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The amygdala and the experience of affect

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The current study examined the hypothesis that amygdala activation serves as a neural precondition for negative affective experience. Participants' affective experience was measured by asking them to report on their momentary experiences several times a day over the course of a month using an electronic experience-sampling procedure. One year later, participants viewed backwardly masked depictions of fear while functional magnetic resonance imaging was used to measure their amygdala and fusiform gyrus activation. Negative affect, as measured during the experience-sampling procedure 1-year prior, was positively correlated with amygdala activation in response to these brief presentations of fear depictions. Furthermore, descriptive analyses indicated that fusiform gyrus activation and negative affective experience in the scanner were associated for participants reporting increased nervousness during the imaging procedure. The results are consistent with the interpretation that the amygdala contributes to negative affective experience by increasing perceptual sensitivity for negative stimuli.

Affect is an elemental aspect of conscious life. People are always in some state that can be described on a continuum of pleasant to unpleasant, high arousal to low arousal, and this state informs people about their relation to the world around them (for a review, see Russell and Barrett, 1999). Affective states help to establish whether an object is a threat or a reward and serve as a later signal of the object's value (cf. Nauta, 1971). Affect serves to select the contents of consciousness via bottom-up forms of attention (cf. Duncan and Barrett, in press; Edelman and Tononi, 2000). The conscious experience of affect (i.e. affective feelings) is also an elemental, core feature of emotion experience (for a review, see Barrett, 2006a, b). The extent or intensity of pleasant and unpleasant affective states is correlated with peripheral nervous system activation (Cacioppo *et al.*, 1997; Bradley and Lang, 2000; Cacioppo *et al.*, 2000), facial muscle movements (Cacioppo *et al.*, 1997, 2000; Messinger, 2002), vocal cues (Bachorowski, 1999), expressive behavior (Cacioppo and Gardner, 1999), and neural activations (Wager *et al.*, 2003). One goal of affective neuroscience is to understand how the brain entails affective feelings. The current study begins to address that question by investigating the association between affective experience and activation in the amygdala.

There is currently mixed evidence regarding the importance of the amygdala to affective experience. Evidence from lesion patients suggests that the amygdala is not necessary for the experience of affect (Anderson and Phelps, 2001, 2002). Yet, the amygdala often shows robust activation in imaging studies of emotion experience. Of the 102 imaging studies that have imaged experiences of emotion or affect (i.e. participants were either asked to rate their experience in the scanner, undergo a mood induction or were shown evocative scenes or images), 42 reported significant amygdala activation over baseline.[1] Furthermore, participants with various mood disorders involving enhanced negative affect also show significantly higher amygdala activation (Drevets *et al.*, 1992; Breiter *et al.*, 1996; Rauch *et al.*, 1996; Abercrombie *et al.*, 1998; Birmbauer *et al.*, 1998; Rauch *et al.*, 2000; Sheline *et al.*, 2001; Siegle, 2002; Etkin *et al.*, 2004; Shin and Wright, 2005). Taken together, these findings can be reconciled by hypothesizing that amygdala activation might serve to cause affective experience via its influence on perception and memory, but it does not directly entail or instantiate experience itself. Our working hypothesis is that amygdala activation does not itself produce affective experience, but may set the neural preconditions (i.e. enhanced perceptual sensitivity for negative objects) for negative feelings to arise by influencing how sensory information from evocative stimuli is processed in the brain. Specifically, negative affective experiences are associated with sensitivity to negative stimuli, and this sensitivity appears to be instantiated, in part, by increases in amygdala activity.

Negative affect and perceptual sensitivity

Converging evidence from research on both clinical and normal samples suggests that people who routinely experience negative affect show a perceptual sensitivity to negative

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1 The 102 neuroimaging studies are from two meta-analyses of neuroimaging studies involving affect and emotion (Phan *et al.*, 2002; Wager et al., 2003) as well as MEDLINE, Psychinfo and BrainMap literature searches of peer-reviewed PET and fMRI studies of affect and emotion from January 2001 to December 2005.

stimuli. First, individuals with anxiety disorders, as well as healthy individuals who report high trait-anxiety, have difficulty disengaging their attention from negative features of the environment, and are biased to shift their attention to spatial locations where negative events have previously occurred. Numerous experimental paradigms have been used to demonstrate this effect, including dot-probe tasks (MacLeod *et al.*, 1986; Broadbent and Broadbent, 1988; Mogg *et al.*, 1992; Mogg *et al.*, 1995; Mathews *et al.*, 1996; Bradley *et al.*, 1997; Broomfield and Turpin, 2005), emotional-Stroop tasks (MacLeod and Hagan, 1992; MacLeod and Rutherford, 1992; Mogg *et al.*, 1993; Mogg *et al.*, 1995; Myers and McKenna, 1996), and dichotic-listening tasks (Nielsen and Sarason, 1981; Foa and McNally, 1986; Mathews and MacLeod, 1986). The failure to disengage attention from negative objects, along with the bias to shift attention towards the location of negative objects, indicates that top-down attentional mechanisms are disrupted in clinical disorders associated with negative affect, and suggests that these attentional propensities may be active whenever a person experiences strong or persistent negative affect. The result is that anxious and/or depressed individuals are more sensitive to negatively valenced stimuli.

Second, recent evidence suggests that normal variation in affective experience is linked with a perceptual sensitivity to affective information in the environment. In a study by Barrett and Niedenthal (2004), individuals participated in a computerized experience sampling study, where they rated their online affective experience several times a day over the course of a month. Two weeks after the conclusion of the experience sampling procedure, participants completed a morph movies task. This involved viewing a series of faces displaying a neutral expression that participants could gradually change to depict either a happy, angry or sad expression over the course of a 100-frame computerized movie. Participants were required to detect the moment of expression onset for each movie, allowing a precise estimate of their sensitivity to perceptual information that was either pleasant or unpleasant. Individuals who focused on feelings of pleasure and displeasure in their momentary emotional experiences (as measured during the computerized experience sampling procedure) perceived the onset of angry and sad faces much earlier than did those individuals who were less focused on valence, demonstrating an enhanced perceptual sensitivity to affective information in the environment.

Finally, the amygdala's connectivity with other areas of the brain suggests it may be a nexus for modulating both perceptual sensitivity and affective experience. The amygdala has strong, excitatory afferent projections to all portions of the ventral visual stream (Amaral and Price, 1984; Amaral *et al.*, 2003; Freese and Amaral, 2005), suggesting that it modulates sensory processing, especially when an organism must learn more about a stimulus so as to better determine its predictive value for well-being and survival (Whalen, 1998; Davis and Whalen, 2001; Kim *et al.*, 2003). Furthermore, the amygdala (along with the OFC and ventromedial prefrontal cortex) contributes to a widely distributed neural circuit that integrates external sensory information about a stimulus and with internal sensory information regarding changes in an organism's somatovisceral state (Ongur and Price, 2000; Ghashghaei and Barbas, 2002; Barbas *et al.*, 2003; Ongur *et al.*, 2003; Kringelbach and Rolls, 2004). These internal somatovisceral changes contribute to a person's affective state. Given this connectivity, activity in the amygdala should be strongly correlated with both affective experience and neural activity in the ventral visual stream.

Overview of the present study

The purpose of this study was to examine whether amygdala activation was correlated to negative affective experience. Specifically, we examined whether individuals who reported intense and consistent momentary feelings of negative affect (in a computerized experience-sampling study) showed enhanced amygdala activation to briefly presented depictions of fear. Furthermore, we also explored the relationship between affective experience and fusiform gyrus (FG) activation in response to visual stimuli.

This study incorporated several design features that make a significant contribution to the existing literature on amygdala activity and function. First, negative affective experience was measured as the mean rating of momentary experiences over a 28-day experience-sampling period. Our focus on momentary experience stands in contrast to many studies that have examined the relation between memory-based ratings of affective experience and amygdala response. For example, two previous studies by Anderson and Phelps (2002) showed that individuals with unilateral and bilateral amygdala damage reported the same affective experience as healthy controls. In their first study, participants with amygdala damage remembered experiencing the same levels of Positive and Negative Activation (PA and NA) over the prior year as did healthy controls. In their second study, participants completed end of the day reports of PA and NA and again, those with amygdala damaged reported no differences when compared to healthy controls. Reports such as these, which rely on retrospective judgments, are heavily infused with people's beliefs about their emotional lives and may not correspond to experience as it is actually felt (Barrett, 1997; Robinson and Clore, 2002). By using experience-sampling procedures, the current study was able to more accurately assess the intensity of momentary affective experience. Second, ours was a prospective study that occurred over a one-year period, because momentary ratings of negative affective experience were captured a year before amygdala and FG responses to viewing backwardly masked faces depicting fear were measured. As a result, our findings would speak to the stability of a link between amygdala activation and affective

experience, and build a functional consequence onto existing evidence that amygdala response to fear faces is stable across an 8 week period (Johnstone *et al.*, 2005). Finally, to test whether the amygdala-FG circuit creates a perceptual context that allows for negative affective experience, we had subjects rate their experience in the scanner during the face presentation task.

We predicted that participants who reported greater levels of negative affective experience during the month-long experiencing sampling procedure would have greater amygdala activation to backwardly masked fearful faces than those reporting lower levels of negative affect. We also examined whether amygdala activation would be correlated with FG activation, and explored the possibility that this correlation would be higher for those who reported greater negative affect during the face presentation task. Since the amygdala has been shown to habituate to repeated presentations of complex stimuli, such as faces (Wright *et al.*, 2001; Fischer *et al.*, 2003), we expected that these correlations would surface during the first blocks of experimental stimuli.

METHOD

Participants

Participants were 13 Boston College undergraduates (six males) who were paid $120 for their participation. These 13 individuals were from a larger sample of 86 participants in an experience sampling study (Barrett, 2004, Study 3).[2] Participants completed 28 days of recording their experiences of emotion (although a few were sampled for more days). The number of usable measurement moments ranged substantially from 107 to 368, with a mean of 218.13 and a s.d. of 57.38. One calendar year after participants completed the experience-sampling paradigm, those individuals ($n = 13$) who were still available participated in the current neuroimaging study. The mean levels of affective experience for the current sample were similar to those in the larger study (see Table 1; all *P*-values >0.05 using independent samples *t*-tests).

Table 1 Mean affective levels for the current sample compared to those from the larger experience sampling study

	Participants in current study ($n = 13$)		Experience sampling study[a] ($n = 73$)	
	M	s.d.	*M*	s.d.
Negative high arousal	2.06	0.66	2.21	0.73
Negative moderate arousal	2.01	0.67	2.32	0.73
Negative low arousal	2.99	0.75	3.35	0.64
Positive high arousal	3.64	0.73	3.38	0.79
Positive moderate arousal	4.18	0.77	3.98	0.78
Positive low arousal	4.15	0.81	3.91	0.70
Neutral high arousal	2.44	0.94	2.51	0.83
Neutral low arousal	3.19	0.88	3.65	0.84

[a]This group includes participants in the experience sampling study who did not later participate in the current neuroimaging study.

Procedure

Experience sampling procedure. During the experience sampling protocol, participants visited the lab five times. During the first lab session, participants were assigned a palm-top computer (Hewlett Packard 360 LX), and received instructions regarding the experience-sampling portion of the study. The palm-tops ran on custom software (Experience Sampling Program or ESP; Barrett and Barrett, 1999). Affect terms were presented in a random order at each trial. Participants made their ratings on a 7-point Likert scale (0 = not at all, 3 = a moderate amount, 6 = a great deal) measured by pressing numbers on the keyboard of the palm-top computer. Participants were told that they would be beeped randomly 10 times per day for a 28 day period and asked about their momentary affective experience using 29 emotion-related terms (potentially resulting in 280 affect measurement trials per participant, each of which contained ratings for 29 terms). Participants were instructed to respond as quickly as possible without compromising their accuracy. If they did not respond to the first prompt, they would be beeped again 2 min later. If they failed to respond to that prompt as well, then the trial was recorded as missing data. Participants were run through a practice trial of ESP and given a written set of instructions about the experience-sampling procedure before leaving the lab. Both ratings of experience, and latencies to make those ratings, were recorded. We combined items to examine the mean level of each type of experience described by the affective circumplex (Barrett and Russell, 1999). All combinations of valence and arousal were sampled (see Figure 1). Affective levels were quantified by computing the mean level of high arousal, neutral valence ('aroused,' 'surprised'), high arousal, positive valence ('active,' 'alert,' 'eager,' 'enthusiastic,' 'excited,' 'interested,' 'peppy,' 'proud'), moderate arousal, positive valence ('content,' 'happy,' 'satisfied'), low arousal, positive valence ('calm,' 'relieved,' 'relaxed'), low arousal, neutral valence ('sleepy'), low arousal, negative valence ('bored,' 'tired,' 'sluggish'), moderate arousal, negative valence ('disappointed,' 'guilty,' 'sad'), and high arousal, negative valence ('ashamed,' 'afraid,' 'angry,' 'disgusted,' 'nervous') reported across the sampling period (see Barrett and Russell, 1998).

Imaging procedure. One calendar year after participants completed the experience-sampling paradigm, those individuals who were still available for testing completed a masked emotional faces paradigm, based on the paradigms of Rauch *et al.* (2000) and Whalen *et al.* (1998). Face stimuli

2 One participant in the current study had missing behavioral, semantic-similarity data in Barrett (2004; study 3). This participant was used to compare mean affect during experience tracking between the current sample, and other participants from Barrett (2004; study 3) although they were not included in the original study.

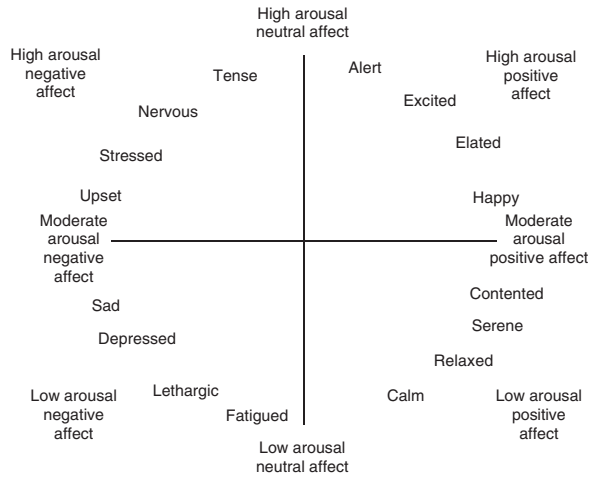

Fig. 1 The affective circumplex.

consisted of fearful and neutral depictions by eight individuals. Fearful (F) and neutral (N) facial depictions (Ekman and Friesen, 1976) were presented in alternating blocks, with interspersed rating blocks (R) where subjects reported on their affective experience using nine emotion adjectives (active, angry, calm, excited, happy, nervous, quiet, sad, sluggish). Participants saw four counterbalanced runs (two each of +RNRFRNRFRNRF+ and +RFRNRFRNRFRN+). During the F and N blocks, fearful and neutral faces were backwardly masked by neutral faces of different identities. In each block, 48 trials (each lasting 24 s) consisted of a target face (neutral in the N blocks or fearful in the F blocks) presented for 16 ms, followed by a neutral mask presented for 112 ms. During the rating blocks, participants had 4 s to rate their affective experience for each of the nine adjectives (a total of 36 s). Imaging data were available for all 13 participants, but rating data were missing in 4 due to an implementation error. Trials were separated by a 372 s inter-trial interval (each trial lasted 500 ms). The face stimuli (in PICT format) were displayed using standardized software (MacStim 2.5.9) and a Sharp XG-2000V color LCD projector (Osaka, Japan). Stimulus presentation times were matched with the refresh rate of the projector to ensure that the experimental stimuli were presented for the appropriate amount of time.

Image Acquisition. A Sonata 1.5 Tesla whole body high-speed imaging device equipped for echo planar imaging (EPI) (Siemens Medical Systems, Iselin NJ) was used with a 3-axis gradient head coil. Head movement was restricted using expandable foam cushions. After an automated scout image was acquired and localized shimming procedures were performed to optimize field homogeneity, high-resolution 3D MPRAGE sequences (TR/TE/flip angle = 7.25 ms/3 ms/7°) with an in-plane resolution of 1.3 mm, and 1 mm slice thickness, were collected for spatial normalization and for positioning the slice prescription of the subsequent sequences. Then a T1-weighted (TR/TE/flip angle = 8 s/39 ms/90°) and a T2-weighted (TR/TE/flip angle = 10 s/48 ms/120°) sequence were used to gather images to assist in registration of the functional data to the high-resolution anatomical scan. Functional MRI images (blood oxygenation level dependent or BOLD) (Kwong *et al.*, 1992) were acquired using a gradient echo T2*-weighted sequence (TR/TE/flip angle = 2.4 s/40 ms/90°). Prior to each scan, four time points were acquired and discarded to allow longitudinal magnetization to reach equilibrium. The T1, T2, and gradient-echo functional images were collected in the same plane (24 coronal slices angled perpendicular to the ac-pc line) with the same slice thickness (7 mm, skip 1 mm; voxel size 3.125 × 3.125 × 8 mm), excitation order (interleaved) and phase encoding (foot-to-head). These parameters were used for the functional images as earlier work suggested that they help to minimize susceptibility in medial temporal lobe regions (Wright *et al.*, 2003a; Wright *et al.*, 2003b).

fMRI Data analyses

Data in each functional run was spatially smoothed (full width half maximum = 7 mm) using a 3D Gaussian filter (http://surfer.nmr.mgh.harvard.edu). Functional data were then normalized to correct for global signal intensity changes. Following signal intensity normalization, the functional runs were motion corrected using AFNI (http://afni.nimh.nih.gov/afni/index.shtml) (Cox, 1996; Cox, 1999). Processing of the functional data included polynomial drift correction that entailed two nuisance regressors spanning the space of a 2nd order polynomial to account for low-frequency drift, and removal of temporal autocorrelation by whitening based on a single autocorrelation function estimated across all brain voxels (Burock and Dale, 2000). The normalized, motion-corrected, whitened functional images were then aligned to a 3D structural image created by motion correcting and averaging the high-resolution 3D sagittal images. As part of the alignment procedure, the raw functional data from each subject were visualized over the high-resolution 3D anatomical images from that individual to ensure that the BOLD signal in the amygdala, an *a priori* region of interest, was not obscured by susceptibility artifact. Individual subject functional data were subsequently spatially normalized using an optimal linear transformation method (Fischl *et al.*, 2002). After spatial normalization, registration of the spatially transformed anatomical and the original individual subject 3D anatomical images were manually verified. For consistency across studies, we displayed group statistical maps on a group averaged Talairach brain, and present Talairach coordinates that are based on registration of the images from the optimal linear transformation with the Talairach atlas (Talairach and Tournoux, 1988).

Functional MRI data were analyzed using the standard processing stream of the Martinos Center for Biomedical Imaging (software and documentation is

available at http://www.nmr.mgh.harvard.edu/P41/resources SoftDescription.html). Functional images were averaged across subjects according to condition for each block in each run (i.e. fixation, neutral, fearful, rating). Group statistical maps were then computed using a random-effects model. A functionally based, region of interest (ROI) analysis was used to investigate the effects of affective reactivity and amygdala and FG responses to masked threat-related stimuli. The ROIs were defined by the contrast of all faces (neutral and fearful) *vs* the fixation cross across all subjects to assess the role of affective reactivity in a manner that was unbiased with respect to between group differences.

ROIs (i.e. clusters of significant voxels) were chosen based on statistical significance thresholds for our *a priori* regions of interest ($P \leq 0.004$ for the amygdala, and $P \leq 0.0005$ for FG). This represents a Bonnferoni type correction for multiple comparisons based on the approximate total volume (L + R) of the amygdala ($3.5\,cm^3 \approx 13$ resolution elements) (Brierley *et al.*, 2002) and FG ($26.4\ cm^3 \approx 96$ resolution elements) (Kennedy *et al.*, 1998), and the degree of smoothing applied (yielding a resolution element of $2744\,mm^3$). For the faces *vs* fixation contrast, regions in the bilateral amygdala (Right: peak $P = 0.004$; Talairach Coordinates $x = 28$, $y = -1$, $z = -19$; Left: $P =$ peak, $P < 0.0003$; Talairach Coordinates $x = 30$, $y = -8$, $z = -18$) and FG (Right: peak $P < 0.0001$; Talairach Coordinates $x = 34$, $y = -50$, $z = -13$; Left: peak $P = 0.0001$; Talairach Coordinates $x = -34$, $y = -45$, $z = -16$) met the significance criteria. Labels were made based on the coordinates of contiguous functional voxels in each cluster in the group statistical map that had task correlated activity at a level of $P < 0.01$. Nine voxels in the right amygdala, six voxels in the left amygdala and 32 voxels in each the right and left FG met these criteria (see Figure 2). The functionally defined labels from these regions were then used to extract BOLD signal intensity data from the amygdala and FG of each of the individual subjects in the study. These data were used to calculate % BOLD signal change for each condition *vs* fixation for each subject, and this information was used to examine for correlations with our measures of affective experience.

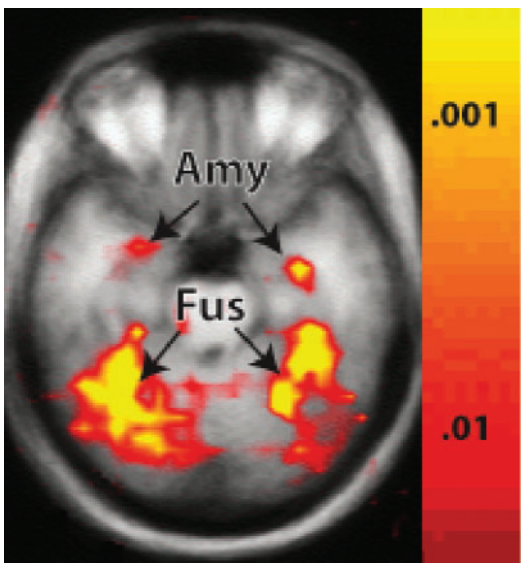

Fig. 2 Neural activations within the amygdala and FG for the first block of fearful faces *vs* fixation cross. These regions were chosen as our *a priori* regions of interest ($P \leq 0.004$ for the amygdala, and $P \leq 0.0005$ for FG) and were used in further correlational analyses.

To assess how affective experience during the experience-sampling procedure related to activity outside of the amygdala and FG, we performed *post-hoc* whole-brain cortical surface analyses, correlating measures of affect experience measures with fMRI activations across the whole cerebral cortex (Fischl and Dale, 2000; Wright *et al.*, 2006). We focused on negative affect measures in these *post-hoc* analyses as this is where significant results were found in our *a priori* analyses. For the whole-brain analyses, the averaged high-resolution 3D anatomical images were used to construct inflated (2D) models of individual cortical surfaces using an automated procedure (Fischl and Dale, 2000; Wright *et al.*, 2006). Individual subject functional data for the relevant contrasts of interest were resampled on the cortical surface. These data were then used to compute a group cortical surface average displaying the statistical results of a general linear model assessing negative affect effects on fMRI activation. The statistical threshold for these analyses was $P < 0.0001$ reflecting an approximate correction for the multiple comparisons across the cortical surface without *a priori* hypotheses.

RESULTS

Amygdala activity and affective experience

The correlations between mean levels of affective experience and amygdala activity are presented in Table 2. As predicted, individuals who reported greater experiences of negative affect (at all levels of arousal) across 28 days of experience sampling demonstrated significantly greater amygdala activation during the first block of briefly presented, masked fearful faces compared to those who reported lower levels of

Table 2 Correlations between dispositional negative affect and amygdala activation during the first block of fearful faces

	Right amygdala		Left amygdala	
	r	*P*	*P*	*P*
High arousal, negative	0.79	0.001	0.51	0.08
Moderate arousal, negative	0.67	0.02	0.52	0.07
Low arousal, negative	0.81	0.001	0.39	0.18
High arousal, positive	−0.13	0.67	0.04	0.90
Moderate arousal, positive	0.04	0.90	0.04	0.90
Low arousal, positive	0.09	0.76	0.12	0.69
High arousal, neutral valence	−0.06	0.86	0.10	0.74
Low arousal, neutral valence	0.61	0.03	0.20	0.51

negative affective experience. Individuals who reported greater mean levels of high, moderate and low arousal, negative affect showed a significant signal increase in the right amygdala when viewing the first block of masked fear faces within each run, compared to those who reported lower levels of negative experience (Figure 3). The correlations between experiences of negative affect and amygdala activation were not statistically significant for subsequent blocks, as predicted. There were no statistically significant correlations between the experience of positive affect or highly activated, neutral valence experiences during experience-sampling and amygdala activity, but reports of deactivated, neutral valence experiences ('sleepy') were associated with greater right amygdala response.

Similar results were observed when we correlated affective experience with the difference between amygdala activation during the first fear block and amygdala activation during the first neutral block. Individuals who reported greater high and moderate arousal negative affect across the experience sampling period also showed enhanced right amygdala activations in response to fear relative to neutral faces during the first blocks, $r = 0.52$, $P < 0.07$ and $r = 0.63$, $P < 0.05$. Similar patterns were observed for the left amygdala, but these correlations did not reach conventional levels of statistical significance (all *P*-values >0.05).

Amygdala and fusiform activity

Amygdala and FG activation were correlated during the first blocks of fearful faces. Specifically, higher activation in the left amygdala was associated with higher activation in the left FG, $r = 0.68$, $P < 0.01$. However, right amygdala activation was not significantly correlated with right or left FG activation during the first blocks of fearful faces (see Table 3).

Affective experience during imaging

Self-report ratings of affective experience during the imaging experiment were only available for 9 of the 13 participants. With data on only nine participants, it was not possible to inferentially test whether those who experienced greater negative affect in response to the briefly presented fear faces showed a stronger association between amygdala and FG activation that would be indicative of visual awareness of the faces. Descriptive analyses were consistent with this hypothesis, however. Three participants reported an increase in nervousness to the first fear block (*M* increase in nervousness = 0.58, s.d. = 0.38), compared with six participants who did not ($M = -0.13$, s.d. = 0.21). This is in the face of reasonable stability in reports of nervousness across the period of a year (ratings of nervousness after the first fear block and mean high arousal, negative affect during experience sampling a year prior were correlated 0.64, $P < 0.06$). The amygdala-FG correlations for these two groups, presented in Table 4 for descriptive purposes, clearly illustrate that individuals who were experiencing enhanced negative affect in response to backwardly-masked fear faces had very strong correlations between amygdala and FG activation, whereas those who experienced no change in nervousness showed considerably weaker (or even negative) correlations.

Moreover, task-related levels of affective experience appeared to moderate the relationship between negative affect reported during experience sampling and FG activation. Again, these analyses are presented for their descriptive (rather than inferential) value. For the entire sample, there were no statistically significant correlations between dispositional negative affect and FG activity. For the three 3 individuals who reported increases in nervousness during the first fear block, however, the correlation between right FG

Table 3 Correlations between amygdala and fusiform gyrus activation during the first block of fearful faces

	Right fusiform		Left fusiform	
	r	*P*	*r*	*P*
Right amygdala	0.43	0.14	0.34	0.26
Left amygdala	0.24	0.43	0.68	0.01

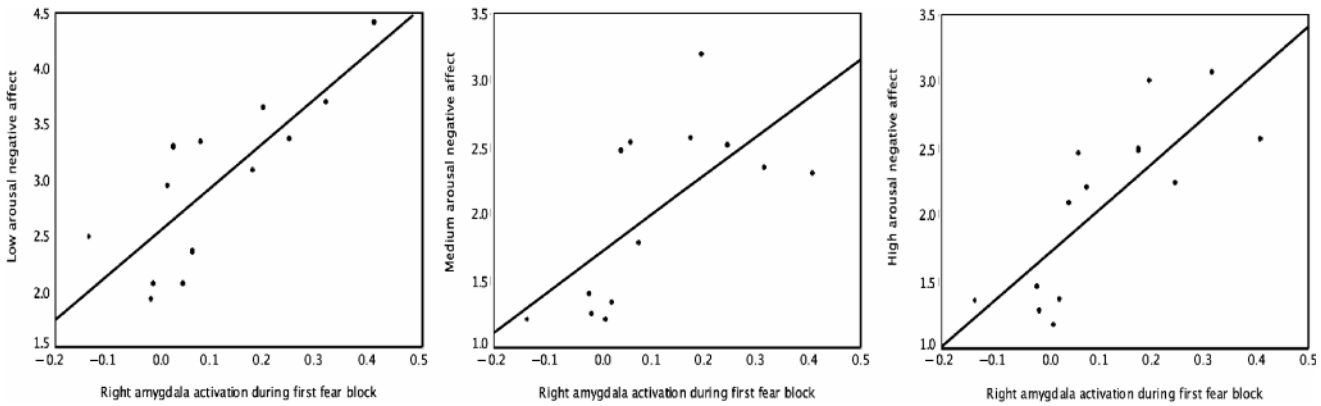

Fig. 3 Scatterplots showing correlations between right amygdala activation and reports of negative affect recorded during experience sampling.

activation and mean levels of high arousal negative affect and moderate arousal negative affect were strong, $r = 0.93$, and $r = 0.96$, respectively.

Post-hoc whole-brain analyses

For the first fear block *vs* fixation contrast, individuals who reported greater high arousal, negative affect during experience sampling demonstrated a greater increase in activation of the left temporal pole (Talairach coordinates: $x = -28$, $y = 8$, $z = -27$; $P < 0.00003$; $r = 0.902$) (see Figures 4A and C), as well as greater decreases in activity in the right precentral gryus (Talairach coordinates: $x = 35$ $y = -9$, $z = 56$; $P < 0.00002$; $r = -0.907$), right precuneus (Talairach coordinates: $x = 8$, $y = -64$, $z = 41$; $P < 0.00006$; $r = -0.884$; Figure 4D) and right calcarine cortex (Talairach coordinates: $x = 18$, $y = -91$, $z = 5$; $P < 0.0001$; $r = -0.875$; Figure 4E). Similar results were obtained for links between reports of moderate arousal, negative affect and activity in the precuneus and temporal pole and between reports of low arousal negative affect and activation in the left paracentral gyrus (Talairach coordinates; $x = -5$, $y = -33$, $z = 52$; $P < 0.00003$; $r = -903$), the left supramarginal gryus (Talairach coordinates; $x = -61$, $y = -27$, $z = 26$; $P < 0.00003$; $r = -0.902$) and the left FG (Talairach coordinates; -28, -52, -8); $P < 0.0001$ $r = -0.880$).

Table 4 Correlations between dispositional negative affect and FG activation for individuals who experienced an increase or no increase in nervousness during the fMRI task

	Right fusiform		Left fusiform	
	r	*P*	*r*	*P*
Increased nervousness during task ($n = 3$)				
Negative high arousal	0.93	0.23	0.76	0.45
Negative moderate arousal	0.96	0.17	0.56	0.62
Negative low arousal	0.99	0.05	0.71	0.50
No increased nervousness during task ($n = 6$)				
Negative high arousal	−0.19	0.72	0.59	0.22
Negative moderate arousal	−0.28	0.60	0.60	0.21
Negative low arousal	0.23	0.66	−0.24	0.64

DISCUSSION

Despite the modest sample size, the present study provides initial support for the hypothesis that amygdala activation supports but does not itself instantiate, affective experience. We demonstrated that, compared to those who reported lower levels of negative affect in their everyday life over a month long period, those who reported high levels showed

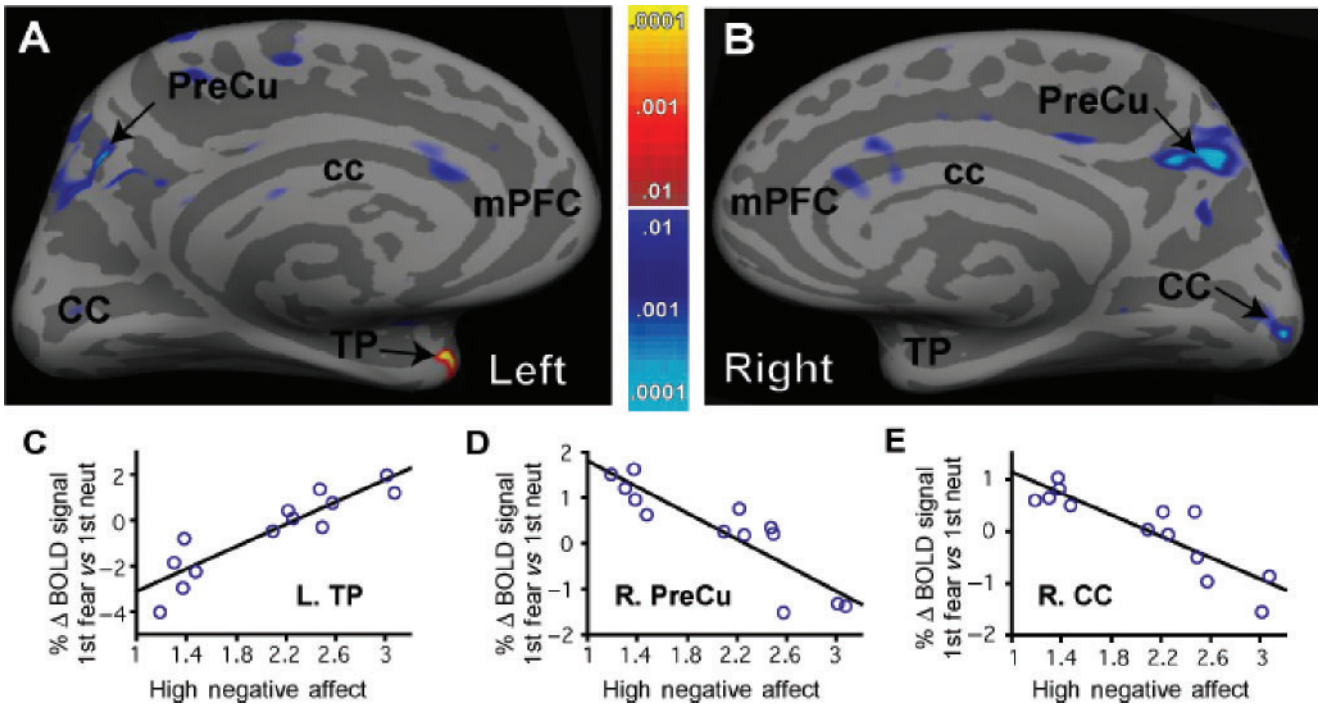

Fig. 4 Correlations between cortical activation to initial fear *vs* neutral blocks and mean levels of high arousal, negative affect reported during the experience-sampling procedure. (**A**) colorized statistical map superimposed upon an inflated group average cortical surface. The medial aspect the right hemisphere is shown. Significant positive correlations were found between high arousal negative affect and activations in right temporal polar cortex (TP). Trend negative correlations were present in the right precuneus (PreCu). (**B**) Colorized statistical map superimposed upon an inflated group average cortical surface. The medial aspect of the left hemisphere is shown. Significant negative correlations were found between high arousal negative affect and activations the precuneus (PreCu) and calcarine cortex (CC). Dark gray regions are sulci, light gray are gyri. Colorized scale bars show the *P*-value for positive (red-yellow) and negative (blue) correlations. The corpus callosum (cc) and medial prefrontal cortex (mPFC) are indicated. (**C**) Scatter plot and regression line demonstrating a significant positive correlation between the TP. These values were extracted from the peak surface point of the TP locus shown in (**A**). (**D**) Scatter plot and regression line from the peak of the PreCu locus in (**A**). (**E**) Scatter plot and regression line from peak of the CC locus in (**A**).

greater amygdala activation in response to briefly presented negative stimuli 1 year later. Furthermore, individuals with greater amygdala activation also showed greater FG activation. These findings are consistent with the hypothesis that the amygdala, possibility with the FG, helps to create the perceptual context for negative affective experiences. In the current study, faces were presented for only 16 ms, making it unlikely that most participants were visually aware of the face stimuli (although a few individuals have been shown to objectively detect backwardly-masked, 16 ms presentations of fearful faces above chance levels; Pessoa *et al.*, 2005). Nonetheless, individuals with greater FG activation may be further along the path to consciously seeing masked faces depicting fear than those with less FG activation.

Post-hoc, whole-brain analyses revealed that participants who experienced greater high arousal negative affect also showed enhanced activation in the temporal pole in response to faces depicting fear 1 year later. There are two possible explanations for this result. First, previous neuroimaging studies have demonstrated greater activation in this region when people observe familiar faces (Nakamura *et al.*, 2000; Griffith *et al.*, 2006), and lesions of the left temporal pole result in deficits in providing proper names for individuals (Papagno and Capitani, 1998; Glosser *et al.*, 2003), suggesting that participants in the current study who experienced greater dispositional negative affect may have interpreted the facial stimuli as having greater personal relevance. Second, temporal pole activation has been observed in fMRI contrasts where participants focus on the affective states of others *vs* their own (Ruby and Decety, 2004). Participants in the current study who had greater dispositional negative affect, then, may have also allocated greater resources towards interpreting the affective state of the fearful face stimuli than participants with less dispositional negative affect, even though they very likely did not have subjective awareness of the face stimuli.

Post-hoc, whole-brain analyses also showed that participants who experienced greater levels of high arousal negative affect showed decreased activity in the right precuneus and calcarine cortex in response to faces depicting fear 1 year later. Such task-related deactivations may result from participant's cognitive activity during baseline (Gusnard and Raichle, 2001; Newman *et al.*, 2001). A number of fMRI studies have reported increased precuneus activity when participants engage in mental imagery (for a metanalytic review see Cavanna and Trimble, 2006) and precuneus activity is commonly observed during baseline resting tasks (for meta-analytic review see Mazoyer *et al.*, 2001). Decreased activity in precuneus may reflect engagement by external stimuli, such as stimuli from the scanning environment. These findings suggest that individuals who reported greater negative affect 1 year prior to imaging may have been more engaged by the backwardly masked fear faces than those with lower dispositional negative affect, again suggesting that they were more affected by the stimuli.

The current study has several limitations. First, only three individuals experienced an increase in negative affective experience during scanning. As a result, the correlations coefficients that we report for the association between amygdala and FG activity in these individuals are offered as purely descriptive data, and should be interpreted with caution. Second, it is always possible that the presentation times for backwardly masked faces depicting fearful and neutral expressions actually exceeded 16 ms, because presenting visual stimuli very quickly using LCD-projectors is notoriously difficult. If the actual presentation times of backwardly masked fearful and neutral stimuli did exceed 16 ms, however, this would not have compromised the internal validity of our study because our goal was to measure amygdala activation, not to compare conscious *vs* unconscious perception of visual stimuli,. Backwardly masked depictions of fear were used only because they reliably elicit amygdala activity in scanning environments.

Future directions

Along with previous studies showing increased amygdala activation among clinically depressed and anxious individuals, the current study suggests that amygdala responses to negative stimuli may serve as a more pervasive vulnerability factor to develop affect-related disturbances. This speculation is consistent with recent research showing enhanced amygdala activity in healthy first-order relatives of clinically depressed individuals (Drevets *et al.*, 1992) and greater amygdala activation in healthy individuals with a specific polymorphism of the 5-HTT gene, which is associated with a risk for developing clinical depression and anxiety disorders (Hariri *et al.*, 2002; Hariri and Holmes, 2006). It is also consistent with findings that amygdala responses to facial depictions of fear are stable across an 8-week period (Johnstone *et al.*, 2005), and that depressed individuals with hyper-active amygdala responses to backwardly-masked fear faces demonstrated reduced amygdala activation in response to 8 weeks of treatment with the antidepressant sertraline, a selective serotonin reuptake inhibitor (SSRI), demonstrated reduced amygdala activation to masked fearful faces (Sheline *et al.*, 2001). Ours is the first study to show that high levels of negative affect are associated with increased amygdala activation in normal individuals one year after affective experience was recorded. These findings not only suggest that the association between negative affect and amygdala activation generalizes to a non-clinical sample, but that the association is quite stable over time.

Finally, we observed that amygdala activity was positively correlated with activity in the FG (this relation was higher for those who reported increases in negative affect during the face presentation task, but due to the small sample size, the results should be considered descriptive only and interpreted cautiously). Given the amygdala's role in modulating the FG during visual awareness of valenced stimuli (Pessoa and Padmala, 2005; Pessoa *et al.*, 2006), our findings

suggest the possibility that the amygdala-FG circuit forms a perceptual precondition that allow for unpleasant affective experiences. Objective awareness of valenced stimuli (i.e. greater perceptual sensitivity in signal detection terms, even when participants report no conscious awareness of the stimulus) is associated with increased amygdala activation to facial expressions depicting fear, whereas its absence is associated with no increase in amygdala activation over baseline levels (Pessoa *et al.*, 2006). Furthermore, increased amygdala activation co-occurs with increased activation in fusiform gyrus (FG; a portion of the brain involved in complex object recognition that is activated when objects reach visual awareness; Tong *et al.*, 1998), but only when people are objectively aware of the stimuli (i.e. faces) presented to them (Pessoa *et al.*, 2006). Although we did not find that an overall association between FG and affective experience (as measured by experience-sampling), we did observe an association between negative affect during experience-sampling and FG activity among the few participants who experienced an increase in nervousness over the course of the scanning session, suggesting that these participants might have had enhanced awareness of faces depicting fear. The results of this study are correlational, however, and further research is needed to fully understand how the amygdala FG circuit relates to affective experience.

Conflict of Interest

None declared.

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