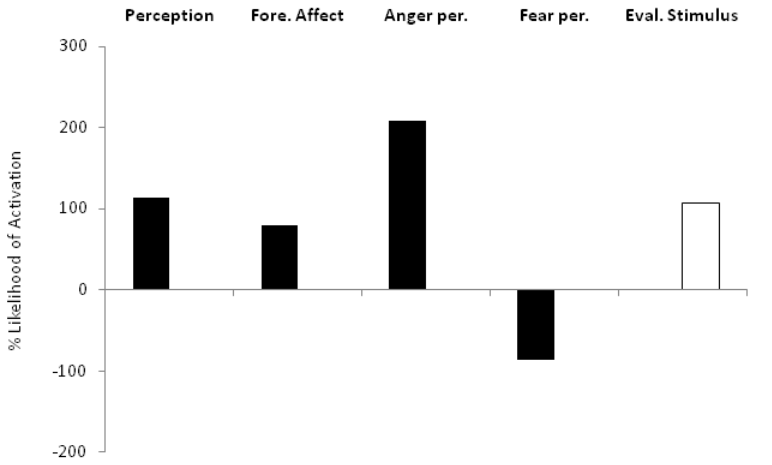
Percent likelihood that a variable was associated with increased activation in the L. VLPFC (-42, 16, 12) in the logistic regressions is presented in black. Percent likelihood that a variable was associated with increased activation in the R. VLPFC (46, 28, 10) is presented in white.

Figure 17. Variables Associated with an Increase in bilateral ATL Activation



Percent likelihood that a variable was associated with increased activation in the L. ATL (-34, 14, 20) in the logistic regressions is presented in black. Percent likelihood that a variable was associated with increased activation in the R. ATL (40, 14, -22) is presented in white.

Figure 18. Variables Associated with an Increase in Bilateral DLPFC Activation



Percent likelihood that a variable was associated with increased activation in the L. DLPFC (-46, 16, 24) in the logistic regressions is presented in black. Percent likelihood that a variable was associated with increased activation in the R. DLPFC (46, 10, 30) is presented in white.

Tables

Table 1. Locationist Hypotheses of Brain-Emotion Correspondence

Brain Region	Emotion
Amygdala	Fear
Insula	Disgust
OFC	Anger
ACC	Sadness

Table 2. Constructionist Hypotheses of Brain-Function Correspondence

Brain Region	Ingredient and role
Amygdala	Core affect; Signaling uncertainty
Insula	Core affect; Interoception
OFC	Core affect; Sensory integration for context-based behavior
ACC	Core affect; Conflict resolution

Table 3. Psychological Ingredients and their Functional Neural Networks

Ingredient	Network
Core Affect	medial OFC (BAs 10m, 11m, 13a, m, b, 14r, c), lat. OFC (BAs ,47/12), ACC (BAs, 32, 24, 25) insula, amygdala, basal ganglia
Core Association	DMPFC (BAs 9, 10p), medial temporal lobe (hippocampus, entorhinal cortex, parahippocampal cortex), posterior cingulate cortex/retrosplenial area (BA 23, 31)
Language	VLPFC (BAs 44, 45, 46), anterior temporal lobe (BA 38)
Executive Control	DLPFC (BAs 9, 10, 46), VLPFC (BAs 44, 45, 46)

Table 4. Prior Meta-analytic Findings for Locationist Hypotheses of Brain-Emotion Correspondence

		Meta-analysis			
Region	Emotion	Phan et al. (55)	Murphy et al. (28)	Vytal & Hamann (81)	Fusar-Pioli et al.* (105)
Amygdala	Anger			✓	
	Disgust			✓	✓
	Fear	✓	✓	✓	✓
	Happiness				✓
	Sadness				✓
Insula	Anger				✓
	Disgust		✓	✓	✓
	Fear			✓	
	Happiness			✓	✓
	Sadness			✓	
OFC	**Anger**		✓	✓	
	Disgust			✓	
	Fear			✓	
	Sadness			✓	
vACC	Anger			✓	
	Sadness	✓			
	Happiness			✓	
dACC	Anger			✓	
	Disgust			✓	
	Fear			✓	
	Happiness		✓		✓
	Sadness		✓		

Note: The sample size of studies analyzed is noted in parentheses next to each meta-analysis. Brain regions hypothesized a priori are listed in bold.
**Denotes that this meta-analysis assessed perception of emotional faces but not experience or perception more generally.*

Table 5. Prior Meta-analytic Findings for Constructionist Hypotheses of Brain-Emotion Correspondence

Region	Ingredient	Meta-analysis	
		Murphy et al. (36)	Wager et al. (65)
Amygdala	Affect (Withdraw)		✓
Insula	Affect (Negative)		✓
	Affect (Avoid)		✓
Lat. OFC			
	Affect (Positive)		✓
	Affect (Approach)		✓
dACC	Affect (Avoid)		✓
Striatum	Affect (Avoid)		✓

Table 6. Studies in Meta-analysis

First Author	Year	N	Fixed v. Random	Modality	Emotion(s)
Aalto	2002	11	Random	Experience	Sadness
Ashwin	2007	13	Random	Perception	Fear
Beauregard	1998	7	Fixed	Experience	Sadness
Blair	1999	13	Fixed	Perception	Anger
Breiter	1996	10	Fixed	Perception	Fear, Happiness
Britton	2006a	12	Random	Experience	Sadness, Disgust
Britton	2006b	12	Random	Experience, Perception	Anger, Fear, Happiness, Sadness
Buchanan	2000	10	Random	Perception	Happiness, Sadness
Calder	2007	12	Random	Experience	Disgust
Cooney	2007	14	Fixed	Experience	Sadness
Damasio	2000	25*	Fixed	Experience	Anger, Fear, Happiness, Sadness
Dannlowski	2007	23	Random	Perception	Anger, Sadness
Das	2005	28	Random	Perception	Fear
Deeley	2006	9	Fixed	Perception	Fear
Dolan	1996	8	Fixed	Perception	Happiness
Dougherty	1999	8	Fixed	Experience	Anger
Eugene	2003	20	Random	Experience	Sadness
Fischer	2004	24	Random	Perception	Anger
Fitzgerald	2004	12	Random	Experience	Disgust
Fitzgerald	2006	20	Random	Perception	Anger, Disgust, Fear, Happiness, Sadness
George	1994	21	Fixed	Experience	Sadness
George	1995	11	Fixed	Experience	Sadness
George	1996b	10	Fixed	Experience	Happiness, Sadness
Grandjean	2005	15	Random	Perception	Anger
Grezes	2007	16	Random	Perception	Fear
Grosbras	2006	20	Random	Perception	Anger
Hutcherson	2005	28	Random	Experience	Sadness
KeslerWest	2001	21	Fixed	Perception	Anger, Fear, Happiness, Sadness
Killgore	2004	12	Random	Perception	Happiness, Sadness
Kilts	2003	13	Random	Perception	Anger, Happiness
Kimbrell	1999	16	Fixed	Experience	Anger
Lane	1997c	11	Fixed	Experience	Disgust, Happiness, Sadness
Lange	2003	9	Random	Perception	Fear
Lee	2006	18	Random	Perception	Anger, Happiness, Sadness
Levesque	2003	20	Random	Experience	Sadness
Liddell	2005	25	Random	Perception	Fear
Liotti	2000	8	Fixed	Experience	Sadness
Malhi	2007	10	Random	Perception	Disgust, Fear
Marci	2007	10	Fixed	Experience	Anger, Happiness, Sadness
Markowitch	2003	13	Random	Experience	Happiness, Sadness
Mayberg	1999	8	Fixed	Experience	Sadness
Minzenberg	2007	12	Fixed	Perception	Anger, Fear
Mitchell	2007	15	Random	Perception	Fear
Mitterschiffthaler	2007	16	Random	Experience	Happiness, Sadness
Mizuno	2007	18	Random	Perception	Happiness, Sadness
Moll	2005	13	Fixed	Experience	Anger, Disgust
Nomura	2004	9	Fixed	Perception	Anger
Ottowitz	2004	8	Fixed	Experience	Sadness
Paradiso	1997	8	Fixed	Experience	Disgust, Happiness

Pardo	1993	7	Fixed	Experience	Sadness
Partiot	1995	12	Fixed	Experience	Sadness
Peelen	2007	18	Random	Perception	Anger, Disgust, Fear, Happiness, Sadness
Pessoa	2002	21	Fixed	Perception	Disgust, Fear
Phillips	1997	7	Random	Perception	Disgust, Fear
Phillips	1998a	6	Random	Perception	Disgust, Fear
Phillips	1998b	8	Random	Perception	Happiness, Sadness
Phillips	2004	8	Random	Perception	Disgust, Fear
Pietrini	2000	15	Random	Experience	Anger
Pourtois	2005	8	Random	Perception	Fear, Happiness
Rauch	2007	20	Random	Perception	Anger, Fear, Happiness
Reinders	2005	15	Random	Perception	Fear
Salloum	2007	11	Random	Perception	Anger, Disgust, Fear, Happiness, Sadness
Sambataro	2006	24	Random	Perception	Disgust
Sato	2004	10	Random	Perception	Anger
Schacher	2006	17	Random	Perception	Fear
Schafer	2005	40	Fixed	Experience	Disgust, Fear
Schienle	2002	12	Random	Experience	Disgust, Fear
Schienle	2006	12	Random	Experience	Disgust, Fear
Schroeder	2004	20	Random	Perception	Disgust
Silvert	2007	10	Random	Perception	Fear
Simon	2006	17	Random	Perception	Anger
Somerville	2004	16	Random	Perception	Happiness
Sprengelmeyer	1998	6	Fixed	Perception	Anger, Disgust, Fear
Stark	2003	19	Random	Experience	Disgust, Fear
Stark	2005	15	Random	Experience	Disgust, Fear
Stark	2007	66	Random	Experience	Disgust, Fear
Vuilleumier	2001	12	Random	Perception	Fear
Wang	2005	12	Random	Perception	Sadness
Whalen	2001	8	Fixed	Perception	Anger, Fear
Wicker	2003	14	Random	Experience, Perception	Disgust
Williams, L.	2001	11	Fixed	Perception	Fear
Williams, L.	2004	22	Fixed	Perception	Fear
Williams, C.	2005	13	Random	Perception	Anger, Disgust, Fear
Williams, L.	2006a	15	Random	Perception	Fear
Williams, L.	2006b	13	Random	Perception	Fear
Williams, L.	2006c	15	Random	Perception	Fear
Wright, C	2006	18	Random	Perception	Fear
Wright, P	2004	8	Fixed	Experience	Disgust, Fear
Yamasaki	2002	10	Fixed	Experience	Disgust

Note: Damasio et al. 2000 had differing Ns across contrasts (ranging from 16-25)

Table 7. Neural Reference Space for Discrete Emotion

Region	BA	Threshold	Coordinates (MNI)			Volume (voxels)
			x	y	z	
Core affect						
L. amygdala		*Height*	-20	-4	-16	1259
R. amygdala		*Height*	22	-4	-16	919
L. lat. OFC	47	*Height*	-34	18	-16	1
			-40	26	-6	173
			-52	28	-6	1
L. lat OFC/a. ins		*Extent*	-42	28	-8	191
			-32	24	-10	183
			-46	24	0	337
L .anterior insula		*Height*	-40	14	14	2
		Extent	-38	8	-10	123
L. mid insula		*Extent*	-38	8	-8	193
R. lat. OFC	47	*Height*	44	26	0	6
			44	26	-6	1
			48	28	0	2
			48	16	4	1
		Extent	42	24	-4	286
R. anterior insula		*Extent*	48	12	0	28
		Extent	42	24	-4	286
ACC	32	*Extent*	3	20	-2	31
			0	38	6	62
L. Putamen		*Extent*	-22	8	-10	123
			-28	-2	-4	68
R. Putamen		*Extent*	28	6	0	243
Midbrain (PAG)		*Height*	-14	-28	-2	4
		Extent	-2	-30	-6	109
Midbrain		*Height*	0	-4	4	1
			10	-20	-8	1
		Extent	-4	0	2	337
			-6	-8	0	258
			16	-18	-8	206
			8	-14	-2	176
			6	-4	-2	150
Core Association						
DMPFC	9	*Height*	-8	46	34	2
	8		-2	18	50	12
			-4	16	56	1
	9	*Extent*	-6	40	32	21
			-6	48	34	171
			2	42	44	38
DMPFC/d. ACC	9/32	*Extent*	2	46	14	52
			-6	44	24	140
DMPFC	8	*Extent*	-4	18	48	112
			-8	24	54	75
			-8	16	56	138
			0	12	56	39
L. Parahippcampal cortex		*Extent*	-18	-12	-26	79
			-10	-34	-2	25
R. Parahippcampal cortex			16	-10	-26	85
L. Entorhinal cortex		*Extent*	-12	4	-18	72
			-24	-14	-8	65
R. Entorhinal cortex		*Extent*	12	24	-14	80
			24	6	-14	97

L. Hippocampus		*Extent*	-32	-8	-20	91
R. Hippocampus		*Extent*	30	-12	-16	70
L. Temporal-parietal junction	39	*Height*	-48	-76	8	1
			-52	-60	10	1
			-48	-68	8	77
R. Temporal-parietal junction		*Extent*	54	-60	0	50
			44	-58	2	164
			54	-46	2	134
Language						
R. Anterior temporal lobe	38	*Height*	36	6	-26	6
			48	8	-16	1
		Extent	40	14	-22	78
L. Anterior temporal lobe		*Extent*	-34	14	-20	260
R. Superior temporal cortex	22	*Height*	54	-46	4	18
			54	-34	6	1
L. VLPFC	44	*Extent*	-42	16	12	114
R. VLPFC	45	*Height*	48	22	14	160
			44	22	16	1
	45	*Extent*	46	28	10	241
			48	20	20	301
			42	16	6	227
Executive control						
R. DLPFC	9	*Height*	46	10	30	6
		Extent	46	8	32	153
			52	22	30	105
L. VLPFC	9	*Height*	-46	16	24	202
L. DLPFC		*Extent*	-52	18	26	65
			-46	8	24	61
L. DLPFC	46	*Extent*	-40	18	26	110
Extraceptive Sensory Input						
L. peristriate cortex	19	*Height*	-46	-74	-6	1
			-48	-68	8	77
			-48	-76	8	1
		Extent	-40	-80	-14	176
			-48	-72	-6	139
R. peristriate cortex		*Extent*	50	-74	4	186
L. occipitotemporal cortex	37	*Height*	-48	-72	-2	1
			-48	-68	0	1
			-42	-58	18	462
		Extent	-46	-52	-24	89
			-34	-62	-22	61
			-38	-44	-20	118
			-38	-72	-14	170
			-42	-58	-10	83
R. occipitotemporal cortex	37	*Height*	42	-54	20	437
			40	-66	-12	1
			40	-62	-8	1
			48	-66	4	249
		Extent	42	-54	-20	437
			40	-66	-12	1
			40	-62	-8	1
			48	-66	4	249
R. middle temporal cortex	21	*Height*	52	4	-14	1
R. middle temporal cortex	21	*Extent*	50	2	-18	81
			50	10	-18	136
			54	4	-12	51
Optic tract		*Extent*	0	-20	4	122
Uncus		*Extent*	-28	4	-28	157
			36	0	-26	157
			24	6	-26	85
L. thalamus		*Height*	-8	-24	6	1

			-10	-20	6	1
			-12	-26	4	7
		Extent	-20	-28	2	61
			-12	-28	2	152
			-10	18	8	149
R. thalamus		*Height*	8	-16	0	5
		Extent	12	-20	2	80
Additional regions						
L. cerebellum		*Extent*	-44	-68	-24	112
R. cerebellum			36	-58	-26	66
R. SMA	6	*Extent*	46	0	40	67

Table 8. Brain Regions Significantly More Active for a Category in the Absolute Analyses

Region	BA	Contrast	Coordinates (MNI)			Volume (voxels)
			x	y	z	
Amygdala-Fear Hypothesis						
L. Amygdala		Disgust experience	-20	-6	-24	50
			-32	-2	-20	2
R. Amygdala		Disgust experience	26	2	-20	59
Insula-Disgust Hypothesis						
L. a. insula		Anger experience	-44	20	-2	1
R. a. insula		Disgust perception	42	14	4	4
			34	20	6	3
			36	18	2	1
OFC-Anger Hypothesis						
L. lat OFC	11	Disgust experience	-30	36	-18	167
R. lat OFC	47	Disgust perception	38	22	0	8
Other regions in the neural reference space						
Core association						
L. entorhinal cortex	34	Disgust experience	-26	-6	-20	1
		Sad experience	-24	2	-12	10
R. entorhinal cortex	34	Anger perception	16	-10	-16	23
DMPFC	9	Sad experience	2	50	38	1
Executive control						
L. DLPFC	9	Anger perception	-52	14	24	27
R. DLPFC	9	Anger perception	54	22	28	130
R. VLPFC	44	Disgust perception	46	20	10	66
Exteroceptive Sensory Processing						
R. parastriate	18	Anger perception	52	-76	-2	2
			48	-76	0	1
			42	-86	2	3
		Fear experience	8	-96	4	10
L. occipitotemporal	37	Disgust experience	-46	-58	-14	1
			-42	-58	-10	1
R. occipitotemporal	37	Fear experience	48	-72	2	127
		Anger perception	44	-56	-24	1
			38	-52	-14	6
L. middle temporal	21	Fear experience	-52	-70	8	72
R. middle temporal	21	Sad experience	52	-10	-16	2
Other						
R. SMA	6	Anger perception	44	-2	56	1
R. putamen		Sad experience	26	4	-4	1
			28	8	-2	3
			22	4	-2	1
PAG		Sad experience	0	-38	-10	1

Table 9. Brain Regions Significantly More Active for a Category in the Relative Analyses

Region	BA	Threshold	Contrast	Coordinates (MNI)			Volume (voxels)
				x	y	z	
Amygdala-Fear Hypothesis							
L. Amygdala		*Height*	Disgust experience	-32	-2	-20	2
				-20	-4	-22	124
			Fear perception	-30	-2	-24	1
				-24	-2	-12	360
			Sad perception	-30	-4	-20	1
R. Amygdala		*Height*	Disgust experience	26	0	-22	121
			Fear perception	24	-4	-12	250
Insula-Disgust Hypothesis							
L. a. insula		*Height*	Anger experience	-42	22	-2	2
		Extent	Disgust perception	-26	22	-12	252
R. a. insula/R. lat. OFC		*Height*	Disgust perception	38	20	4	37
OFC-Anger Hypothesis							
L. lat OFC	11	*Height*	Disgust experience	-30	36	-18	167
Core association							
L. dorsal entorhinal	34	*Height*	Disgust experience	-16	2	-16	1
				-18	0	-14	1
		Height	Sad experience	-24	2	-12	19
L. hippocampus		*Height*	Sad perception	-28	-10	-20	1
R. dorsal entorhinal	34	*Height*	Anger perception	18	-12	-16	27
		Height	Fear perception	14	-6	-12	1
DMPFC	9	*Extent*	Happy experience	-2	44	20	324
		Height	Sad experience	2	50	38	1
Executive control							
R. VLPFC	44	*Height*	Disgust perception	46	18	10	71
L. DLPFC	9	*Height*	Anger perception	-52	12	24	27
R. DLPFC	9	*Height*	Anger perception	54	22	28	130
Exteroceptive Sensory Processing							
R. parastriate	18	*Height*	Anger perception	52	-76	-2	2
				48	-76	0	1
				42	-86	2	3
		Height	Fear experience	8	-96	4	10
L. peristriate	19	*Extent*	Anger perception	-48	-80	-8	197
L. occipitotemporal	37	*Height*	Disgust experience	-44	-58	-12	7
		Extent	Anger perception	-44	-54	-20	232
				-50	-62	6	254
R. occipitotemporal	37	*Height*	Anger perception	44	-56	-24	1
				40	-56	-20	1
				40	-54	-14	7
L. middle temporal	21	*Height*	Fear experience	-52	-70	8	72
		Extent	Sad perception	-66	-48	8	323
R. middle temporal	21		Sad experience	52	-10	-16	2
Uncus		*Height*	Fear perception	-30	4	-22	5
Other							
R. SMA	6	*Height*	Anger perception	44	-2	56	1

R. putamen	*Height*	Sad experience	26	4	-4	1
			28	8	-2	3
			22	4	-2	1
PAG	*Height*	Sad experience	0	-38	-10	1

Table 10. Logistic Regression Analyses

Region	voxels	BA	threshold	Predictors	Coefficient		Dir.	Odds	Model fit	
					β	*p*			χ^2	*p*
L. amygdala [-20, -4, -16]	1259		*Height*	Experience disgust	1.08	<.003	+	2.95	36.11	<.0001
				Perception fear	.75	<.001	+	2.12		
				Perception anger	-.76	<.02	-	2.15		
				Experience fear	-2.18	<.003	+	8.89		
				Recall	-.54	<.05	-	1.71		
				Mental Imagery	-1.21	<.07	-	3.35		
R. amygdala [22, -4, -16]	919		*Height*	High arousal	.22	<.03	+	1.25	4.93	<.03
L. a. insula* [-40, 26, -6]	173		*Height*	Experience anger	1.23	<.04	+	3.41	4.12	<.04
L. mid. insula [-38, 8, -8]	193		*Extent*	Experience anger	1.33	<.03	+	3.80	4.53	<.03
R. a. insula [48, 12, 0]	28		*Extent*	Evaluation, feelings	.63	<.04	+	1.87	4.06	<.04
R. lat. OFC [44, 26, 0]	6	47	*Height*	Perception disgust	1.18	<.04	+	7.17	10.64	<.005
				Auditory stimuli	1.97	<.004	+	3.26		
L. lat. OFC [-42, 28, -8]	191	47	*Extent*	Experience anger	1.42	<.04	+	4.14	15.35	<.002
				Auditory stimuli	1.26	<.03	+	3.54		
				Picture stimuli	.97	<.01	+	2.63		
R. lat. OFC [42, 24, -4]	286	47	*Extent*	Perception disgust	1.68	<.001	+	5.36	24.35	<.0001
				Experience disgust	.56	<.09	+	1.75		
				Auditory stimuli	1.53	<.014	+	4.63		
				Mode, experience	.39	<.01	+	1.48		
				High arousal	.34	<.03	+	1.41		
dACC** [3, 20, -2]	31	32	*Extent*	Cognitive load	.76	<.03	+	2.14	15.47	<.0002
				Evaluation, feelings	.66	<.09	+	1.93		
dACC** [0, 38, 6]	62	32	*Extent*	Recall	.88	<.05	+	2.50	9.99	<.01
DMPFC [-6, 48, 34]	171	9	*Extent*	Mode, perception	-.36	<.03	-	1.43	10.66	<.004
				High arousal	-.26	<.07	-	1.30		
DMPFC [2, 42, 44]	38	9	*Extent*	Mode, perception	-.48	<.02	+	1.62	6.20	<.01
DMPFC [-6, 40, 32]	21	9	*Extent*	High arousal	-.31	<.05	-	1.37	3.87	<.05
DMPFC/d.ACC [2, 46, 14]	52	9/32	*Extent*	Recall	2.54	<.003	+	34.57	14.44	<.002
				Mode, perception	.73	<.035	+	2.07		
				Film stimuli	1.72	<.08	+	5.60		
DMPFC/dACC [-6, 44, 24]	140	9/32	*Extent*	High arousal	-.49	<.001	-	1.63	22.95	<.0001
				Mode, perception	-.35	<.02	-	1.41		
				Perception disgust	1.08	<.05	+	2.94		
				Cognitive load	.36	<.06	+	1.43		
L. dorsal entorhinal [-12, 4, -18]	72	34	*Extent*	Perception fear	.50	<.05	+	1.65	5.46	<.02
R. dorsal entorhinal [12, 24, -18]	80	34	*Extent*	Perception fear	.64	<.02	+	1.89	7.50	<.01
L. hippocampus [-32, -8, -20]	91		*Extent*	Perception fear	1.12	<.001	+	3.07	18.19	<.001
				Perception anger	-1.25	<.03	-	3.51		
R. hippocampus [30, -12, -16]	70		*Extent*	Mode, perception	.39	<.02	+	1.47	14.48	<.001
L. VLPFC [-42, 16, 12]	114	44	*Extent*	Experience anger	2.19	<.01	+	8.89	27.88	<.0001
				Foregrounded affect	.52	<.01	+	1.68		
				Perception disgust	1.50	<.001	+	4.48		
				Face stimuli	.25	<.07	+	1.28		
R. VLPFC [48, 22, 14]	160	45	*Height*	Auditory stimuli	1.78	<.04	+	6.04	3.54	<.06

R. VLPFC [46, 28, 10]	241	44	*Extent*	Perception disgust	1.79	<.001	+	5.97	16.23	<.0001
L. a. temporal [-34, 14, 20]	260	38	*Extent*	Mode, perception	-.26	<.05	-	1.29	11.08	<.01
				Experience anger	1.07	<.08	+	2.92		
				Negative valence	.15	<.10	+	1.16		
R. a temporal [40, 14, -22]	78	38	*Extent*	Mode, perception	-1.21	<.0001	-	3.34	34.51	<.0001
				Auditory stimuli	2.40	<.001	+	11.07		
				Evaluation, stimulus	1.02	<.001	+	2.78		
				Pictures	-1.36	<.011	-	3.88		
				Personal events	.91	<.09	+	2.49		
				Perception fear	-.59	<.01	-	1.87		
L. DLPFC [-46, 16, 24]	202	9	*Height*	Mode, perception	.76	<.003	+	2.14	30.25	<.0001
				Foregrounded affect	.57	<.011	+	1.80		
				Perception anger	1.13	<.003	+	3.09		
				Perception fear	-.63	<.033	-	.1.87		
R. DLPFC [46, 10, 30]	6	9	*Height*	Evaluation, stimulus	.73	<.05	+	2.07	5.20	<.02
L. DLPFC [-40, 18, 26]	110	46	*Extent*	Mode, perception	.56	<.003	+	1.76	15.86	<.001
				Foregrounded affect	.64	<.01	+	1.90		
				Perception anger	.61	<.09	+	1.84		
PAG [-14, -28, -2]	4		*Height*	High arousal	.62	<.06	+	1.87	9.76	<.001
				Imagery	-4.94	<.001	-	140.32		
PAG [-2, -30, -6]	109		*Extent*	Experience fear	1.88	<.003	+	6.61	27.69	<.0001
				Evaluation, stimulus	.92	<.015	+	2.52		
				Auditory stimuli	1.54	<.07	+	4.68		
				Visual methods	-.78	<.001	-	2.17		
				Personal events	-1.60	<.01	-	4.94		
				Cognitive load	-.34	<.06	-	1.42		
L. peristriate [-48, -68, 8]	77	19	*Height*	Experience happiness	3.96	<.002	+	52.93	28.61	<.001
				Foregrounded affect	2.56	<.001	+	13.13		
				Evaluation, stimulus	.56	<.04	+	1.75		
				Film stimuli	.50	<.03	+	1.64		
				Unpleasant affect	.34	<.06	+	1.41		
R. peristriate [50, -74, 4]	186	19	*Extent*	Experience fear	1.72	<.032	+	5.57	54.85	<.0001
				Perception disgust	1.16	<.05	+	3.18		
				Picture stimuli	9.57	<.002	+	140,000		
				Mode, perception	4.69	<.004	+	108.87		
				Visual methods	2.47	<.07	+	11.88		
				Face stimuli	-3.88	<.001	-	48.53		
				Experience disgust	-1.86	<.003	-	6.42		
L. occipitotemporal [-42, -58, 18]	462	37	*Height*	Evaluation, stimulus	.65	<.01	+	1.91	40.93	<.0001
				Visual method	2.11	<.04	+	8.26		
				Cognitive load	-.51	<.001	-	1.66		
R. occipitotemporal [42, -54, 20]	437	37	*Height*	Mode, perception	.42	<.01	+	1.51	17.20	<.001
				Personal event stimuli	-.77	<.03	-	2.16		
				Cognitive load	-.77	<.03	-	1.34		
R. occipitotemporal [48, -66, 4]	249	37	*Height*	Experience fear	2.01	<.009	+	7.48	56.77	<.0001
				Perception disgust	1.71	<.003	+	5.54		
				Mode, perception	3.08	<.001	+	21.94		

				Film stimuli	7.84	<.0002	+	561.83		
				Picture stimuli	6.59	<.0001	+	729.79		
				Experience disgust	-1.24	<.04	+	3.44		
				Mental imagery	2.87	<.03	+	17.58		
R. middle temporal [50, 2, -18]	18	21	*Height*	Perception fear	1.06	<.014	+	2.89	14.91	<.002
				Mental imagery	1.85	<.016	+	6.37		
				Film stimuli	1.12	<.06	+	3.06		

Note: Odds are the likelihood of an independent variable predicting activation in a given cluster. Odds are determined by dividing the probability of one outcome by the probability of another. The column Dir. refers to the directionality of the findings. + denotes odds of predicting activation; - denotes odds of failing to predict activation. *this cluster extended partially into the L. lat. OFC ** the vACC did not appear in the height-based or most stringent threshold of the neural reference space so I did not investigate this region

Acknowledgements

First, I would like to acknowledge the researchers involved in compiling and coding the database used in this meta-analytic review, including Hedy Kober, Eliza Bliss-Moreau, Tor Wager and Lisa Feldman Barrett. Second, I would like to thank Lisa Feldman Barrett for her guidance and mentorship, both on this project, and throughout the years. Third, I would like to thank my committee members James Russell, Hiram Brownell, Elizabeth Kensinger and Gerald Clore for their invaluable input and support. This project has benefitted from the input of many individuals including Amitai Shenhav, who had the insight that the amygdala is involved in uncertainty, Eliza Bliss-Moreau, who has expertise on the functional role of the ACC, Natasha Sarkisian who gave advice on logistic regression, and Matt Panichello and Krystal Yu who graciously helped with figures. In addition, I'd like to thank the members of the Interdisciplinary Affective Science Laboratory for their valued support. Finally, I thank the National Science Foundation for the Graduate Research Fellowship that supported my graduate education and helped make this research happen.

Appendix A. References included in meta-analysis

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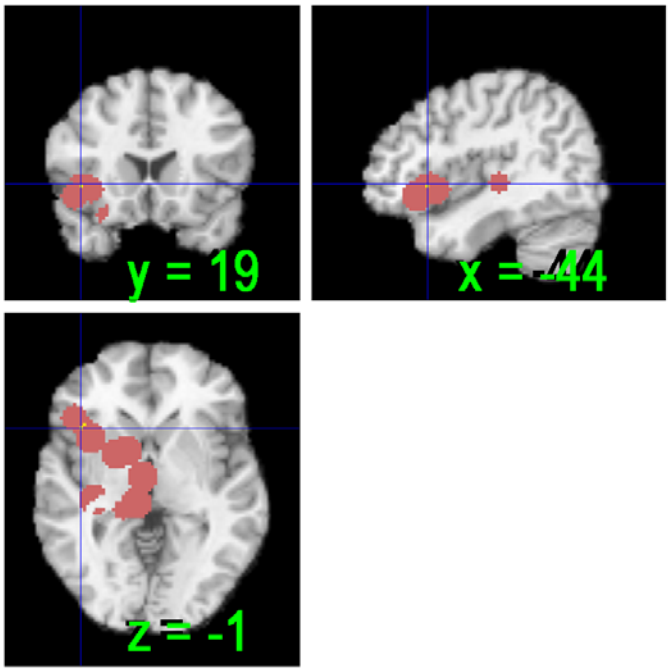
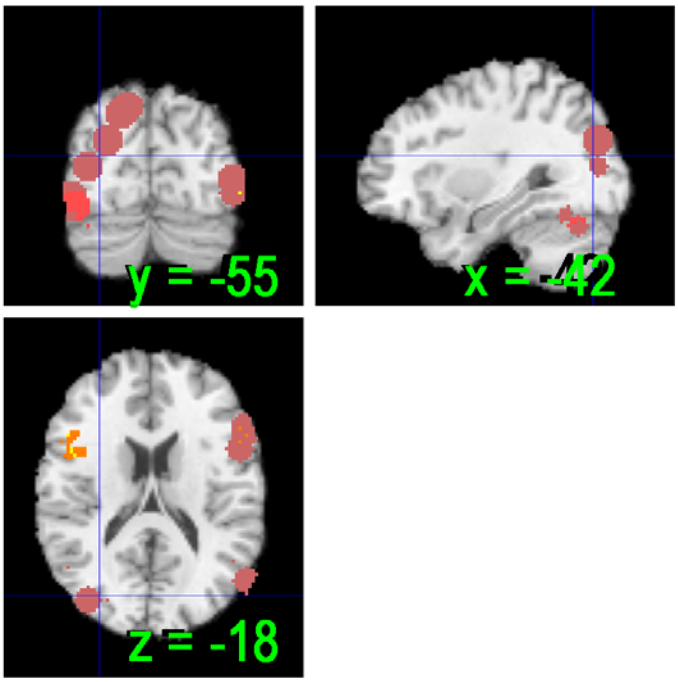
Appendix B. The Neural Reference Spaces for the Experience and Perception of Anger, Disgust, Fear, Happiness and Sadness

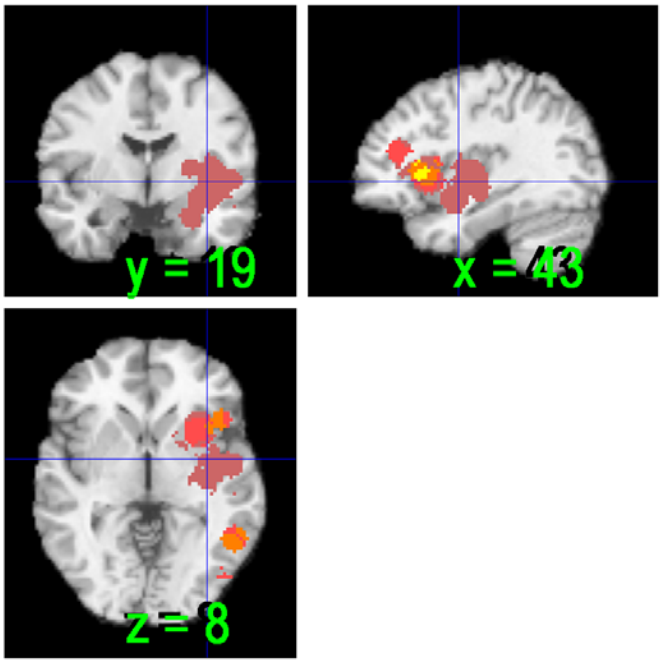
Anger Experience



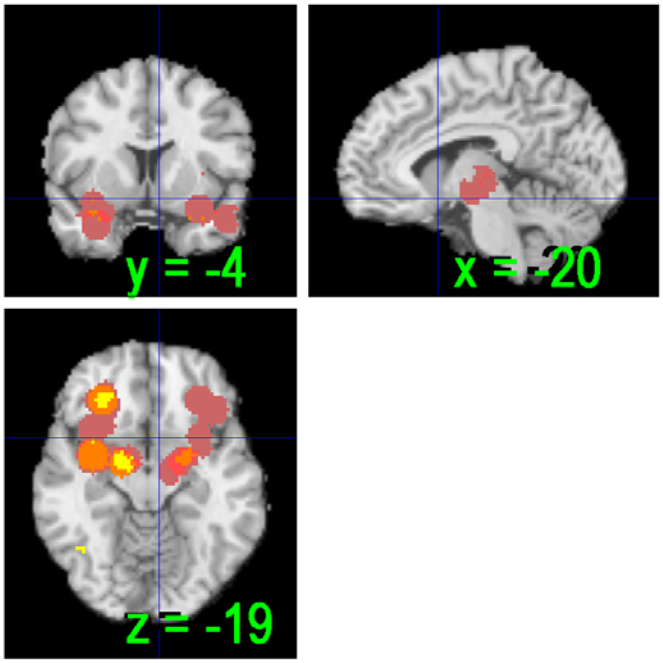
Anger Perception



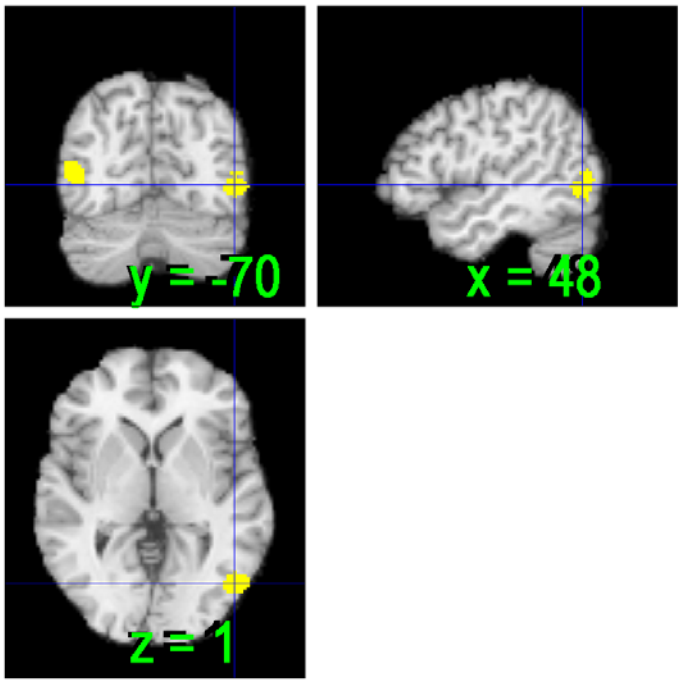
Disgust Experience



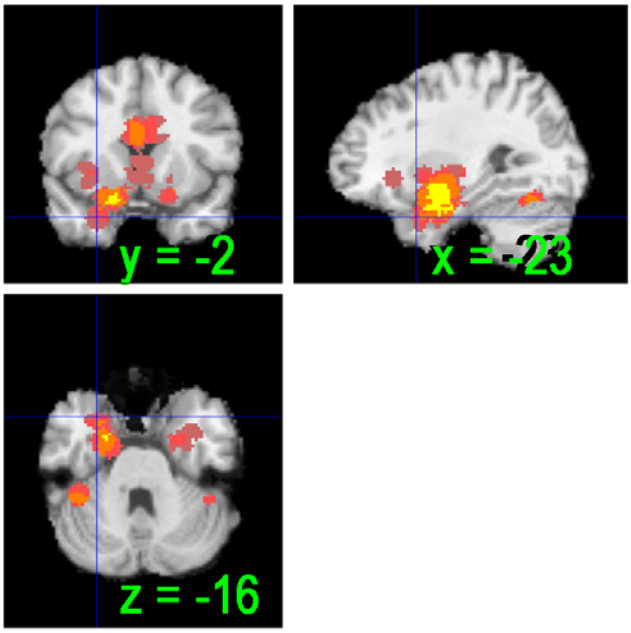
Disgust Perception



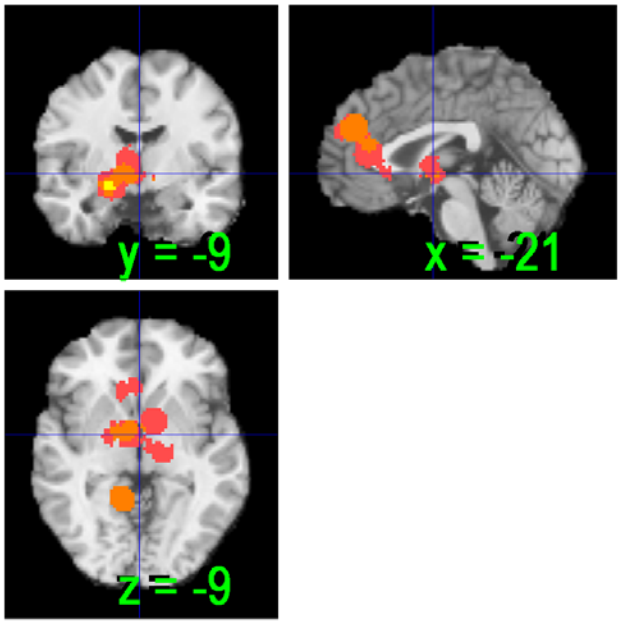
Fear Expereince



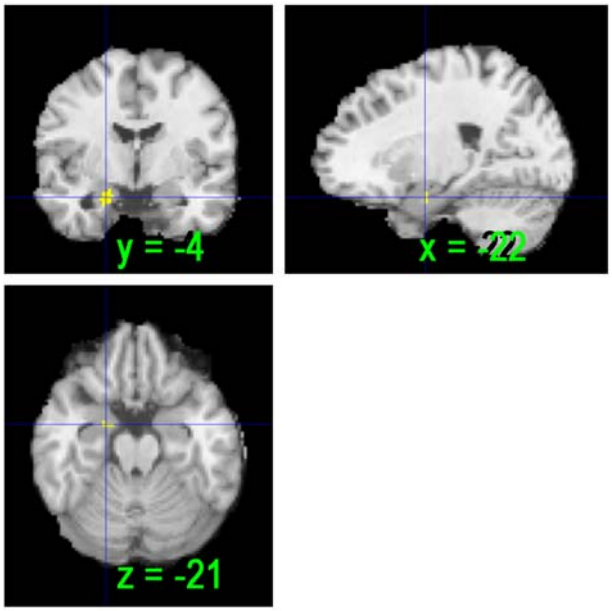
Fear Perception



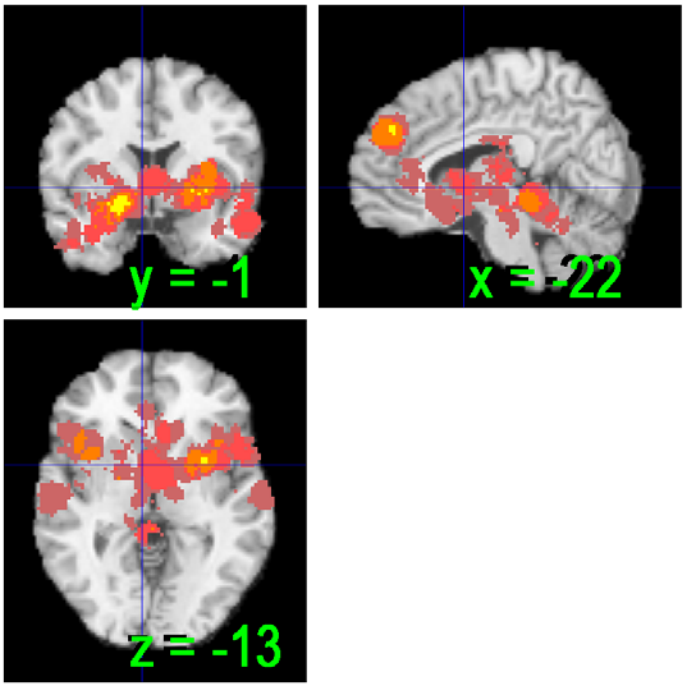
Happy Experience



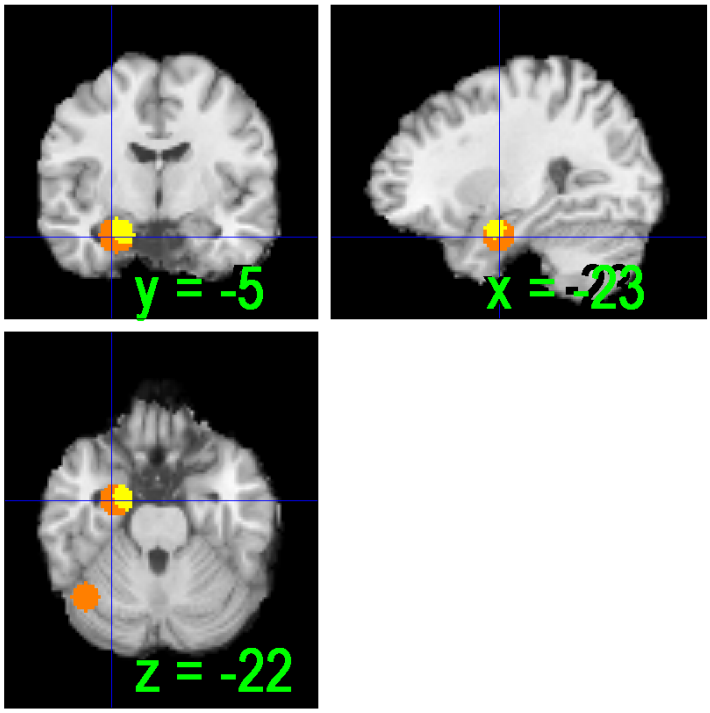
Happy Perception



Sad Experience



Sad Perception



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