Emotion
2010, Vol. 10, No. 3, 377–389

1528-3542/10/$12.00 DOI: 10.1037/a0018484

Neural Systems Subserving Valence and Arousal During the Experience of Induced Emotions

Tiziano Colibazzi, Jonathan Posner, Zhishun Wang, Daniel Gorman, Andrew Gerber, Shan Yu, Hongtu Zhu, Alayar Kangarlu, and Yunsuo Duan
The New York State Psychiatric Institute, Columbia College of Physicians and Surgeons

James A. Russell
Boston College

Bradley S. Peterson
The New York State Psychiatric Institute, Columbia College of Physicians and Surgeons

The circumplex model of affect construes all emotions as linear combinations of 2 independent neurophysiological dimensions, valence and arousal. We used functional magnetic resonance imaging to identify the neural networks subserving valence and arousal, and we assessed, in 10 participants, the associations of the BOLD (blood oxygen level-dependent) response, an indirect index of neural activity, with ratings of valence and arousal during the emotional experiences induced by the presentation of evocative sentences. Unpleasant emotional experience was associated with increased BOLD signal intensities in the supplementary motor, anterior midcingulate, right dorsolateral prefrontal, occipitotemporal, inferior parietal, and cerebellar cortices. Highly arousing emotions were associated with increased BOLD signal intensities in the left thalamus, globus pallidus, caudate, parahippocampal gyrus, amygdala, premotor cortex, and cerebellar vermis. Separate analyses using a finite impulse response model confirmed these results and revealed that pleasant emotions engaged an additional network that included the midbrain, ventral striatum, and caudate nucleus, all portions of a reward circuit. These findings suggest the existence of distinct networks subserving the valence and arousal dimensions of emotions, with midline and medial temporal lobe structures mediating arousal and dorsal cortical areas and mesolimbic pathways mediating valence.

Keywords: affect, valence, arousal, dimensional, fMRI

Supplemental materials: http://dx.doi.org/10.1037/a0018484.supp

A long theoretical tradition and recent empirical imaging studies together are yielding important insights into the neurobiological basis of emotional experience (Calder, Lawrence, & Young, 2001; Phan, Wager, Taylor, & Liberzon, 2002). Numerous competing models of emotion have evolved since the seminal studies of the early 20th century (Cannon & Britton, 1925; Lange & James, 1922; Scherer, 2001). Among these theories, one of the most influential has arguably been the theory of basic or discrete emotions (Ekman, 1992a, 1992b; Tomkins, 1962). The theory of basic emotions posits that all affective experiences derive from a core set of basic emotions, that those basic emotions are distinct and independent, and that a discrete neural system subserves each of these emotions. Moreover, it is thought that unique sets of neurobehavioral, neurophysiological, and psychological attributes accompany each basic emotion and can therefore be used as proxies to investigate affective states (Posner, Russell, & Peterson, 2005). Basic emotion theorists believe, for example, that the emotions of fear and happiness are mediated by different neural structures and that these emotions are expressed through nonoverlapping patterns of autonomic responses, motor behaviors, and facial expressions (Panksepp, 1992, 1998).

The theory of basic emotions, however, has certain limitations (Posner et al., 2005). First, animal studies are frequently invoked to support the theory (Berridge, 2003), even though emotions experienced by an animal are at best only inferred. Second, most studies of affect in humans rely on facial expressions. Faces may not be a valid measure of emotionality, however, given that a specific facial expression does not always accompany the same emotion. A smile, for instance, can signal pleasure, discomfort, or even anger, depending on the specific context in which it is displayed. Third, the precise number of basic emotions is in considerable dispute, and its various schemes of categorizations

Tiziano Colibazzi, Jonathan Posner, Zhishun Wang, Daniel Gorman, Andrew Gerber, Shan Yu, Hongtu Zhu, Alayar Kangarlu, Yunsuo Duan, and Bradley S. Peterson, Division of Child and Adolescent Psychiatry, The New York State Psychiatric Institute, Columbia College of Physicians and Surgeons; James A. Russell, Department of Psychology, Boston College.

We thank Satie Shova for her technical assistance.

Correspondence concerning this article should be addressed to Tiziano Colibazzi, College of Physicians and Surgeons and New York State Psychiatric Institute, Unit 74, 1051 Riverside Drive, New York, NY 10032. E-mail: tc2237@columbia.edu; or (for reprints) Bradley S. Peterson, College of Physicians and Surgeons and New York State Psychiatric Institute, Unit 74, 1051 Riverside Drive, New York, NY 10032. E-mail: petersob@childpsych.columbia.edu

often reflect semantic differences that map poorly onto behavioral repertoires and discrete neural systems (Kober et al., 2008).

An alternate approach to the study of emotions has been the development of dimensional models of affect, which posit that all emotions derive from a combination of two or more underlying psychological "dimensions" (Posner et al., 2005; Rolls, 1999; Schlosberg, 1954; Watson & Tellegen, 1985). These models have a long history of study using introspectionist (Wundt & Wirth, 1905), semantic (Osgood, 1969) and psychophysiological (Lang, 1995; Lang, Bradley, & Cuthbert, 1990) experimental frameworks. Factor analyses of self-reported emotional states, and multidimensional scaling (MDS) of similarity judgments about pairs of affect-laden words or emotional facial expressions, have repeatedly yielded a number of underlying dimensions, with two dimensions accounting for the vast majority of the observed variance in the emotional labeling of experimental stimuli. According to the circumplex model of affect (Posner et al., 2005; Russell, 1980; Russell, Weiss, & Mendelsohn, 1989), these two dimensions form a circular array (i.e., a circumplex) of emotional labels representing a pleasure–displeasure continuum along one dimension and arousal level along the other. Thus, every affective experience is the product of a linear combination of these two underlying dimensions, valence and arousal, that two corresponding underlying neurophysiological "systems" are hypothesized to subserve (Feldman Barrett, 1998). The sensations produced by these systems are interpreted, and emotional labels are assigned, on the basis of a cognitive appraisal of recent experiences that may have incited them (see Figure 1).

Our use of the terms *valence* and *arousal* is based on the use of those terms in previous studies of dimensional models (Russell, 2003). We use the term *valence* when referring to the hedonic tone of the subjectively experienced emotions, which may range from highly negative (i.e., unpleasant) emotions, such as terror or despair, to extremely positive (i.e., pleasant) ones, such as joy (Colombetti, 2005; Russell, 2003). Thus, we distinguish *valence* from *approach* and *avoidance*, which, although related to hedonic tone, are conceptually distinct because they refer to observable behaviors. Furthermore, when we use the term *valence*, we describe an attribute of psychological experience, rather than features of physical stimuli that may themselves may be either pleasant (warm temperature) or unpleasant (scalding temperature) when experienced, and that may correlate in various ways with the reporting of pleasant or unpleasant emotions. Finally, we distinguish *valence* from *salience*, with the latter referring to the extent that a stimulus attracts attentional resources (Kastner & Ungerleider, 2000).

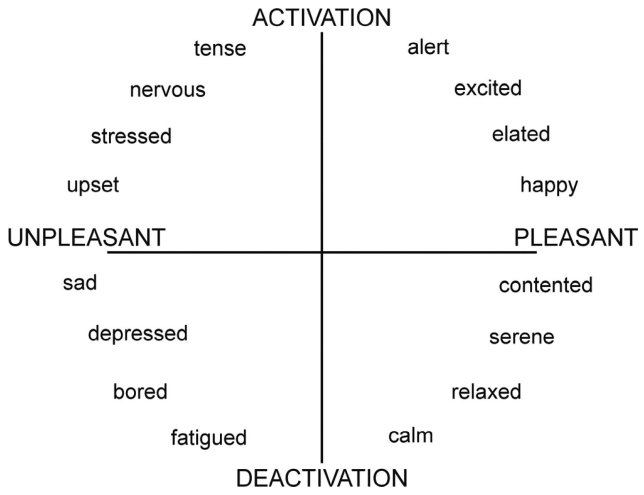

Figure 1. A schematic representation of the affective circumplex. The horizontal axis represents the valence dimension (pleasure–unpleasure), and the vertical axis represents the arousal dimension. We have superimposed the names of some discrete emotions onto the circumplex space to emphasize that the two models are not mutually exclusive but simply different conceptual frameworks.

By *arousal*, we mean the neurophysiological alertness or the state of responsiveness of a person to sensory stimuli (Kandel, Schwartz, & Jessell, 2000). We distinguish several levels of arousal, such as coma, stupor, sleep, vigilance, and panic. Attention and arousal can be intercorrelated, but they are dissociable (Sarter, Givens, & Bruno, 2001), in that attention refers specifically to the allocation of cognitive resources, which either may or may not correlate with the level of arousal (e.g., a patient in an agitated delirium has high arousal but has grossly impaired attention). Although arousal can also be defined operationally through measures of peripheral autonomic activity, hereinafter we refer to *arousal* strictly as an attribute of an internally experienced feeling (Russell, 2003), consistent with its use in dimensional literature, where it is construed as "energy" or "activation" (Lang & Davis, 2006). In this sense, arousal is not a property of the stimulus but rather of the feeling experienced.

We have previously reported two functional magnetic resonance imaging (fMRI) studies on the same sample of patients using emotion-denoting words (Posner et al., 2009) and emotional faces (Gerber et al., 2008). In these two previous studies, participants were asked to rate the emotional value of the stimulus (a face or word). Rating the emotional value of word and face stimuli, however, may risk activating semantic or facial processing networks. Therefore, in the present study, we instead explicitly asked participants to rate online the arousal and valence of their emotions as they imagined themselves in various paradigmatically emotion-evoking situations. Although similar emotion induction paradigms have been used in previous studies of affect (Gilet, 2008; Harrison et al., 2008), some of these have used subtraction rather than parametric designs, and they have focused on the induction of a limited set of emotions, mostly sadness (Liotti, Mayberg, McGinnis, Brannan, & Jerabek, 2002; Segal et al., 2006), or they have probed the two dimensions separately rather than simultaneously. Subtraction paradigms compare a single emotional stimulus, such as fear or joy, with a baseline condition. They are not ideally suited to the study of emotions because establishing a true neutral baseline devoid of emotional value for all participants is difficult, and any discrepancies in the subjective experience of the stimuli from the emotion intended in the design of the paradigm will impair the ability to isolate brain regions that subserve the specific emotion being studied. Finally, because the affective circumplex model was initially developed using either emotional faces or emotion-denoting words, we reasoned that the use of evocative sentences would have the advantage of being a stimulus type that had not been used to derive the circumplex model that we wanted to test.

The aim of this study was to investigate whether subjective ratings of valence and arousal during the experience of a wide

range of induced emotions would relate differentially to distinct neural systems, as predicted by the circumplex model of affect. We used a parametric design (Buchel, Holmes, Rees, & Friston, 1998) to study how the fMRI-based BOLD (blood oxygen level-dependent) signal varied with online ratings of valence and arousal for emotions experienced with each stimulus by each participant. Because we treated valence and arousal as continuous psychological dimensions rather than as behavioral responses (e.g., approach and avoidance) and because we used a parametric design, we did not analyze positive and negative valence or high and low arousal separately, as is frequently done in studies using block designs and a subtraction paradigm. On the basis of previous imaging studies of emotion, we hypothesized that brain regions where the BOLD signal is associated with arousal ratings would include the amygdala (Anderson et al., 2003; Small et al., 2003) and the thalamus (Anders, Lotze, Erb, Grodd, & Birbaumer, 2004; Heilman, 2000; Huguenard & McCormick, 2007; Portas et al., 1998), whereas regions where the BOLD signal is associated with valence ratings would include prefrontal and cingulate cortices (Beauregard, Levesque, & Bourgouin, 2001; Levesque, Eugene, et al., 2003; Levesque, Joanette, et al., 2003; Mayberg et al., 1999). Our approach builds on numerous previous studies using similar parametric methods to study dimensional aspects of affective experience (Cunningham, Raye, & Johnson, 2004; Lewis, Critchley, Rotshtein, & Dolan, 2007). We extend, however, those previous approaches by introducing a design in which participants rate their subjective valence and arousal responses to each emotional stimulus, directly in circumplex space and while they are in the scanner.

Method

Sample

The procedures of this study were approved by the Institutional Review Board of the New York State Psychiatric Institute. Participants were recruited through advertisements in the New York City area. Ten control participants were scanned (5 men, 5 women; mean age = 25.51 ± 4.58 years; age range = 19–34 years). Each was screened using the Structured Clinical Interview for *DSM–IV* (SCID-IV; Spitzer & Gibbon, 1995) to ensure the absence of psychiatric disorders. The Abbreviated Scale of Intelligence was used to obtain estimates of full-scale IQ, and socioeconomic status was assessed with the Hollingshead Index of Social Status (Hollingshead, 1975). Handedness was assessed with the Edinburgh Handedness Inventory (Oldfield, 1971). Written informed consent was obtained from each participant, and all were paid for their participation. All were right-handed, Caucasian, and native English speakers, with IQs slightly above average (mean full-scale IQ = 112.4 ± 13.7). Participants belonged to households of high average socioeconomic status, and all were medication free.

Behavioral Task

Each trial of the task consisted of three time blocks: (1) For 30 s, participants were shown a sentence intended to evoke a specified emotional state (e.g., "Imagine that you just won the lottery and you will have all the money you could ever want"; a list of all sentences used is included online in supplementary Table S1). The emotionally evocative sentences were selected to probe all four quadrants of the affective circumplex. Participants were asked to try to experience the affective state that they would feel if the situation were real (Velten, 1968). To accomplish this induction of an emotion, we gave participants the following instruction: "Try to think about how the emotion feels. Some people think about situations, and others draw on memories of situations that have made them feel the emotion in the past." They were not asked to close their eyes while performing the task, because doing so and then requiring them to open their eyes afterward to rate their emotion would have introduced unnecessary noise due to motor actions. (2) Participants were then shown a 9 × 9 grid displaying the dimensions of valence and arousal as visual analogue scales on the *x*- and *y*-axes, respectively, ranging from 1 to 9 in each dimension. (3) Participants gazed at a cross-hair at the center of a blank screen for variable durations, so that the time from offset of the previous evocative sentence to the presentation of the next sentence (i.e., the total time for rating and gaze fixation) equaled 20 s. The 9 × 9 grid used to obtain affective ratings allowed participants to rate simultaneously both arousal and valence. The participants selected the ratings directly on the grid using a mouse while in the scanner (during Part 2 of each trial). Because this affective grid provides ratings of valence and arousal similar to those obtained when the two dimensions are rated independently (Posner et al., 2005), we elected not to have participants rate these two dimensions separately as different tasks. We conducted two scanning runs, each comprising 15 stimulus presentations of emotionally evocative sentences. We used a pseudorandomized sequence of the emotion induction sentences, with the constraint that the 15 sentences in each run were each presented once. The order of stimulus presentation was maintained across participants. Two runs per participant yielded a total of 30 stimuli for each participant.

All behavioral stimuli were presented through LCD goggles (Resonance Technology Inc., Northridge, CA). EPrime software (Version 1.0; Psychology Software Tools Inc., Pittsburgh, PA) was used for the presentation of behavioral stimuli and for recording responses. Participants made responses with their right hand using a computer mouse modified for use in the MRI environment. Before the scanning session, task instructions were explained to each participant, and each practiced a version of the task using emotionally evocative sentences different from those presented during the scanning session. Each participant practiced with five stimulus presentations outside the scanner to ensure that they understood the instructions and were able to perform the ratings.

Image Acquisition

Images were acquired on a 3.0T Signa GE scanner (Milwaukee, WI) operating the E2-M4 platform and using a quadrature head coil. Acquisition of oblique echoplanar images oriented parallel to the anterior commissure–posterior commissure (AC-PC) line was achieved using T1-weighted scout images. A 3D high-resolution fast spoiled gradient recall (fSPGR) image was acquired for coregistration with echoplanar images and for normalization with a reference brain from the Montreal Neurological Institute (MNI). Axial functional images were obtained with a $T2^*$- weighted gradient recalled single shot echoplanar pulse sequence with a repetition time of 2.8 s, echo time of 25 ms, a 90° flip angle, single

excitation per image, 24 × 24-cm field of view, a 64 × 64 matrix, a slice thickness of 3 mm and a 0.5-mm gap to provide an effective resolution of 3.75 × 3.75 × 3.5 mm. A total of 273 volumes per run were obtained, with each volume comprising 43 axial oblique slices acquired in interleaved order to provide whole brain coverage.

Image Processing

The processing of images and statistical analyses used SPM2 on a MATLAB 6.5 platform. All images were inspected visually and discarded if artifacts such as ghosting were found. According to standard fMRI procedures, the six initial volumes (dummy scans) were also eliminated from the original series. We applied slice-timing correction, with the middle slice of each run used as the reference image for motion correction of all other functional images using three translational directions and rotation (Friston, Ashburner, et al., 1995). Motion-corrected echoplanar images were then coregistered with the same participant's SPGR image and subsequently normalized to the MNI template using a hybrid algorithm of affine transform and nonlinear warping, with reslicing into a 2 × 2 × 2-mm resolution space. Normalized images underwent Gaussian spatial filtering (full-width, half maximum 8 mm). We removed drift from the baseline image intensity using a discrete cosine transform-based high-pass temporal filter with a basis function length of 128 s and then removed high-frequency noise with a low-pass Butterworth filter with a cutoff frequency of 0.15 Hz. We did not apply grand mean scaling or intensity normalization.

Statistical Analysis

The time course of the BOLD response at each voxel was modeled by convolving a boxcar function (BCF), representing the duration of stimulus presentation, with the canonical hemodynamic response function (HRF; Friston, Holmes, et al., 1995). This convolved function was then parametrically weighted in SPM2 with the parametric modulation function, which consists of weighting (i.e., multiplying) the amplitude of the convolved function by the value of a specified parameter according to a relationship (e.g., linear or quadratic) that is assumed a priori. Parametric modulations are used to determine the effects across different levels of a specified parameter (Buchel, Holmes, Rees, & Friston, 1998).

We modeled the time series data for each participant using five independent functions and a constant. The variables were (a) the canonical HRF convolved with the BCF indexing stimulus presentation (as given earlier), which we term *Function A*; (b) Function A weighted by the arousal rating for each stimulus; (c) Function A weighted by the valence rating for each stimulus; (d) the canonical HRF convolved with a BCF indexing the presentation of the 9 × 9 response grid; and (e) the canonical HRF convolved with a BCF indexing gaze fixation. Interactions were modeled with a regressor representing the product of valence and arousal convolved with the canonical HRF. We modeled at each voxel a linear relationship between the online ratings of arousal and valence with the time-varying BOLD signal. Neither euclidean normalization nor parameter orthogonalization was applied. We decided not to orthogonalize the dimensions because orthogonalization was neither statistically necessary nor useful. It was unnecessary because, according to statistical theory, although some covariates are correlated, one does not need to orthogonalize them to interpret the statistical significance of these covariates individually. More important, orthogonalizing one variable over the other arbitrarily assigns shared variance to the regressor onto which the second variable is orthogonalized, which renders interpretation either difficult or impossible in terms of the affective circumplex.

Our findings did not change appreciably when we analyzed the data using orthogonalization or when we reversed the order of the valence or arousal regressors in the design matrix, indicating the robustness of the findings to model specification. We determined voxel-based correlation estimates for each participant using ordinary least squares. By modeling the stimulus presentation, rating, and fixation each as separate epochs, we ensured that our analyses were not confounded by the motor activity associated with mouse-clicking during the rating of valence and arousal.

We conducted two levels of analysis. The first assessed whether the BOLD signal was associated significantly with ratings of emotional valence and arousal *within* each participant. In this level, we used the general linear model to estimate the parameter estimates of the regressor of interest (valence or arousal). We established these associations within participants using 30 stimulus presentations over a total of 546 imaging volumes for each participant. The parameter estimate for each participant at the first level assessed whether a particular contrast (i.e., a linear combination of parameter estimates) differed significantly from zero.

Subsequently, these contrasts (one for each participant) generated at the first level were entered into a second-level analysis of random effects, in which we accommodated the randomness of differential responses by comparing the mean parameter estimate from the first-level analysis *across participants* to the variability in the parameter estimate from participant to participant. This particular type of analysis is conservative, but it allows generalization of results to the entire population, as it takes into account interparticipant variance (Ashburner, Friston, & Penny, 2004; Huettel, Song, & McCarthy, 2004). In this second-level analysis, we tested the hypothesis that the first-level contrasts differed significantly from zero at a group level using the "simple *t*-test" function in SPM2. Statistical parametric maps were thresholded with the conjoint requirement of an uncorrected, two-tailed $p < .025$ and a cluster of 790 contiguous voxels, which yielded an effective corrected $p < .05$ across the imaging volume (Cao & Worsley, 2001). The combined application of a statistical threshold and cluster filter has been shown to reduce substantially the false-positive identification of activated pixels at any given threshold (Forman et al., 1995). A cluster of 790 voxels in the resliced space (2 × 2 × 2-mm resolution) corresponded to a cluster of 128 voxels in the original space (3.75 × 3.75 × 3.5-mm resolution), yielding a cluster volume of 1,027 mm^3, a size consistent with clusters used in previous studies (Tian et al., 2007). Group-level effects were then assessed with an *F* test and a statistical threshold of $p < .001$.

Power Analysis

We conducted a comprehensive power analysis for this study. Associations of the BOLD signal with affective ratings were established in the first-level analysis using the general linear model, using 30 stimulus presentations per participant, with 546 total data points or imaging volumes for each participant (273 data

points per run). The power to detect an effect for each participant in the first-level analyses was high (80% for a two-sided Type I error rate of 0.05).

Contrast images for positive and negative parameter estimates were then entered into the second-level, random effects analysis. For the second-level, random effects analysis, power was moderate (60% for a two-sided, error rate of 0.05), thus risking the presence of false negative (Type II) errors. Because we used a stringent correction for multiple comparisons (Cao & Worsley, 2001), the risk of false-positive errors was low (corrected $p < .05$) (Mumford & Nichols, 2008; Zarahn & Slifstein, 2001). Thus, our power analysis indicated that the effects detected were likely to be real, although we may have missed detection of other real effects.

FIR Analysis

In addition to using the classic time derivatives of the HRF to model the fMRI time series, we also applied a finite impulse response (FIR) analysis to model different temporal components of the BOLD response and to ensure that our findings were robust to the specific modeling technique. FIR models signal changes with a set of basis functions to capture a range of time courses (Windischberger et al., 2008). We used two basis functions to model the time course of the BOLD signal, as previously described (Dale & Buckner, 1997), modulating each basis function with individual valence and arousal ratings in a manner identical to our HRF analysis. The lag between the peak of the first and the peak of the second basis function was 14 s (approximately five imaging volumes with a repetition time [TR] of 2.8 s), allowing us to model an early (FIR 1) and a delayed component (FIR 2) of the BOLD signal response within the 30 s of stimulus presentation. Contrasts were generated for second-level analyses in a manner analogous to those for the HRF analysis, except that two regressors were available for valence and arousal in the FIR analysis.

Results

Behavioral Results

Online ratings of valence and arousal indicated that the paradigm successfully probed emotions from all four quadrants of the affective circumplex (see Figure 2 and Table 1). We calculated correlations between ratings on the two dimensions of the circumplex. These correlations were low (valence with arousal: $r = -0.064$, $p = .266$; $N = 300$, $p = .266$), consistent with a model in which the dimensions of arousal and valence are largely independent. Residual correlations in our regression model were expected, however, given that response dimensions can never be made perfectly independent. Although the range of possible arousal ratings varied from −4 to 4 and valence ratings varied from 1 to 9, this difference in ranges is irrelevant to modeling because correlations (in this case, with the BOLD signal) are not affected by linear transformations of the data.

Valence

Significant inverse associations of valence ratings with the BOLD signal intensities were detected in the right dorsolateral prefrontal cortex and frontal pole, anterior midcingulate cortex (aMCC, also termed the *rostral dorsal ACC* in previous studies), supplementary motor area (SMA), right occipito-temporal junction, inferior parietal cortex, and right cerebellar hemisphere (see Figure 3). Thus, BOLD signal intensity in these regions increased as valence ratings declined and as the induced emotion was expe-

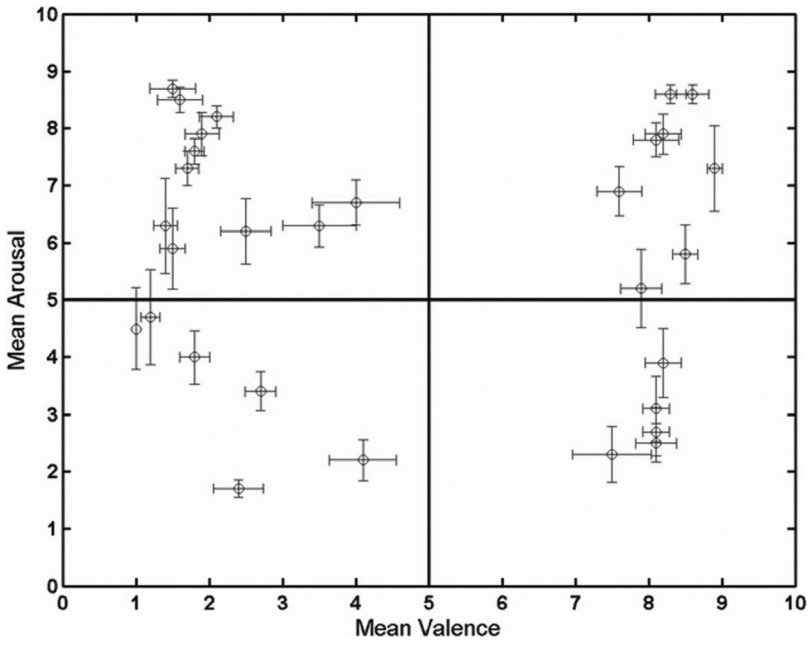

Figure 2. Mean ratings for each stimulus type across all 10 participants plotted in circumplex space. Error bars represent standard errors. Mean valence scores are reported on the *x*-axis, and mean arousal scores are reported on the *y*-axis.

Table 1
Mean Valence and Arousal Ratings Reported With Their Standard Errors for Each Stimulus Type

Stimulus type (sentence number)	*n*	Run	Arousal		Valence	
			M	*SEM*	*M*	*SEM*
26	10	1	1.70	0.153	2.40	0.340
8	10	1	5.80	0.512	8.50	0.167
3	10	1	7.60	0.221	1.80	0.133
11	10	1	6.30	0.831	1.40	0.163
19	10	1	8.50	0.224	1.60	0.306
23	10	1	4.50	0.719	1.00	0.000
21	10	1	3.40	0.340	2.70	0.213
22	10	1	5.90	0.706	1.50	0.167
18	10	1	8.70	0.153	1.50	0.307
5	10	1	2.50	0.342	8.10	0.277
25	10	1	4.70	0.831	1.20	0.133
15	10	1	8.60	0.163	8.60	0.221
30	10	1	8.20	0.200	2.10	0.233
2	10	1	7.30	0.300	1.70	0.153
17	10	1	7.30	0.746	8.90	0.100
11	10	2	8.60	0.163	8.30	0.213
24	10	2	4.00	0.471	1.80	0.200
9	10	2	3.90	0.605	8.20	0.249
10	10	2	5.20	0.680	7.90	0.277
16	10	2	6.90	0.433	7.60	0.306
29	10	2	6.70	0.396	4.00	0.596
20	10	2	7.90	0.379	1.90	0.233
13	10	2	7.80	0.291	8.10	0.314
27	10	2	2.30	0.496	7.50	0.543
4	10	2	2.20	0.359	4.10	0.458
7	10	2	2.70	0.423	8.10	0.180
14	10	2	7.90	0.348	8.20	0.249
1	10	2	6.20	0.573	2.50	0.342
28	10	2	6.30	0.367	3.50	0.500
6	10	2	3.10	0.567	8.10	0.180

Note. Each stimulus was presented once to each participant for a total of 10 presentations per stimulus. The run (1 or 2) in which the stimulus appeared is also indicated. Sentence numbers indicate which sentence was presented to participants (refer to Table S1).

rienced as increasingly unpleasant. No significant positive associations of valence with the BOLD signal were detected.

Arousal

Significant positive associations of arousal ratings with BOLD signal intensities were detected in the left thalamus, left caudate nucleus, left globus pallidus, left parahippocampal gyrus, left amygdala, premotor cortex, occipito-temporal junction, and cerebellar vermis (see Figure 4). BOLD signal intensity in these regions increased as the induced emotion was experienced as increasingly more arousing. No significant inverse associations were detected.

Interactions

We detected no significant interaction of valence with arousal.

Lateralization Effects

The findings suggested that valence and arousal systems might be lateralized. To test whether this was indeed the case, we performed a post hoc analysis in which we flipped in the left–right direction the unthresholded, first-level contrasts for either valence or arousal in each participant. We then performed a paired *t* test between 10 pairs of first-level contrasts, with each pair composed of the flipped and the unflipped first-level contrasts for the same participant. A group-level paired *t* test did not detect any significant asymmetry in activation across hemispheres. Viewing the results of the second-level analysis at a reduced statistical threshold indicated that the apparent lateralization was an artifact of thresholding of the statistical and cluster thresholds, as symmetric activations that were weaker and of smaller spatial extent were observed in the contralateral hemispheres.

FIR Analyses

FIR analyses for the early component of the BOLD signal (FIR 1) confirmed the results obtained with the more conventional HRF method. Associations of the second delayed component (FIR 2) were significant for valence ratings but not arousal ratings. Greater BOLD signal of the delayed component accompanied increasing valence ratings in the midbrain, ventral striatum, and right caudate nucleus (refer to Fig. S1 in the online supplemental data).

Discussion

Our findings indicated that dimensional ratings of valence and arousal during the experience of induced emotions correlated with

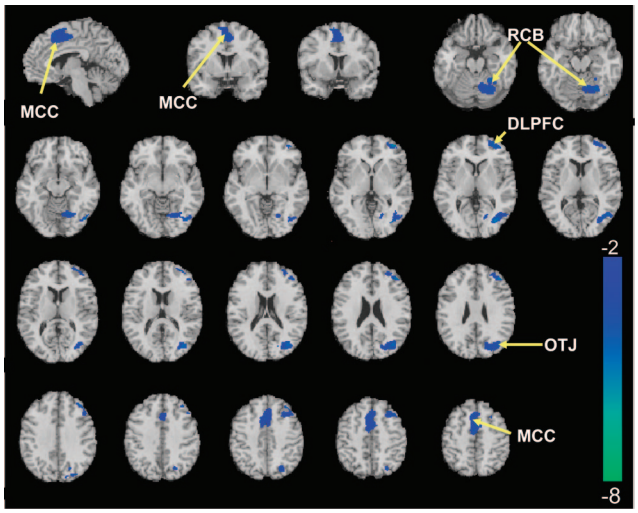

Figure 3. Statistical parametric maps showing areas where BOLD (blood oxygen level-dependent) signal intensity is associated with valence ratings. From left to right and top to bottom: 1 sagittal section, 2 coronal sections, and 18 axial sections. Inverse associations are in cold colors. Different shades of colors correspond to different *T* values at each voxel. Specifically, cold colors represent negative *T* values ranging from −2 (dark blue) to −8 (light blue). Images are displayed according to the neurological convention (the left and right side of the figure correspond to the left and right side of the brain). MCC = midcingulate cortex/rostral dorsal ACC/SMA; RCB = right cerebellum. DLPFC = dorsolateral prefrontal cortex; OTJ = occipito-temporal junction.

activity in discrete sets of neural structures, consistent with the predictions of the circumplex model of affect. We parametrically varied the emotional valence and arousal of affect-inducing stimuli to assess whether variations in BOLD signal intensity levels were associated linearly with variations in valence or arousal.

Valence

Our findings suggest that more unpleasant emotions induced greater neural activity in a set of regions that included the dorsolateral prefrontal cortex (DLPFC), frontal pole, the aMCC/rostrodorsal ACC, supplementary motor area, occipito-temporal junction, and cerebellar hemisphere (see Figure 3 and Table 2). BOLD signal intensity in these regions increased progressively with greater degrees of unpleasantness.

Both the MCC and SMA may constitute an interface between limbic and motor-executive systems. The location of the SMA and its connections with the limbic system suggest that this region supports the transformation of affective experiences into complex motor plans (Oliveri et al., 2003). The SMA belongs to a family of premotor cortices that lie adjacent to the superior portion of the MCC and that are thought to be involved in the planning and selection of complex sets of movements. Whereas movements initiated by external cues are mostly associated with activity in the lateral premotor cortex, internally generated or mentally rehearsed movements preferentially engage the SMA (Kandel et al., 2000). The MCC, on the other hand, lies just inferior to the SMA, projecting to motor cortices, spinal cord, amygdala, and other portions of the cingulate cortex that also support emotions and their cognitive correlates (Vogt, 2005). Along with the anterior cingulate (ACC), posterior cingulate (PCC), and retrosplenial cortex (RSC), the MCC is one of four cytoarchitectonic subdivisions of the cingulate cortex (Vogt, Berger, & Derbyshire, 2003; Vogt & Pandya, 1987). The aMCC, in particular, is thought to mediate the transfer of anticipated negative consequences into motor plans, and it is likely involved in the generation of subsequent avoidant behaviors, given its tight anatomical connections with the amygdala (Vogt, 2005). Gray matter volumes in the anterior midcingulate cortex, as well as DLPFC, have been found to correlate inversely with depressive symptoms in people with major depression (Chen et al., 2007), and changes in default-mode network connectivity during self-generated changes in mood also implicate this region in the experience of negative emotion (Harrison et al., 2008).

Activation of the DLPFC by unpleasant stimuli has been reported in previous studies (Kensinger & Schacter, 2006; Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001), consistent with its well-documented roles in subserving executive functioning and attention, particularly during responses to threat-related stimuli (Bishop, Duncan, Brett, & Lawrence, 2004). The observed activation of the DLPFC and inferior parietal cortex may reflect their hypothesized roles as key components of the dorsal attentional system that focuses attention on goal-relevant features of a task (Corbetta & Shulman, 2002). Moreover, the DLPFC and frontal pole have long been implicated in regulating emotions (Beauregard, Levesque, & Bourgouin, 2001; Levesque, Eugene, et al., 2003; O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001; Ochsner, Bunge, Gross, & Gabrieli, 2002), in consciously suppressing negative emotions (Levesque et al., 2004), and in mediating the anticipation of unpleasant experiences (Herwig et al.,

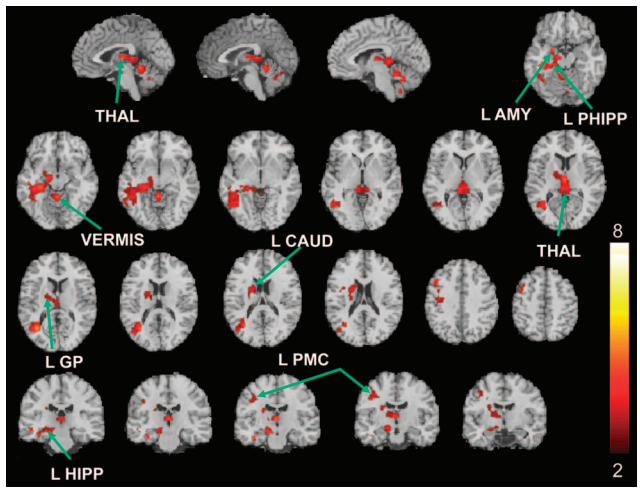

Figure 4. Statistical parametric maps showing areas where BOLD (blood oxygen level-dependent) signal intensity is associated with arousal ratings. From left to right and top to bottom: 3 sagittal sections, 13 axial sections, and 5 coronal sections. Different shades of colors correspond to different *T* values at each voxel. Specifically, warm colors represent positive *T* values ranging from 2 (red) to 8 (yellow). Images are displayed according to the neurological convention. LAMY = left amygdala; L CAUD = left caudate nucleus; L PHIPP = left parahippocampus; L HIPP = left hippocampus; L GP = left globus pallidus; THAL = thalamus.

Table 2
Centers of Activation Within Areas Where BOLD Signal Intensity Shows Significant Direct (Positive) or Inverse Association With Valence Ratings

Anatomical region	Talairach coordinates			BA	*T*
	x	y	*z*		
Positive associations					
None					
Inverse associations					
SMA & MCC/rdACC (bilateral)	0	17.5	39.6	6, 24, 32	3.39
Right frontal pole	37.6	50.4	−0.6	10	6.08
Right occipito-temporal junction & inferior parietal cortex	37.6	−70.9	18.2	37, 39	4.64
Right DLPFC	41.5	39.9	21.9	8, 9	5.27
Right cerebellum	27.7	−60.7	−10.4	N/A	4.49

Note. Corresponding *T* values are reported. Coordinates of the local maxima are reported in Talairach space with the corresponding Brodmann's areas (BA). SMA = supplementary motor area; MCC = midcingulate cortex; rdACC = rostral dorsal anterior cingulate cortex; DLPFC = dorsolateral prefrontal cortex.

2007). PET studies have revealed that the provocation of sad moods is associated with decreases in activity of the right DLPFC in healthy volunteers (Liotti et al., 2002), and reduced DLPFC activation has been reported in depressed persons (Mayberg et al., 1999).

These previous studies suggest that the DLPFC may be recruited during the experience of unpleasant emotions, perhaps because unpleasant emotions induce goal-directed plans for action, usually in the form of withdrawal from the inciting stimulus. Our findings indicate that the more unpleasant the induced emotion, the higher the activity in the right DLPFC and frontal pole. Although this activity may represent a normal homeostatic mechanism for the regulation of mood in healthy individuals, our participants were not instructed to regulate their mood during the experimental paradigm, and therefore we regard the higher BOLD activity in our experiment as likely signaling a greater recruitment of attentional resources, rather than representing a mood-regulating process per se. Of course, we cannot exclude the possibility that regulatory systems may have been activated automatically, even without explicit instruction to suppress negative emotions.

We also observed a strong inverse association between valence ratings and BOLD signal intensity in the inferior parietal cortex and right temporo-occipital junction, a portion of the ventral visual stream connecting visual areas directly to the medial temporal lobe, where visual memories are stored. We note that, in the right hemisphere, this associative cortex is engaged in the assessment of emotional stimuli, particularly negatively valenced ones (Godinho, Magnin, Frot, Perchet, & Garcia-Larrea, 2006). Finally, BOLD signal intensity in the right dorsal cerebellum increased as the degree of unpleasantness of the induced emotion also increased. The cerebellum is known to participate in motor planning and in a variety of executive, visuospatial, and emotional functions (Schmahmann, 2004; Schmahmann & Sherman, 1998). EEG and fMRI studies suggest that a greater degree of aversion to painful stimuli accompanies greater activation of the cerebellum and other areas involved in attending to, planning, initiating, propagating, and executing motor behaviors—regions such as the ACC, SMA, and primary motor cortices (Schmahmann, 2004). Our findings are consistent with those reported in these previous studies, in that highly unpleasant emotions seem to engage the cerebellar hemispheres. Finally, analyses of a delayed component of the BOLD response revealed that greater neural activity accompanies increasingly pleasant emotions in a network that includes the midbrain (mostly the substantia nigra), ventral striatum, and right caudate nucleus, regions frequently implicated in previous studies of affect (Kober et al., 2008). These regions belong to the mesolimbic dopaminergic system that subserves the experience of pleasure and reward (Posner et al., 2009) and appetitive learning (Heimer, 2003; O'Doherty, Critchley, Deichmann, & Dolan, 2003; O'Doherty et al., 2004; O'Doherty, Dayan, Friston, Critchley, & Dolan, 2003).

In summary, we speculate that unpleasant, negatively valenced emotions engage executive-motor and attentional systems, perhaps through the aMCC/rostral dorsal ACC, which coordinates the exchange of emotional information between the SMA, DLPFC, frontal pole, and cerebellar hemispheres, mediating emotional appraisal and preparation for withdrawal from an inciting unpleasant stimulus. We also speculate that, whereas negatively valenced emotions engage preferentially an attentional circuit that is biased toward detecting and responding to unpleasant stimuli, positively valenced emotions may preferentially engage the delayed activation of classic reward circuits.

Arousal

Highly arousing emotions engaged a set of regions that included the left premotor cortex, thalamus, globus pallidus, caudate, amygdala, parahippocampus, hippocampus, and dorsal cerebellar vermis (see Figure 4 and Table 3). We interpret these findings as suggesting that highly intense emotional experiences are stored and retrieved within the medial temporal lobe (McGaugh, 2004; Richardson, Strange, & Dolan, 2004), with the basal ganglia and thalamic nuclei possibly signaling motor planning regions of the cerebral cortex to prepare for action (Aguilar & Castro-Alamancos, 2005; Huguenard & McCormick, 2007).

Activity in both the right amygdala and parahippocampus was associated strongly with arousal ratings. Although activation of the amygdala during the presentation of emotional stimuli has been well documented (LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998; LaBar, LeDoux, Spencer, & Phelps, 1995; Zald, 2003), its precise role in emotional processing and the direction of its acti-

Table 3
Centers of Activation Within Areas Where BOLD Signal Intensity Shows Significant Direct or Inverse Association With Arousal Ratings

Anatomical region	Talairach coordinates			BA	*T*
	x	y	*z*		
Inverse associations					
None					
Positive associations					
Vermis	0	−44.9	−6.1	N/A	5.03
Left amygdala	−21.7	−2.6	−13.3	34, 28	4.31
Left parahippocampus & hippocampus	−27.7	−27.7	−10.3	28	4.86
Premotor cortex	−43.5	18.0	44.0	6	4.41
Left caudate nucleus	−17.8	2.8	18.2	N/A	3.38
Left and right thalamus	0	−16.0	8.1	N/A	4.82
Left globus pallidus	−15.0	−3.4	9.3	N/A	2.62

Note. Corresponding *T* values are reported. Coordinates of the local maxima are reported in Talairach space with the corresponding Brodmann's areas (BA).

vation have been the subject of considerable controversy. The amygdala has been implicated, for example, in mediating emotional responses either to discrete emotions, such as fear (LaBar et al, 1995), or to the properties of emotional cues, such as valence (Garavan, Pendergrass, Ross, Stein, & Risinger, 2001; Killgore & Yurgelun-Todd, 2004), intensity (Anders et al., 2004; Glascher & Adolphs, 2003; Kensinger & Corkin, 2004; Kensinger & Schacter, 2006; Small et al., 2003), or a combination of both valence and intensity (Anders et al., 2004; Kensinger & Corkin, 2004; Winston, Gottfried, Kilner, & Dolan, 2005). Our findings, however, suggest that the amygdala is preferentially involved in the processing of arousal rather than of valence components of emotional stimuli, consistent with numerous previous studies reporting amygdala activation in encoding intensity rather than emotional valence (Anderson et al., 2003; Lewis et al., 2007; Small et al., 2003).

We found that our emotion induction paradigm also activated the hippocampus and parahippocampus, structures that are intimately related to one another anatomically and functionally and that are involved in establishing and maintaining memory traces (Squire, Wixted, & Clark, 2007). Furthermore, animal studies have shown increased cell firing and neurotransmitter release in these medial temporal lobe (MTL) regions during the processing of arousing stimuli (Acquas, Wilson, & Fibiger, 1996; Green & Arduini, 1954; Inglis & Fibiger, 1995), perhaps mediating the increase of overall behavioral alertness. Recruitment of the MTL with increasing arousal is consistent with previous theories that memory traces must be activated upon presentation of emotionally relevant stimuli, so that these stimuli can be recognized, related to past and present contextual experiences, and ultimately assigned an emotion-denoting label (McGaugh, 2004; Paz, Pelletier, Bauer, & Pare, 2006; Pelletier & Pare, 2004). The concurrent activation of the amygdala, hippocampus, and parahippocampus further corroborates previous suggestions that the amygdala-hippocampal network is a coordinated neural system that supports the effects of emotional arousal on the consolidation of affectively charged memories (Adolphs et al., 2005; Kensinger & Corkin, 2004; Kilpatrick & Cahill, 2003; LaBar & Cabeza, 2006; LaLumiere, Buen, & McGaugh, 2003). The basolateral amygdala may modulate memory consolidation in this system, whereas the hippocampus and parahippocampus support the processing of personal memories (Malin & McGaugh, 2006; McGaugh, 2004; Phelps, 2004).

The thalamus was also activated by our paradigm, likely because it mediates arousal functions in both emotional and nonemotional tasks, as suggested by previous findings that peripheral physiological indicators and subjective reports of arousal correlate with thalamic activity (Anders et al., 2004; Portas et al., 1998). The thalamus belongs to a set of midline structures that process conditioned fear stimuli (LaBar & Cabeza, 2006) and that serve as critical links between the hippocampus, which receives sensory inputs from primary sensory cortices, and the medial prefrontal cortex (Vertes, Hoover, Szigeti-Buck, & Leranth, 2007). Thus, the thalamus is an integral part of a distributed neural network that uses sensory information to alert the organism to stimuli that are emotionally arousing and therefore behaviorally relevant. Moreover, the reticular nucleus within the thalamus is uniquely positioned to modulate the transfer of sensory information to the cortex, consistent with the well-known role of the thalamus in regulating the overall activity of the cortical mantle (Heilman, 2000; Huguenard & McCormick, 2007).

We found that arousing emotional experiences engage the left caudate nucleus and the globus pallidus, perhaps reflecting the unique role of the basal ganglia in the learning of stimulus–response associations (Featherstone & McDonald, 2005; Rogan, Leon, Perez, & Kandel, 2005). We speculate that activation of these basal ganglia networks may encode the procedural memory traces and stimulus–response associations that accompany intense emotional experiences (Steidl, Mohi-uddin, & Anderson, 2006). Finally, arousal ratings were associated with BOLD signal intensity in the cerebellar vermis, which participates in motor control, attentional processes (Bobee, Mariette, Tremblay-Leveau, & Caston, 2000), and modulation of fear-related behaviors (Albert, Dempesy, & Sorenson, 1985; Supple, Leaton, & Fanselow, 1987). The cerebellar vermis is also regarded as the "limbic cerebellum" (Schmahmann, 2004), as lesions of this structure have been implicated in producing the prominent emotional dysregulation that is characteristic of the "cerebellar cognitive-affective syndrome" (Schmahmann & Sherman, 1998).

In summary, we speculate that highly arousing emotions activate an amygdalo-hippocampal-parahippocampal system that likely stores intense emotional experiences in long-term memory (Dolcos, LaBar, & Cabeza, 2004). Arousing emotions also activate a subcortical circuit that guides the learning and shaping of associations between intensely arousing stimuli and their appropriate behavioral responses.

Lateralization

Negatively valenced stimuli seemed to engage structures located predominantly within the right hemisphere, whereas highly arousing stimuli appeared to activate neural systems primarily within the left hemisphere. Although the increasing activation of right-sided structures during the processing of increasingly unpleasant stimuli that we observed is consistent with a long-noted preferential role of the right hemisphere (the frontal pole in particular) in processing negatively valenced stimuli (Canli, Desmond, Zhao, Glover, & Gabrieli, 1998; Demaree, Everhart, Youngstrom, & Harrison, 2005; Smith & Bulman-Fleming, 2005), our analysis indicated that this lateralization was likely the consequence of statistical thresholding, which excluded smaller and less significant clusters of activation in the contralateral hemisphere.

Comparisons With Findings Using Word and Face Stimuli

Our findings differed somewhat from those reported using emotion-denoting word and face stimuli in these same participants. This is not surprising, given the nonisomorphic nature of these stimuli. We note, moreover, that comparing the findings across these three studies may be misleading, because the activation maps have not been compared statistically to determine whether regional activations differed according to stimulus type. Nevertheless, some regions were engaged by all three types of stimuli. These included the amygdala, cingulate gyrus, prefrontal cortex, and dorsal cerebellum, all of which have been implicated previously in the processing of emotions. Their parametric association with subjective ratings of valence or arousal across a wide range of stimuli is unlikely to reflect merely the processing of lexical and other nonemotional information. The induction of emotions produced statistically much stronger associations between the BOLD signal and affective ratings compared with associations when using face or word stimuli. We attribute our more robust findings to the fact that participants experienced much stronger subjective emotions during emotion induction than they did when evaluating the emotion associated with a word or face stimulus.

Comparison With Findings From Previous Meta-Analyses

Our findings are generally consistent with those reported in previous meta-analyses of emotional activation of the brain. One study, for example, identified coherent functional groups that likely represented large-scale networks for emotion processing (Kober et al., 2008). Our valence network overlaps with a cognitive motor group in that report, which included the pre-SMA, and it overlaps with a medial PFC group, which included the rdACC. Our arousal network best approximates the core limbic group reported in this metanalysis, comprising the amygdala, thalamus, and periaqueductal gray. This common set of findings was possible although the meta-analysis did not specifically examine the correlates of valence and arousal.

Limitations

We assessed the processing of emotional stimuli by presenting evocative sentences to the participants, and therefore we cannot exclude the possibility that the task may have engaged semantic processing more readily than may have been engaged by other stimuli, such as affective pictures. Nevertheless, we did not detect activation of areas primarily related to cognitive processing of language and semantic stimuli, which would be expected to include Broca's or Wernicke's areas. Therefore, we are confident that our findings were not driven primarily by semantic processing demands. More important, the parametric design we used deconfounded the effects of linguistic stimuli, which were held constant, with the effects of affective valence and arousal, which were systematically and independently varied according to the classic principles of parametric designs.

More generally, parametric analyses such as ours obviate the methodological limitations of the more traditional subtraction paradigms because they allow one to control for a variety of linguistic and nonlinguistic confounds. Thus, the areas that we identified in our study are regions in which the BOLD signal is specifically associated with variations along the valence and arousal dimensions, and they are unlikely to reflect neural activity associated with semantic processing. We report linear associations of brain activity with valence and arousal ratings because these linear relationships best explained our data. Other models (i.e., quadratic or cubic associations) could be used in future studies, but they accounted for little variance in our present data.

We have restricted our definition of arousal and valence to the meanings assigned to these terms within the tradition of research on the affective circumplex. The terms are conventional labels assigned to dimensions identified in MDS, just as labels are assigned to the factors identified in a factor analysis. Thus, although the terms *approach* and *avoidance* connote components of a somewhat different conceptual construct than the terms *pleasure* and *unpleasure*, each of these terminologies has been ascribed to the same dimensional output of MDS analyses, depending on the experimental context and the theoretical orientation of the investigator. Furthermore, because we were interested in assessing valence as a continuous psychological dimension, we elected not to analyze positive and negative valence separately.

Because this study used subjective ratings of internally generated emotional experiences, we cannot ultimately differentiate activity in the neural systems that generated those emotional states from activity in the neural structures that mediated the generation of their subjective ratings (i.e., the cognitively based judgments about emotions). This limitation applies broadly to most studies in affective neuroscience, however, and not to any specific theoretical model or experimental paradigm. Indeed, judgments are an integral and defining part of any emotional experience and cannot exist in isolation (Pessoa, 2008). Nevertheless, we doubt that activity in dorsal cortices reflected only or primarily attention or other cognitive processes, because the cognitive activity and attention associated with evaluating the reading stimulus and what it

denotes is unlikely to vary systematically and parametrically with ratings of arousal or valence. Therefore, brain activity associated with those cognitive processes would not be registered in our valence or arousal maps to begin with. Finally, because of the conservative nature of the corrections used for our statistical analyses, we may have failed to detect other valid but statistically weaker effects.

Conclusion

Our findings provide biological plausibility for the existence of distinct neural systems that underlie the affective dimensions of valence and arousal, therefore supporting the validity of the circumplex model of affect and its utility in studies of affective neuroscience. Our findings also support a role for the amygdala in encoding the intensity of emotional stimuli rather than the presence of specific emotions.

References

Acquas, E., Wilson, C., & Fibiger, H. C. (1996). Conditioned and unconditioned stimuli increase frontal cortical and hippocampal acetylcholine release: Effects of novelty, habituation, and fear. *Journal of Neuroscience, 16,* 3089–3096.

Adolphs, R., Gosselin, F., Buchanan, T. W., Tranel, D., Schyns, P., & Damasio, A. R. (2005). A mechanism for impaired fear recognition after amygdala damage. *Nature, 433,* 68–72.

Aguilar, J. R., & Castro-Alamancos, M. A. (2005). Spatiotemporal gating of sensory inputs in thalamus during quiescent and activated states. *Journal of Neuroscience, 25,* 10990–11002.

Albert, T. J., Dempesy, C. W., & Sorenson, C. A. (1985). Anterior cerebellar vermal stimulation: Effect on behavior and basal forebrain neurochemistry in rat. *Biological Psychiatry, 20,* 1267–1276.

Anders, S., Lotze, M., Erb, M., Grodd, W., & Birbaumer, N. (2004). Brain activity underlying emotional valence and arousal: A response-related fMRI study. *Human Brain Mapping, 23,* 200–209.

Anderson, A. K., Christoff, K., Stappen, I., Panitz, D., Ghahremani, D. G., Glover, G., . . . Sobel, N. (2003). Dissociated neural representations of intensity and valence in human olfaction. *Nature Neuroscience, 6,* 196–202.

Ashburner, J., Friston, K., & Penny, W. (2004). In R. S. W. Frackowiak (Ed. in Chief), *Human brain function* (2nd ed., Part 2). San Diego, CA: Academic Press. Retrieved from http://www.fil.ion.ucl.ac.uk/spm/doc/books/hbf2/

Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience, 21,* RC165.

Berridge, K. C. (2003). *Comparing the emotional brains of humans and other animals.* Oxford, England: Oxford University Press.

Bishop, S., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience, 7,* 184–188.

Bobee, S., Mariette, E., Tremblay-Leveau, H., & Caston, J. (2000). Effects of early midline cerebellar lesion on cognitive and emotional functions in the rat. *Behavioural Brain Research, 112,* 107–117.

Buchel, C., Holmes, A. P., Rees, G., & Friston, K. J. (1998). Characterizing stimulus-response functions using nonlinear regressors in parametric fMRI experiments. *Neuroimage, 8,* 140–148.

Calder, A. J., Lawrence, A. D., & Young, A. W. (2001). Neuropsychology of fear and loathing. *Nature Reviews Neuroscience, 2,* 352–363.

Canli, T., Desmond, J. E., Zhao, Z., Glover, G., & Gabrieli, J. D. (1998). Hemispheric asymmetry for emotional stimuli detected with fMRI. *Neuroreport, 9,* 3233–3239.

Cannon, W., & Britton, S. (1925). Pseudoaffective meduli-adrenal secretion. *American Journal of Physiology, 72,* 283–294.

Cao, J., & Worsley, K. J. (2001). Applications of random fields in human brain mapping. In M. Moore (Ed.), *Springer Lecture Notes in Statistics* (Vol. 159, pp. 170–182). New York: Springer.

Chen, C. H., Ridler, K., Suckling, J., Williams, S., Fu, C. H., Merlo-Pich, E., & Bullmore, E. (2007). Brain imaging correlates of depressive symptom severity and predictors of symptom improvement after antidepressant treatment. *Biological Psychiatry, 62,* 407–414.

Colombetti, G. (2005). Appraising valence. *Journal of Consciousness Studies, 12,* 103–126.

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3,* 201–215.

Cunningham, W. A., Raye, C. L., & Johnson, M. K. (2004). Implicit and explicit evaluation: fMRI correlates of valence, emotional intensity, and control in the processing of attitudes. *Journal of Cognitive Neuroscience, 16,* 1717–1729.

Dale, A., & Buckner, R. (1997). Selective averaging of rapidly presented individual trials using fMRI. *Human Brain Mapping, 5,* 329–340.

Demaree, H. A., Everhart, D. E., Youngstrom, E. A., & Harrison, D. W. (2005). Brain lateralization of emotional processing: Historical roots and a future incorporating "dominance." *Behavioral and Cognitive Neuroscience Reviews, 4,* 3–20.

Dolcos, F., LaBar, K. S., & Cabeza, R. (2004). Interaction between the amygdala and the medial temporal lobe memory system predicts better memory for emotional events. *Neuron, 42,* 855–863.

Ekman, P. (1992a). An argument for basic emotions. *Cognition and Emotion, 6,* 169–200.

Ekman, P. (1992b). Are there basic emotions? *Psychological Review, 99,* 550–553.

Featherstone, R. E., & McDonald, R. J. (2005). Lesions of the dorsolateral or dorsomedial striatum impair performance of a previously acquired simple discrimination task. *Neurobiology of Learning and Memory, 84,* 159–167.

Feldman Barrett, L. R., & Russell, J. A. (1998). Independence and bipolarity in the structure of current affect. *Journal of Personality and Social Psychology, 74,* 976–984.

Forman, S. D., Cohen, J. D., Fitzgerald, M., Eddy, W. F., Mintun, M. A., & Noll, D. C. (1995). Improved assessment of significant activation in functional magnetic resonance imaging (fMRI): use of a cluster-size threshold. *Magnetic Resonance in Medicine, 33,* 636–647.

Friston, K. J., Ashburner, J., Frith, C. D., Poline, J.-B., Heather, J. D., & Frackowiak, R. S. J. (1995). Spatial registration and normalization of images. *Human Brain Mapping, 3,* 165–189.

Friston, K. J., Holmes, A. P., Poline, J. B., Grasby, P. J., Williams, S. C., Frackowiak, R. S., et al. (1995). Analysis of fMRI time-series revisited. *Neuroimage, 2,* 45–53.

Garavan, H., Pendergrass, J. C., Ross, T. J., Stein, E. A., & Risinger, R. C. (2001). Amygdala response to both positively and negatively valenced stimuli. *Neuroreport, 12,* 2779–2783.

Gerber, A. J., Posner, J., Gorman, D., Colibazzi, T., Yu, S., Wang, Z., . . . Peterson, B. S. (2008). An affective circumplex model of neural systems subserving valence, arousal, and cognitive overlay during the appraisal of emotional faces. *Neuropsychologia, 46,* 2129–2139.

Gilet, A. L. (2008). Procédures d'induction d'humeurs en laboratoire : une revue critique [Mood induction procedures: A critical review]. *L'Encephale, 34,* 233–239.

Glascher, J., & Adolphs, R. (2003). Processing of the arousal of subliminal and supraliminal emotional stimuli by the human amygdala. *Journal of Neuroscience, 23,* 10274–10282.

Godinho, F., Magnin, M., Frot, M., Perchet, C., & Garcia-Larrea, L. (2006). Emotional modulation of pain: Is it the sensation or what we recall? *Journal of Neuroscience, 26,* 11454–11461.

Green, J. D., & Arduini, A. A. (1954). Hippocampal electrical activity in arousal. *Journal of Neurophysiology, 17,* 533–557.

Harrison, B. J., Pujol, J., Ortiz, H., Fornito, A., Pantelis, C., & Yucel, M. (2008). Modulation of brain resting-state networks by sad mood induction. *PLoS ONE, 3,* e1794.

Heilman, K. (2000). *Cognitive neuroscience of emotion.* New York: Oxford University Press.

Heimer, L. (2003). A new anatomical framework for neuropsychiatric disorders and drug abuse. *American Journal of Psychiatry, 160,* 1726–1739.

Herwig, U., Baumgartner, T., Kaffenberger, T., Bruhl, A., Kottlow, M., Schreiter-Gasser, U., . . . Rufer, M. (2007). Modulation of anticipatory emotion and perception processing by cognitive control. *Neuroimage, 37,* 652–662.

Hollingshead, A. (1975). *Four-factor index of social status.* New Haven, CT: Yale University Press.

Huettel, S. A., Song, A. W., & McCarthy, G. (2004). Functional magnetic resonance imaging. Sunderland, MA: Sinauer Associates.

Huguenard, J. R., & McCormick, D. A. (2007). Thalamic synchrony and dynamic regulation of global forebrain oscillations. *Trends in Neurosciences, 30,* 350–356.

Inglis, F. M., & Fibiger, H. C. (1995). Increases in hippocampal and frontal cortical acetylcholine release associated with presentation of sensory stimuli. *Neuroscience, 66,* 81–86.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed.). New York: McGraw-Hill, Health Professions Division.

Kastner, S., & Ungerleider, L. G. (2000). Mechanisms of visual attention in the human cortex. *Annual Review of Neuroscience, 23,* 315–341.

Kensinger, E. A., & Corkin, S. (2004). Two routes to emotional memory: Distinct neural processes for valence and arousal. *Proceedings of the National Academy of Sciences of the United States of America, 101,* 3310–3315.

Kensinger, E. A., & Schacter, D. L. (2006). Processing emotional pictures and words: Effects of valence and arousal. *Cognitive, Affective, and Behavioral Neuroscience, 6,* 110–126.

Killgore, W. D., & Yurgelun-Todd, D. A. (2004). Activation of the amygdala and anterior cingulate during nonconscious processing of sad versus happy faces. *Neuroimage, 21,* 1215–1223.

Kilpatrick, L., & Cahill, L. (2003). Amygdala modulation of parahippocampal and frontal regions during emotionally influenced memory storage. *Neuroimage, 20,* 2091–2099.

Kober, H., Barrett, L. F., Joseph, J., Bliss-Moreau, E., Lindquist, K., & Wager, T. D. (2008). Functional grouping and cortical-subcortical interactions in emotion: A meta-analysis of neuroimaging studies. *Neuroimage, 42,* 998–1031.

LaBar, K. S., & Cabeza, R. (2006). Cognitive neuroscience of emotional memory. *Nature Reviews Neuroscience, 7,* 54–64.

LaBar, K. S., Gatenby, J. C., Gore, J. C., LeDoux, J. E., & Phelps, E. A. (1998). Human amygdala activation during conditioned fear acquisition and extinction: A mixed-trial fMRI study. *Neuron, 20,* 937–945.

LaBar, K. S., LeDoux, J. E., Spencer, D. D., & Phelps, E. A. (1995). Impaired fear conditioning following unilateral temporal lobectomy in humans. *Journal of Neuroscience, 15,* 6846–6855.

LaLumiere, R. T., Buen, T. V., & McGaugh, J. L. (2003). Post-training intra-basolateral amygdala infusions of norepinephrine enhance consolidation of memory for contextual fear conditioning. *Journal of Neuroscience, 23,* 6754–6758.

Lang, P. J. (1995). The emotion probe. Studies of motivation and attention. *American Psychologist, 50,* 372–385.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review, 97,* 377–395.

Lang, P. J., & Davis, M. (2006). Emotion, motivation, and the brain: Reflex foundations in animal and human research. *Progress in Brain Research, 156,* 3–29.

Lange, K. G., & James, W. (1922). *The emotions.* Baltimore: Williams & Wilkins.

Levesque, J., Eugene, F., Joanette, Y., Paquette, V., Mensour, B., Beaudoin, G., & Beauregard, M. (2003). Neural circuitry underlying voluntary suppression of sadness. *Biological Psychiatry, 53,* 502–510.

Levesque, J., Joanette, Y., Mensour, B., Beaudoin, G., Leroux, J. M., Bourgouin, P., & Beauregard, M. (2003). Neural correlates of sad feelings in healthy girls. *Neuroscience, 121,* 545–551.

Levesque, J., Joanette, Y., Mensour, B., Beaudoin, G., Leroux, J. M., Bourgouin, P., & Beauregard, M. (2004). Neural basis of emotional self-regulation in childhood. *Neuroscience, 129,* 361–369.

Lewis, P. A., Critchley, H. D., Rotshtein, P., & Dolan, R. J. (2007). Neural correlates of processing valence and arousal in affective words. *Cerebral Cortex, 17,* 742–748.

Liotti, M., Mayberg, H. S., McGinnis, S., Brannan, S. L., & Jerabek, P. (2002). Unmasking disease-specific cerebral blood flow abnormalities: Mood challenge in patients with remitted unipolar depression. *American Journal of Psychiatry, 159,* 1830–1840.

Malin, E. L., & McGaugh, J. L. (2006). Differential involvement of the hippocampus, anterior cingulate cortex, and basolateral amygdala in memory for context and footshock. *Proceedings of the National Academy of Sciences of the United States of America, 103,* 1959–1963.

Mayberg, H. S., Liotti, M., Brannan, S. K., McGinnis, S., Mahurin, R. K., Jerabek, P. A., . . . Fox, P. T. (1999). Reciprocal limbic-cortical function and negative mood: Converging PET findings in depression and normal sadness. *American Journal of Psychiatry, 156,* 675–682.

McGaugh, J. L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annual Review of Neuroscience, 27,* 1–28.

Mumford, J. A., & Nichols, T. E. (2008). Power calculation for group fMRI studies accounting for arbitrary design and temporal autocorrelation. *Neuroimage, 39,* 261–268.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An FMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14,* 1215–1229.

O'Doherty, J., Critchley, H., Deichmann, R., & Dolan, R. J. (2003). Dissociating valence of outcome from behavioral control in human orbital and ventral prefrontal cortices. *Journal of Neuroscience, 23,* 7931–7939.

O'Doherty, J., Dayan, P., Schultz, J., Deichmann, R., Friston, K., & Dolan, R. J. (2004). Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science, 304,* 452–454.

O'Doherty, J., Kringelbach, M. L., Rolls, E. T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience, 4,* 95–102.

O'Doherty, J. P., Dayan, P., Friston, K., Critchley, H., & Dolan, R. J. (2003). Temporal difference models and reward-related learning in the human brain. *Neuron, 38,* 329–337.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia, 9,* 97–113.

Oliveri, M., Babiloni, C., Filippi, M. M., Caltagirone, C., Babiloni, F., Cicinelli, P., . . . Rossini, P. M. (2003). Influence of the supplementary motor area on primary motor cortex excitability during movements triggered by neutral or emotionally unpleasant visual cues. *Experimental Brain Research, 149,* 214–221.

Osgood, C. (1969). On the whys and wherefores of E, P, and A. *Journal of Personality and Social Psychology, 12,* 194–199.

Panksepp, J. (1992). A critical role for "affective neuroscience" in resolving what is basic about basic emotions. *Psychological Review, 99,* 554–560.

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

Paz, R., Pelletier, J. G., Bauer, E. P., & Pare, D. (2006). Emotional

enhancement of memory via amygdala-driven facilitation of rhinal interactions. *Nature Neuroscience, 9,* 1321–1329.

Pelletier, J. G., & Pare, D. (2004). Role of amygdala oscillations in the consolidation of emotional memories. *Biological Psychiatry, 55,* 559–562.

Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience, 9,* 148–158.

Phan, K. L., Wager, T., Taylor, S. F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: A meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage, 16,* 331–348.

Phelps, E. A. (2004). Human emotion and memory: Interactions of the amygdala and hippocampal complex. *Current Opinion in Neurobiology, 14,* 198–202.

Portas, C. M., Rees, G., Howseman, A. M., Josephs, O., Turner, R., & Frith, C. D. (1998). A specific role for the thalamus in mediating the interaction of attention and arousal in humans. *Journal of Neuroscience, 18,* 8979–8989.

Posner, J., Russell, J. A., Gerber, A., Gorman, D., Colibazzi, T., Yu, S., . . . Peterson, B. S. (2009). The neurophysiological bases of emotion: An fMRI study of the affective circumplex using emotion-denoting words. *Human Brain Mapping, 30,* 883–895.

Posner, J., Russell, J. A., & Peterson, B. S. (2005). The circumplex model of affect: An integrative approach to affective neuroscience, cognitive development, and psychopathology. *Development and Psychopathology, 17,* 715–734.

Richardson, M. P., Strange, B. A., & Dolan, R. J. (2004). Encoding of emotional memories depends on amygdala and hippocampus and their interactions. *Nature Neuroscience, 7,* 278–285.

Rogan, M. T., Leon, K. S., Perez, D. L., & Kandel, E. R. (2005). Distinct neural signatures for safety and danger in the amygdala and striatum of the mouse. *Neuron, 46,* 309–320.

Rolls, E. T. (1999). *The brain and emotion.* Oxford, England: Oxford University Press.

Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39,* 1161–1178.

Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review, 110,* 145–172.

Russell, J. A., Weiss, A., & Mendelsohn, G. A. (1989). Affect grid: A single-item scale of pleasure and arousal. *Journal of Personality and Social Psychology, 57,* 493–502.

Sarter, M., Givens, B., & Bruno, J. P. (2001). The cognitive neuroscience of sustained attention: Where top-down meets bottom-up. *Brain Research: Brain Research Reviews, 35,* 146–160.

Scherer, K. (2001). *Psychological theories of emotion and neuropsychological research* (Vol. 5, 2nd ed.). New York: Elsevier.

Schlosberg, H. (1954). Three dimensions of emotion. *Psychological Review, 61,* 81–88.

Schmahmann, J. D. (2004). Disorders of the cerebellum: Ataxia, dysmetria of thought, and the cerebellar cognitive affective syndrome. *Journal of Neuropsychiatry and Clinical Neurosciences, 16,* 367–378.

Schmahmann, J. D., & Sherman, J. C. (1998). The cerebellar cognitive affective syndrome. *Brain, 121,* 561–579.

Segal, Z. V., Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry, 63,* 749–755.

Small, D. M., Gregory, M. D., Mak, Y. E., Gitelman, D., Mesulam, M. M., & Parrish, T. (2003). Dissociation of neural representation of intensity and affective valuation in human gustation. *Neuron, 39,* 701–711.

Small, D. M., Zatorre, R. J., Dagher, A., Evans, A. C., & Jones-Gotman, M. (2001). Changes in brain activity related to eating chocolate: From pleasure to aversion. *Brain, 124,* 1720–1733.

Smith, S. D., & Bulman-Fleming, M. B. (2005). An examination of the right-hemisphere hypothesis of the lateralization of emotion. *Brain and Cognition, 57,* 210–213.

Spitzer, R. J., Williams, J. B. W., & Gibbon, M. (1995). *Structured clinical interview for DSM–IV (SCID*). New York: Biometrics Research.

Squire, L. R., Wixted, J. T., & Clark, R. E. (2007). Recognition memory and the medial temporal lobe: A new perspective. *Nature Reviews Neuroscience, 8,* 872–883.

Steidl, S., Mohi-uddin, S., & Anderson, A. K. (2006). Effects of emotional arousal on multiple memory systems: Evidence from declarative and procedural learning. *Learning & Memory, 13,* 650–658.

Supple, W. F., Jr., Leaton, R. N., & Fanselow, M. S. (1987). Effects of cerebellar vermal lesions on species-specific fear responses, neophobia, and taste-aversion learning in rats. *Physiology and Behavior, 39,* 579–586.

Tian, L., Jiang, T., Liu, Y., Yu, C., Wang, K., Zhou, Y., . . . Li, K. (2007). The relationship within and between the extrinsic and intrinsic systems indicated by resting state correlational patterns of sensory cortices. *Neuroimage, 36,* 684–690.

Tomkins, S. S. (1962). *Affect, imagery, consciousness.* New York: Springer.

Velten, E., Jr. (1968). A laboratory task for induction of mood states. *Behaviour Research and Therapy, 6,* 473–482.

Vertes, R. P., Hoover, W. B., Szigeti-Buck, K., & Leranth, C. (2007). Nucleus reuniens of the midline thalamus: Link between the medial prefrontal cortex and the hippocampus. *Brain Research Bulletin, 71,* 601–609.

Vogt, B. A. (2005). Pain and emotion interactions in subregions of the cingulate gyrus. *Nature Reviews Neuroscience, 6,* 533–544.

Vogt, B. A., Berger, G. R., & Derbyshire, S. W. (2003). Structural and functional dichotomy of human midcingulate cortex. *European Journal of Neuroscience, 18,* 3134–3144.

Vogt, B. A., & Pandya, D. N. (1987). Cingulate cortex of the rhesus monkey: II. Cortical afferents. *Journal of Comparative Neurology, 262,* 271–289.

Watson, D. T., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin, 98,* 219–235.

Windischberger, C., Cunnington, R., Lamm, C., Lanzenberger, R., Langenberger, H., Deecke, L., & Moser, E. (2008). Time-resolved analysis of fMRI signal changes using brain activation movies. *Journal of Neuroscience Methods, 169,* 222–230.

Winston, J. S., Gottfried, J. A., Kilner, J. M., & Dolan, R. J. (2005). Integrated neural representations of odor intensity and affective valence in human amygdala. *Journal of Neuroscience, 25,* 8903–8907.

Wundt, W. M., & Wirth, W. (1905). *Grundzéuge der physiologischen Psychologie* (5th ed.). Leipzig, Germany: Engelmann.

Zald, D. H. (2003). The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research: Brain Research Reviews, 41,* 88–123.

Zarahn, E., & Slifstein, M. (2001). A reference effect approach for power analysis in fMRI. *Neuroimage, 14,* 768–779.

Received November 13, 2008
Revision received October 6, 2009
Accepted October 6, 2009 ■