

## Attachment-style differences in the ability to suppress negative thoughts: Exploring the neural correlates<sup>☆</sup>

Omri Gillath,<sup>a,\*</sup> Silvia A. Bunge,<sup>a</sup> Phillip R. Shaver,<sup>a</sup> Carter Wendelken,<sup>a</sup> and Mario Mikulincer<sup>b</sup>

<sup>a</sup>Department of Psychology, University of California, Davis 95616, USA

<sup>b</sup>Department of Psychology, Bar-Ilan University, Ramat Gan 52900, Israel

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Beginning in infancy, people can be characterized in terms of two dimensions of attachment insecurity: attachment anxiety (i.e., fear of rejection and abandonment) and attachment avoidance (distancing oneself from close others, shunning dependency; Bowlby, J., 1969/1982. *Attachment and loss: Vol. 1. Attachment*, 2nd ed., Basic Books, New York). The capacity for emotion regulation varies with attachment style, such that attachment-anxious individuals become highly emotional when threatened with social rejection or relationship loss, whereas avoidant individuals tend to distance themselves or disengage from emotional situations. In the present study, 20 women participated in an fMRI experiment in which they thought about—and were asked to stop thinking about—various relationship scenarios. When they thought about negative ones (conflict, breakup, death of partner), their level of attachment anxiety was positively correlated with activation in emotion-related areas of the brain (e.g., the anterior temporal pole, implicated in sadness) and inversely correlated with activation in a region associated with emotion regulation (orbitofrontal cortex). This suggests that anxious people react more strongly than non-anxious people to thoughts of loss while under-recruiting brain regions normally used to down-regulate negative emotions. Participants high on avoidance failed to show as much deactivation as less avoidant participants in two brain regions (subcallosal cingulate cortex; lateral prefrontal cortex). This suggests that the avoidant peoples' suppression was less complete or less efficient, in line with results from previous behavioral experiments. These are among the first findings to identify some of the neural processes underlying adult attachment orientations and emotion regulation.

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People differ in the ways they form emotional bonds and regulate emotions in close relationships, and the differences have been extensively studied using behavioral methods with people of various ages from birth through early adulthood. We still know relatively little, however, about the neural underpinnings of individual differences in what researchers call “attachment style” (e.g., Shaver and Clark, 1994). The present article reviews some of the behavioral, psychophysiological, and neuroscientific data related to attachment and then describes an initial attempt to use fMRI methods to examine the neural basis of emotion regulation as it relates to attachment style.

Attachment theory (Ainsworth et al., 1978; Bowlby, 1969/1982, 1973, 1980) concerns the formation of emotional bonds between people and the effects of a person's attachment history on emotion regulation and other aspects of personality. According to Bowlby (1969/1982), proximity-seeking behavior, beginning early in infancy, is regulated by an innate *attachment behavioral system*, the function of which is to obtain protection and care from another person, an *attachment figure*. The system becomes adapted to characteristics of key attachment figures over the life course, and the resulting *attachment style* (e.g., secure, anxious, or avoidant) becomes relatively stable and can be measured by self-report questionnaires and structured interviews (see Mikulincer and Shaver, 2003 for a review of behavioral studies of attachment processes). These styles of attachment then affect numerous features of adults' close relationships and emotional experiences.

Attachment styles can be assessed in terms of two dimensions of insecurity: attachment-related anxiety and attachment-related avoidance (e.g., Ainsworth et al., 1978, Fig. 10; Brennan et al., 1998; Fraley and Spieker, 2003). People are roughly normally distributed within the space formed by these two orthogonal dimensions. Since 1987, hundreds of studies have shown that a person's attachment style is a powerful predictor of various psychological and social-relational phenomena including self- and social schemas, self-regulation of stress and emotion, the quality of relations with romantic or marital partners, sexual motivation, and reactions to relationship breakups or losses (see Mikulincer and Shaver, 2003; Shaver and Clark, 1994 for reviews).

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\* Corresponding author. Fax: +1 530 752 2087.

E-mail address: ogillath@ucdavis.edu (O. Gillath).

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Secure individuals (those scoring relatively low on both the anxiety and avoidance dimensions) cope well with stress, either by seeking support from trusted attachment figures or by calling upon mental representations of support received in the past (Mikulincer and Shaver, 2004). Insecure individuals (those scoring relatively high on either the anxiety or the avoidance dimension, or both), who do not feel as confident about the availability and responsiveness of others, are forced to decide between two alternative coping strategies. Individuals labeled “anxious” by attachment researchers hyperactivate their “attachment behavioral system” (Bowlby, 1969/1982), whereas those labeled “avoidant” deactivate the system. Hyperactivation includes a high degree of emotionality and hypersensitivity to signs of acceptance or rejection. Deactivation includes suppression or down-regulation of emotion and is accomplished by a combination of conscious and unconscious processes. (See Cassidy and Kobak, 1988; Mikulincer and Shaver, 2003, for further discussion of hyperactivation and deactivation.) These two coping strategies and their effects on mood and behavior have been documented in both cross-sectional and longitudinal studies.

Although most studies of adult attachment rely on self-report and observational methods, some attention has been paid to psychophysiological processes as well (see Diamond, 2001 for a review). Most such studies, however, have relied on surface indicators of autonomic arousal rather than using brain-imaging techniques (e.g., Carpenter and Kirkpatrick, 1996; Feeney and Kirkpatrick, 1996; Fraley and Shaver, 1997; Mikulincer, 1998). Recently, there has been a call for more direct exploration of the neurophysiological foundations of attachment-related behavior (Diamond, 2001; Hofer, 2003; Schore, 2000). Examination of the neural mechanisms of attachment would help to answer questions that cannot be fully answered using cognitive, behavioral, or surface physiological measures. For example, do attachment-anxious individuals activate emotions more intensely? Why do they find it difficult to regulate them? Do avoidant individuals use a particular kind of mental suppression to keep attachment-related negative emotions out of awareness? The latter question is particularly interesting because several recent studies suggest that avoidant suppression strategies are not as complete or reliable as more secure approaches to emotion regulation. That is, avoidant suppression is more subject to disruption by competing mental processes (e.g., Mikulincer et al., 2004).

To date, the few studies that have examined brain correlates of attachment have focused on laterality differences. Dawson et al. (2001), using EEG, found that insecurely attached infants exhibited relatively reduced left frontal brain activity. In preliminary work on adult emotion regulation (approach and withdrawal tendencies) and brain laterality, Cohen and Shaver (2004) found that avoidant individuals made more errors when judging positive attachment-related words presented to the right hemisphere. A mechanistic account of these laterality differences, and other attachment-related cognitive and emotional processing differences, will require in-depth characterization of attachment-style differences in brain function.

In the study reported here, we used functional magnetic resonance imaging (fMRI) to explore the neural underpinnings of emotion-regulation processes identified in previous behavioral experiments (e.g., Fraley and Shaver, 1997; Mikulincer et al., 2004). In those experiments, people who were involved in long-term romantic or marital relationships were asked, first, to vividly imagine that their partner was leaving them for someone else. After imagining this stressful experience for a few minutes, participants were asked to stop thinking about it. Results indicated that, on average, participants exhibited increased autonomic arousal

(assessed with skin conductance measures) while thinking about the breakup and loss. When asked to stop thinking about these negative scenarios, avoidant and secure individuals were able to down-regulate their negative emotions (as indicated by a reduction in skin conductance), but anxious individuals were not (Fraley and Shaver, 1997). Subsequent experiments showed that avoidant individuals’ ability to down-regulate their emotion in this situation could be disrupted by adding a cognitive load (remembering a 7-digit number). In contrast, a cognitive load did not affect secure individuals’ down-regulation of their emotions. These findings suggest that suppression is somewhat different for avoidant and non-avoidant individuals (Mikulincer et al., 2004).

These findings allowed us to make preliminary predictions to be tested in an fMRI experiment focused on the ability to cope with attachment-related thoughts and emotions. We expected that, when avoidant individuals were asked not to think about something, their pattern of brain activation and deactivation would be different from the pattern exhibited by non-avoidant (more secure) individuals. The nature of this difference might help to explain why avoidant, but not non-avoidant, people experience a rebound of suppressed material when a cognitive load is added, as if their suppression process was not reliable or complete. Several brain regions have been associated in previous studies with regulation and suppression of thoughts and emotions. These regions include the anterior cingulate cortex (ACC), orbital frontal cortex (OFC), lateral prefrontal cortex (LPFC), and more specifically the dorsolateral prefrontal cortex (DLPFC), and the subcallosal cingulate cortex (SCC) (e.g., Anderson et al., 2004; Drevets, 2000; Ochsner et al., 2002; Phan et al., 2002; Rolls et al., 2003; Wyland et al., 2003). These are some of the areas in which we expected to see “don’t think vs. think” differences as well as differences between avoidant and non-avoidant people during thought and emotion suppression.

Turning to the dimension of attachment anxiety, we know that attachment-anxious individuals become more emotional than their less anxious counterparts when thinking about breakups and losses. They also find it difficult to suppress negative thoughts and emotions even when asked to do so (Fraley and Shaver, 1997; Mikulincer et al., 2004). Therefore, compared to individuals who score low on the anxiety dimension, we expected anxious individuals to exhibit greater activation in brain regions associated with emotions and less activation in emotion-regulation areas. Some of the brain regions most consistently associated with emotional arousal are the amygdala, rostral anterior cingulate cortex (rACC), dorsal anterior cingulate cortex (dACC), ventromedial prefrontal cortex (VMPFC), insula, anterior temporal pole (ATP), and ventral striatum (for reviews, see Phan et al., 2002, 2004). Not all of these regions are associated with every emotion, however; and the different regions have different functions related to experiencing emotion or evaluating emotional states. For example, the amygdala is strongly associated with calculating emotional significance (e.g., Hare et al., 2005), whereas the ATP has been associated with sadness (e.g., Beaugard et al., 2001; Eugene et al., 2003; Lévesque et al., 2003) and the retrieval of emotional memories (Dolan et al., 2000; Shin et al., 1999).<sup>1</sup>

We expected attachment anxiety to be positively correlated with activation in the following regions associated with negative

<sup>1</sup> Many researchers associate the ATP with semantic processing. However, a growing number of studies implicate this region in the experience of sadness. In the present study we examined ATP activation in relation to sad thoughts and memories connected with relationship breakups and losses.

emotionality: (1) the amygdala, which is engaged when fear and anxiety are experienced (e.g., Adolphs, 2004); (2) the ATP, which is engaged when sadness is experienced (e.g., Blair et al., 1999; Beauregard et al., 2001; Eugene et al., 2003; Lévesque et al., 2003; Pelletier et al., 2003), especially in a social context (e.g., Calarge et al., 2003; Ranote et al., 2004); and (3) the ACC (both its rostral and dorsal subdivisions). The rACC is known to be engaged during emotion processing (e.g., Bush et al., 2000; Phan et al., 2003), and the dACC has been associated with subjective distress caused by physical pain (Rainville et al., 1997), social rejection (Eisenberger et al., 2003), and neuroticism (Eisenberger et al., *in press*).

The heightened emotionality of attachment-anxious individuals might result, not only from heightened emotionality per se, but also from failure to regulate it. The OFC has been implicated in thought regulation, particularly in relation to emotional and social stimuli (e.g., Beer et al., 2004; Miller and Cohen, 2001; Roberts and Wallis, 2000; Rule et al., 2002). Lévesque et al. (2003, 2004) reported a positive correlation between regulation of sadness and OFC activation in both women and young girls. These authors suggested that the OFC, through its anatomic projections to the limbic and paralimbic regions (specifically the ATP), is involved in behavioral inhibition and voluntary suppression of emotion (see also Cavada et al., 2000; Mesulam and Mufson, 1982). Thus, we expected attachment anxiety to be negatively correlated with activation in the OFC.

Individual differences related to activation of the ATP and the OFC have been demonstrated by Eugene et al. (2003) in a study examining the voluntary expression of sadness. Some study participants recruited the ATP and the insula (regions associated with emotional expression) while watching film clips about bereavement, whereas others recruited OFC and medial PFC. We therefore expected to find that attachment-anxious people would have higher activation in the ATP and lower activation in the OFC when thinking about relationship losses.

Finally, there is considerable evidence indicating that attachment-anxious people tend to ruminate on negative thoughts and feelings and become overwhelmed by distressing memories. For example, when asked to recall one negative memory, they tend to activate many other associated negative memories (Mikulincer and Orbach, 1995), something not observed in secure or avoidant individuals. Therefore, we were interested in exploring the possibility that attachment anxiety would correlate with activation in the hippocampus, a brain structure known to be important for associative memory retrieval (e.g., Cohen et al., 1999; Eichenbaum, 2004). The hippocampus is important for the retrieval of both emotional and non-emotional memories, including autobiographical memories (e.g., Maguire and Frith, 2003). Our prediction was that anxious people, who tend to recall previous negative experiences when thinking about a current negative event, would retrieve more memories while engaged in our task, which would involve greater hippocampal activation.

## Materials and methods

### Participants

Participants were 20 women students from the subject pool at the University of California, Davis, selected on the basis of their attachment scores. Each participant had either (1) a relatively high score on one of the two attachment dimensions (anxiety, avoidance) and a score close to the median on the other or (2)

low scores on the two dimensions (indicating a relatively secure attachment style).<sup>2</sup> Participants were healthy, right-handed, fluent English speakers. Their ages ranged from 18 to 25, with a mean age of 20. None had a prior history of neurological or psychiatric disorders. All gave written informed consent following instructions approved by the university's Institutional Review Board.

Immediately before the fMRI experiment, participants were given a sample task in which they read a hypothetical scenario similar to what they would see during the experiment and were guided through the instructions and the meanings of the different symbols (traffic lights, as explained below). The structure of a typical condition in the upcoming experiment (control block, thinking block, suppressing block, free-thinking block) was explained and demonstrated using the hypothetical scenario.

### Questionnaire measures

In a separate session, several weeks before the experiment took place, participants completed the Experiences in Close Relationships (ECR) scale (Brennan et al., 1998). Eighteen items on the scale measured attachment anxiety (e.g., "I worry about being abandoned") and 18 items measured avoidance (e.g., "I prefer not to show a partner how I feel deep down"). Participants rated the extent to which each item was descriptive of their experiences in close relationships on a 7-point scale ranging from "not at all" (1) to "very much" (7). The reliability and validity of the scales have been repeatedly demonstrated (e.g., Brennan et al., 1998; Mikulincer and Florian, 2000). In the current study, Cronbach alpha coefficients were high for the anxiety and avoidance scales (0.92 and 0.94, respectively). Neuroticism and general anxiety (i.e., trait anxiety) were also assessed because these personality traits are known to be correlated with attachment anxiety and might be legitimately viewed as offering alternative explanations of our findings (see Mikulincer and Shaver, 2003 for a review). Neuroticism was assessed with the 8-item neuroticism subscale from the Big Five Inventory (BFI; Benet-Martinez and John, 1998). Participants rated the extent to which an item was self-descriptive on a 5-point scale ranging from "disagree strongly" (1) to "agree strongly" (5). Cronbach alpha for the 8-item scale was high (0.87). Trait anxiety was assessed using Spielberger et al.'s (1983) 20-item Trait Anxiety Inventory (STAI), on which participants rated statements on a 4-point Likert scale ranging from "almost never" to "almost always," with higher scores indicating greater trait anxiety. Cronbach alpha for the STAI was 0.95.

After the experimental and anatomical scans, participants again completed the ECR scale, as well as questionnaires relating to demographics and their relationship history. The correlations between the pre-study and post-study ECR scores were high (0.83 for anxiety, 0.82 for avoidance). To maximize the reliability of participants' attachment scores, the two estimates were averaged.

### Behavioral tasks

Participants underwent five MRI scans (9 min, 43 s apiece), each of which included four task epochs (blocks). The task design

<sup>2</sup> The scores on attachment anxiety and avoidance (means on 7-point scales) for each participant were as follows: 6.00, 2.06; 1.89, 1.03; 4.69, 2.53; 3.92, 1.31; 2.11, 2.19; 5.08, 1.89; 2.22, 5.45; 4.66, 1.25; 3.83, 3.36; 5.92, 2.92; 4.05, 1.39; 2.70, 1; 5.39; 2.80; 4.64, 2.03; 4.58, 2.00; 1.50, 3.81; 2.55, 1.00; 3.64, 1.28; 1.78, 1.22; 1.72, 1.03.

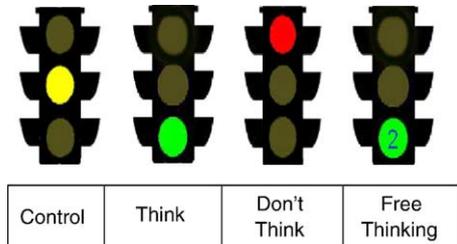


Fig. 1. Order in which the blocks were presented. In the control block, participants pressed a button every time the yellow light appeared. In the Think block, they pressed the button whenever they experienced a shift in thoughts or images. In the Don't Think block, they pressed the button whenever they happened to think about the forbidden topic. In the Free-Thinking block, they pressed the button whenever they happened to think about the formerly forbidden topic.

was based on a prior study of thought suppression (Mitchell et al., unpublished manuscript). A green traffic light (see Fig. 1) instructed participants to think for 2 min about a specific scenario and press a response button whenever they had a new thought or image related to the scenario (henceforth referred to as the Think block). A red traffic light instructed participants to try not to think about the previous scenario but to press the response button whenever they did happen to think about it (Don't Think block). A green traffic light with the number '2' superimposed on it instructed participants to think about whatever came to mind and press the response button if they happened to think about the previously excluded topic. Finally, a yellow traffic light meant simply that participants should press the response button. This yellow light block served as a visual and motor control for the other blocks. Participants indicated a change in thoughts (in the "thinking" conditions) or thinking about the forbidden topic (in the "don't think" conditions) by pressing a button with their right hand. Each block (120 s) occurred only once per scan. Results for the Think and Don't Think blocks are reported here.

The instructions for each block were presented for 20 s followed by a traffic light for 120 s. For example, just before the first Think block participants read the following instructions: "In the next 2 min (during which you will see a green traffic light with no number in it), please imagine as vividly as you can that you are driving alone to Lake Tahoe. Please press the button whenever a major shift in topic, image, scene, or activity occurs." Each of the five scenarios was either emotionally neutral, neutral but relationship-related, or emotionally negative and relationship-related. The order of the scenarios was the same for all participants. The second scenario involved remembering or imagining a neutral activity engaged with the participant's romantic partner, such as shopping in a mall or supermarket. The third scenario involved remembering or imagining a conflict or argument with the romantic partner. The fourth scenario involved remembering or imagining breaking up with the partner. The fifth scenario involved imagining that their partner died. At the end of each scan, participants were given a 30-s rest break. At the end of the fifth scan, they underwent a 10-min anatomical scan. They then rested and completed a series of questionnaires and were debriefed and asked about their thoughts and methods of suppression. Presentation software (Neurobehavioral Systems Inc., San Francisco, CA, USA) was used to present the traffic lights and instructions synchronously with fMRI data acquisition. Participants viewed the stimuli on a screen via a mirror in the head coil of the scanner.

### fMRI data acquisition and analysis

Brain images were acquired with a 1.5 T GE Signa scanner at the UC Davis Imaging Research Center. Head motion was minimized with comfortable padding around the participant's head. Functional images were acquired with gradient-recalled echo EPI sensitive to the blood oxygen level dependent (BOLD) contrast (TR = 2.5 s, TE = 40 ms, 24 contiguous 4 mm oblique axial slices parallel to the AC–PC line). After the experimental scans, high-resolution anatomical images were collected for each participant (TR = 12 ms, TE = 4.5 ms, voxel dimensions =  $1 \times 1 \times 1$  mm).

Data were preprocessed with SPM2 (Wellcome Department of Cognitive Neurology, London, UK) on the Matlab 6.5.1 platform (Mathworks Inc., Natick, MA). The first five brain volumes of each scan were discarded from the analysis to eliminate nonequilibrium effects of magnetization. The remaining 224 volumes were used for the subsequent analyses. Images were corrected for differences in timing of slice acquisition, and were then submitted to rigid body motion correction. Structural and functional volumes were spatially normalized to T1 and EPI templates in SPM, respectively. Templates are based on the MNI305 stereotaxic space (Cocosco et al., 1997), an approximation of Talairach space based on the Talairach and Tournoux (1988) atlas. The normalization algorithm consisted of a 12-parameter affine transformation together with a nonlinear transformation involving cosine basis functions, and resampled the volumes to  $2 \times 2 \times 2$  mm cubic voxels. Functional volumes were spatially smoothed with an 8-mm FWHM isotropic Gaussian kernel.

Statistical analyses were performed on individual participants' data with the general linear model implemented in SPM2. The fMRI time series data were modeled as a series of epochs convolved with a canonical hemodynamic response function. The resulting functions were used as covariates in a general linear model, along with a basis set of cosine functions that high-pass filtered the data, as well as a covariate for session effects. The data were high-pass filtered with the maximal frequency of task alternation (280 s) to cut off baseline drifts and low-pass filtered with a hemodynamic response function to control for temporal autocorrelation. The least-squares parameter estimates of height of the best-fitting synthetic HRF for each condition were used in pairwise contrasts, and the resulting contrast images, computed on a subject-by-subject basis, were submitted to group analyses. At the group level, contrasts between conditions were computed by performing one-tailed *t* tests on these images, treating participants as a random effect. Task-related responses were generally considered significant if they consisted of at least 5 contiguous voxels that exceeded an uncorrected threshold of  $P < 0.001$ , although in a few cases, when exploring details of individual differences, we used a more liberal threshold,  $P < 0.005$ .

Region-of-interest (ROI) analyses were performed with the MarsBar toolbox in SPM2 (Brett et al., 2002; <http://marsbar.sourceforge.net/>). We used functional ROIs for targeted regions identified in contrasts of interest: namely, the ATP, ACC, hippocampus, and SCC. Additionally, we used anatomical ROIs provided by MarsBar for regions that were of interest but not identified in the whole-brain contrasts: OFC (BA 11), LPFC (BA 9), and the amygdala. Mean contrast values for each participant and condition were extracted for each ROI and submitted to a repeated measures analysis and post-hoc comparisons. The goal of this exploratory study was to identify candidate regions that might be differentially engaged by individuals who differed in attachment style. Thus, a number of ROIs were examined (9 total), and the

resulting ROI analyses were not corrected for multiple comparisons. In the whole-brain analyses, we focused on four key contrasts: Don't Think > Think, Don't Think Relationship > Think Relationship, Don't Think Negative > Think Negative, and Think Negative > Think Neutral.

### Statistical models

Regression analyses were used to examine associations between signal intensity in various brain regions and scores on the two attachment-style dimensions. We conducted another set of regression analyses predicting activation in various brain regions from the average number of button presses in each block. When assessing statistical relations between different brain regions, we examined only relations that made sense anatomically (Büchel and Friston, 1997). Based on anatomical studies in non-human primates, we assumed that the ACC, OFC, and the ATP are interconnected in humans (e.g., Cavada et al., 2000; Mesulam and Mufson, 1982; Morán et al., 1987; Morecraft et al., 1992).

## Results

### Questionnaire measures

Attachment anxiety and avoidance were uncorrelated ( $r = 0.034$ ,  $P = 0.88$ ), as intended. As expected, there was a positive correlation between attachment anxiety and trait anxiety ( $r = 0.46$ ,  $P < 0.05$ ) and a trend toward a positive correlation between attachment anxiety and neuroticism ( $r = 0.42$ ,  $P = 0.065$ ). Neuroticism and trait anxiety were highly correlated ( $r = 0.81$ ,  $P < 0.001$ ).

### Behavioral data

Participants made fewer button presses in the not-thinking conditions than in the thinking conditions overall ( $t_{(1,19)} = 5.52$ ,  $P < 0.001$ ), in line with our instructions. Otherwise, the amount of button pressing did not differ significantly across the different scenarios and was not correlated with either attachment-style dimension. Nevertheless, there were small correlations within conditions compatible with previous studies (e.g., Fraley and Shaver, 1997; Mikulincer et al., 2004): i.e., more button presses for more anxious participants in the rebound, or Free Thought (green light with a “2” superimposed

on it), conditions, and fewer button presses for the more avoidant participants. The failure of these trends to reach statistical significance may be due to the small sample size (in contrast, for example, Fraley and Shaver, 1997 studied the reactions of 200 people, and Mikulincer et al., 2004 studied the reactions of 120 people).

### Neuroimaging data

#### Whole-brain comparisons

Our study combines two lines of research, the first dealing with individual differences in attachment style and their effects on emotion regulation and cognitive control and the other dealing with neural correlates of thought suppression. Although our main interest was attachment style, we began our analyses at the level of the entire group of 20 participants to see whether our suppression-related findings were in line with previous findings. In these group-level analyses, thought suppression was associated with higher activation in a region known to be related to control, conflict, and self-regulation: the dorsal ACC (see Fig. 2a; for review of relevant studies, see Botvinick et al., 2004). Specifically, participants exhibited greater activation in this region when trying not to think about a given scenario (collapsing across the five conditions that involved “not thinking”: neutral alone, neutral with partner, conflict with partner, breakup, and partner’s death) than when “thinking” about that same scenario ( $t_{(1,19)} = 4.92$ ,  $P < 0.001$ ). Activation while suppressing was also found in the MPFC (Fig. 2b) ( $t_{(1,19)} = 4.32$ ,  $P < 0.001$ ), a region known to be associated with self-related processing and monitoring (e.g., Gusnard et al., 2001; Johnson et al., 2002; Kelley et al., 2002).

Similar results were achieved when only attachment-related conditions were included in the comparison (ACC:  $t_{(1,19)} = 4.37$ ,  $P < 0.001$ ; MPFC:  $t_{(1,19)} = 4.13$ ,  $P < 0.001$ ) or when only negative conditions were included in it (ACC:  $t_{(1,19)} = 4.19$ ,  $P < 0.001$ ; MPFC:  $t_{(1,19)} = 3.36$ ,  $P = 0.002$ ). In summary, the group-level analyses revealed increased activation during thought suppression in several midline PFC structures, compatible with previous studies (Gusnard et al., 2001; Johnson et al., 2002; Kelley et al., 2002; Wyland et al., 2003).

#### Correlations with avoidance

We hypothesized that avoidant participants, as compared with non-avoidant ones, would have a different pattern of activation related to not thinking about a particular topic because, in behavioral studies, such people seem to retain some activation or availability of

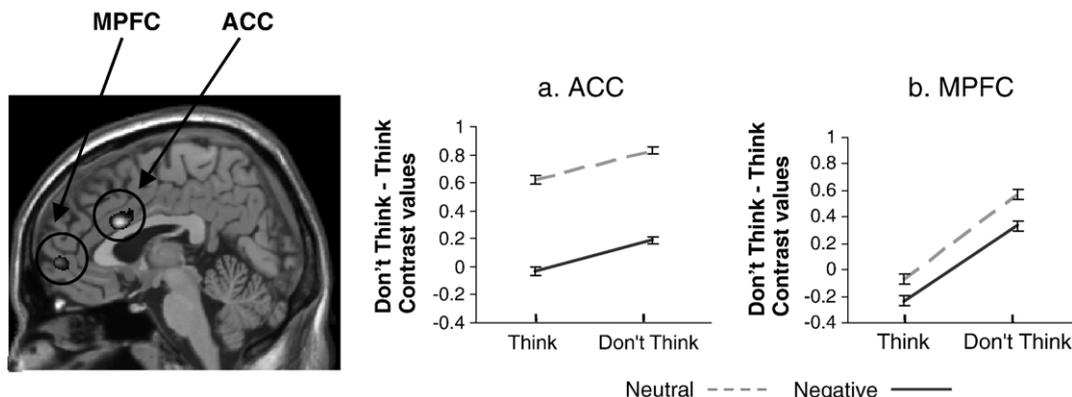


Fig. 2. Neural correlates of thought suppression revealed at the group level. The Don't Think > Think contrast revealed activation in the ACC (a) and MPFC (b).

loss-related thoughts even when some indexes (e.g., skin conductance) indicate that they have successfully suppressed them.

To test this prediction, we first examined whether avoidance is correlated with the pattern of activation noted in the whole-brain comparisons described above. We started by regressing the contrast “Don’t Think–Think” (collapsed across the five scenarios) on avoidance. This analysis revealed a positive association between avoidance and activation in the subcallosal cingulate cortex (SCC (0, 22, -2);  $t_{(1,19)} = 3.21$ ,  $P = 0.002$ ; see Fig. 3a). After looking at the whole-brain results, we examined the way in which attachment avoidance correlated with activation in the SCC while controlling for attachment anxiety (an unlikely confound but one we wished to rule out completely). In the first step of the regression analysis, we entered attachment anxiety, which turned out not to be significantly associated with relative activation in the SCC. In the second step, we entered attachment avoidance, confirming that relative activation in the SCC during “not thinking” was positively associated with avoidance ( $\beta = 0.54$ ,  $t_{(2,17)} = 2.70$ ,  $P = 0.015$ ).

To better understand the role of the SCC in the suppression process and to examine the differences in its activation between avoidant and non-avoidant individuals, we conducted a repeated measures analysis of variance, in which the “Don’t Think” and “Think” conditions were treated as a repeated measure and avoidance was treated as a predictor. The analysis revealed a significant interaction between avoidance and the “Don’t Think–Think” contrast ( $F(1,17) = 5.64$ ,  $P < 0.05$ ; see Fig. 3b). Tests for simple main effects revealed that, among participants scoring low on avoidance, not thinking was associated with lower SCC activation than thinking (Don’t Think:  $M = -0.68$ ,  $SD = 0.27$ ; Think:  $M = -0.22$ ,  $SD = 0.18$ ;  $F(1,9) = 6.03$ ,  $P < 0.05$ ). No such relative deactivation in the SCC was evident among individuals who scored high on avoidance ( $M = -0.09$ ,  $SD = 0.20$  for thinking;  $M = -0.07$ ,  $SD = 0.25$  for not thinking;  $F < 1$ ). Thus, non-avoidant participants tended to increase deactivation in the SCC when not thinking, whereas avoidant people did not. This suggests that, while everyone activated the same regions (ACC and MPFC) to suppress

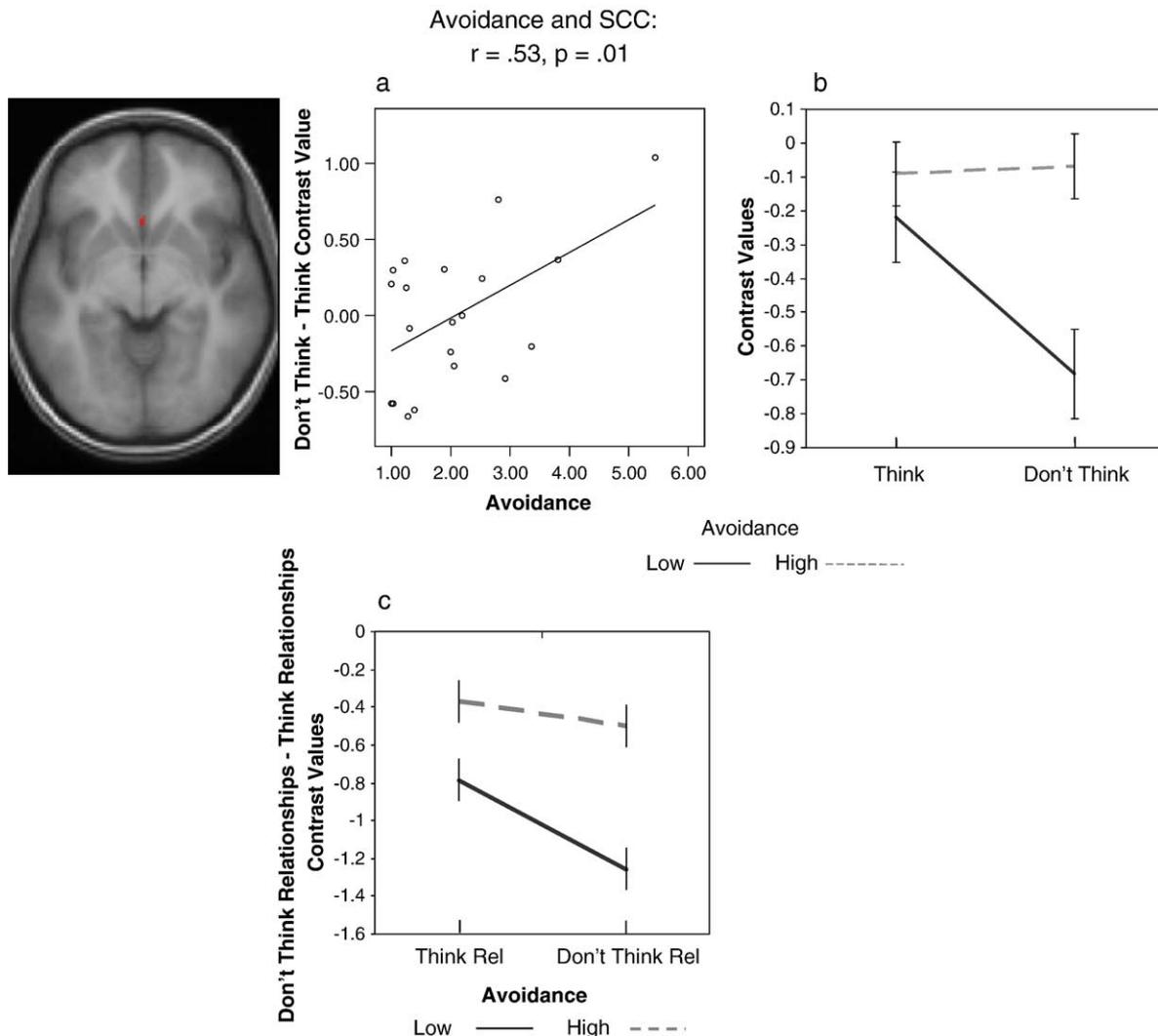


Fig. 3. Avoidant attachment was positively correlated with thought suppression (Don’t Think > Think) in the SCC (BA 25) (a). Interaction between avoidant attachment and Don’t Think vs. Think; the Y-axis in Panel (b) represents the contrast between each task condition (Think or Don’t think) and the control condition (thinking about driving alone). A similar pattern was revealed for the suppression of relationship-related thoughts. Panel (c) shows the interaction between avoidant attachment and “Don’t Think Relationships vs. Think Relationships”; again, the Y-axis represents the contrast between each task condition (Think relationship or Don’t think relationships) and the control condition (thinking about driving alone).

thoughts or “stop thinking,” non-avoidant participants focused their efforts in those regions while deactivating other regions. More avoidant participants activated the ACC and MPFC but did not show corresponding deactivation in the SCC.

The above analyses focused only on individual differences in attachment style, without regard to the nature of the material participants were thinking about or trying not to think about; however, previous studies of attachment-related cognitive and emotional processes focused mainly on relationship-related thoughts and emotions. Therefore, we conducted a series of analyses involving only relationship-related content. The relevant contrast was “Don’t Think Relationship–Think Relationship” (which excludes the condition in which participants thought about or tried not to think about driving alone to Lake Tahoe). As already noted, the whole-brain comparisons revealed similar patterns of activation in the ACC and the MPFC regardless of thought content.

We therefore examined possible effects of avoidance on suppression of relationship-related thoughts. We did this by regressing the contrast “Don’t Think Relationship–Think Relationship” on the avoidance score. The results were similar to those for the regression of the “Don’t Think–Think” contrast on avoidance; that is, although everyone activated certain brain regions during suppression (ACC and MPFC), the more avoidant participants showed more activation (or less deactivation) in another region. However, this time, unlike the previous regression involving avoidance, greater deactivation for the less avoidant participants occurred in the LPFC (BA9 (–38, 12, 26);  $t_{(1,19)} = 3.36$ ,  $P = 0.0017$ ; see Fig. 3c) rather than in the SCC. (As in the previous analysis, as indicated in Table 1, we used a more liberal threshold of  $P < 0.005$ ).

Table 1  
Whole-brain contrasts of interest

Region	~BA	Coordinates of peak activity	$P$ value	$t$	$Z$ scores
<i>(A) Thought suppression group contrast (Don’t Think &gt; Think)</i>					
L anterior cingulate cortex	32	–10 18 26	0.001	4.92	3.90
L medial prefrontal cortex	10	–8 58 2	0.001	4.32	3.56
<i>(B) Attachment-related thought suppression (Don’t Think Relationship &gt; Think Relationship)</i>					
L anterior cingulate cortex	32	–10 18 26	0.001	4.37	3.59
L medial prefrontal cortex	10	–8 56 2	0.001	4.13	3.44
<i>(C) Negative thought suppression group contrast (Don’t Think Negative &gt; Think Negative)</i>					
L anterior cingulate cortex	32	–14 20 24	0.001	4.19	3.48
L medial prefrontal cortex	10	–8 56 0	0.002	3.36	2.94
<i>(D) Regions in which the difference in activation between Think Negative and Think Neutral was correlated with attachment anxiety</i>					
L anterior temporal pole cortex	21	–38 2 –30	0.001	4.63	3.71 <sup>a</sup>
L hippocampus <sup>b</sup>		–32 –24 –14	0.001	3.47	2.99 <sup>a</sup>
L Anterior cingulate cortex <sup>b</sup>	24	–4 24 20	0.001	3.88	3.26 <sup>a</sup>
<i>(E) Region in which the difference in activation between Don’t Think and Think was correlated with attachment avoidance</i>					
Subcallosal cingulate cortex <sup>b</sup>	25	0 22 –2	0.002	3.21	2.82 <sup>a</sup>

<sup>a</sup> The correlation coefficients for these analyses are reported in the corresponding figures.

<sup>b</sup> Regression analyses were conducted using a statistical threshold of  $P < 0.005$ , uncorrected.

To better understand this result, we conducted a repeated measures analysis of variance, where activation in the “Don’t Think Relationship” and “Think Relationship” conditions was treated as a repeated measure and avoidance was the predictor. This yielded a significant interaction between avoidance and the repeated measure comparing the “Don’t Think Relationship” and “Think Relationship” conditions ( $F(1,17) = 6.97$ ,  $P < 0.05$ ; see Fig. 3c). Tests for simple main effects revealed that, among participants scoring low on avoidance, not thinking was associated with lower LPFC activation than thinking (Don’t Think Relationship:  $M = -1.26$ ,  $SD = 0.79$ ; Think:  $M = -0.79$ ,  $SD = 0.85$ ;  $F(1,9) = 31.85$ ,  $P < 0.001$ ). No such relative LPFC deactivation was evident among individuals who scored high on avoidance (Don’t Think Relationship:  $M = -0.50$ ,  $SD = 0.53$ ; Think:  $M = -0.37$ ,  $SD = 0.55$ ;  $F = 2.95$ ,  $P = 0.120$ ). Thus, non-avoidant participants tended to increase deactivation in the LPFC when not thinking about the relationship scenarios, whereas highly avoidant participants did not.

When we focused only on the emotionally negative relationship scenarios (conflict, breakup, and partner’s death), a similar pattern of deactivation in the LPFC was evident. When the “Don’t Think Negative–Think Negative” contrast was regressed on avoidance, we found a difference in the LPFC (BA9 (–38, 12, 26);  $t_{(1,19)} = 3.14$ ,  $P = 0.003$ ). To examine this result in greater detail, we conducted a repeated measures analysis of variance, where activation in the two conditions, Don’t think about-negative-relationship scenarios and Think about them, was treated as the repeated measure and avoidance was the predictor. This revealed a significant interaction between avoidance and the repeated measure comparing the “Don’t Think Negative” and “Think Negative” conditions ( $F(1,17) = 4.19$ ,  $P = 0.05$ ). Tests for simple main effects revealed that, among participants scoring low on avoidance, not thinking was associated with lower LPFC activation (i.e., greater deactivation) than thinking (Don’t Think Negative:  $M = -1.19$ ,  $SD = 0.79$ ; Think:  $M = -0.75$ ,  $SD = 0.92$ ;  $F(1,9) = 25.01$ ,  $P = 0.001$ ). No such relative LPFC deactivation was evident among individuals who scored high on avoidance (Don’t Think Negative:  $M = -0.58$ ,  $SD = 0.63$ ; Think:  $M = -0.45$ ,  $SD = 0.59$ ;  $F = 2.46$ ,  $P = 0.151$ ). Thus, non-avoidant participants tended to increase deactivation in the LPFC when not thinking, but the avoidant participants did not.

#### Correlations with anxiety

As expected, activation in emotion- and conflict-related regions was correlated with attachment anxiety. Specifically, when thinking about the negative scenarios (conflict, breakup, and death versus driving alone or shopping with one’s partner), higher activation was found in the left ATP, the left hippocampus, and the left dorsal ACC as a function of attachment anxiety (see Table 1 and Fig. 4). We conducted region-of-interest (ROI) analyses in each of these regions to better characterize the activation patterns. In these ROIs, we compared activation across the three negative conditions and the neutral “shopping with partner” condition, using the “driving alone” condition as a baseline for all four conditions. We conducted hierarchical regression analyses in which activation in the ATP, hippocampus, or ACC served as the dependent variable. Attachment anxiety, attachment avoidance, neuroticism, and trait anxiety (the latter three being potential confounds) served as the independent variables. In the first step of each regression analysis, we entered neuroticism and trait anxiety scores to control for their effects. In the second step, we entered the two attachment scores

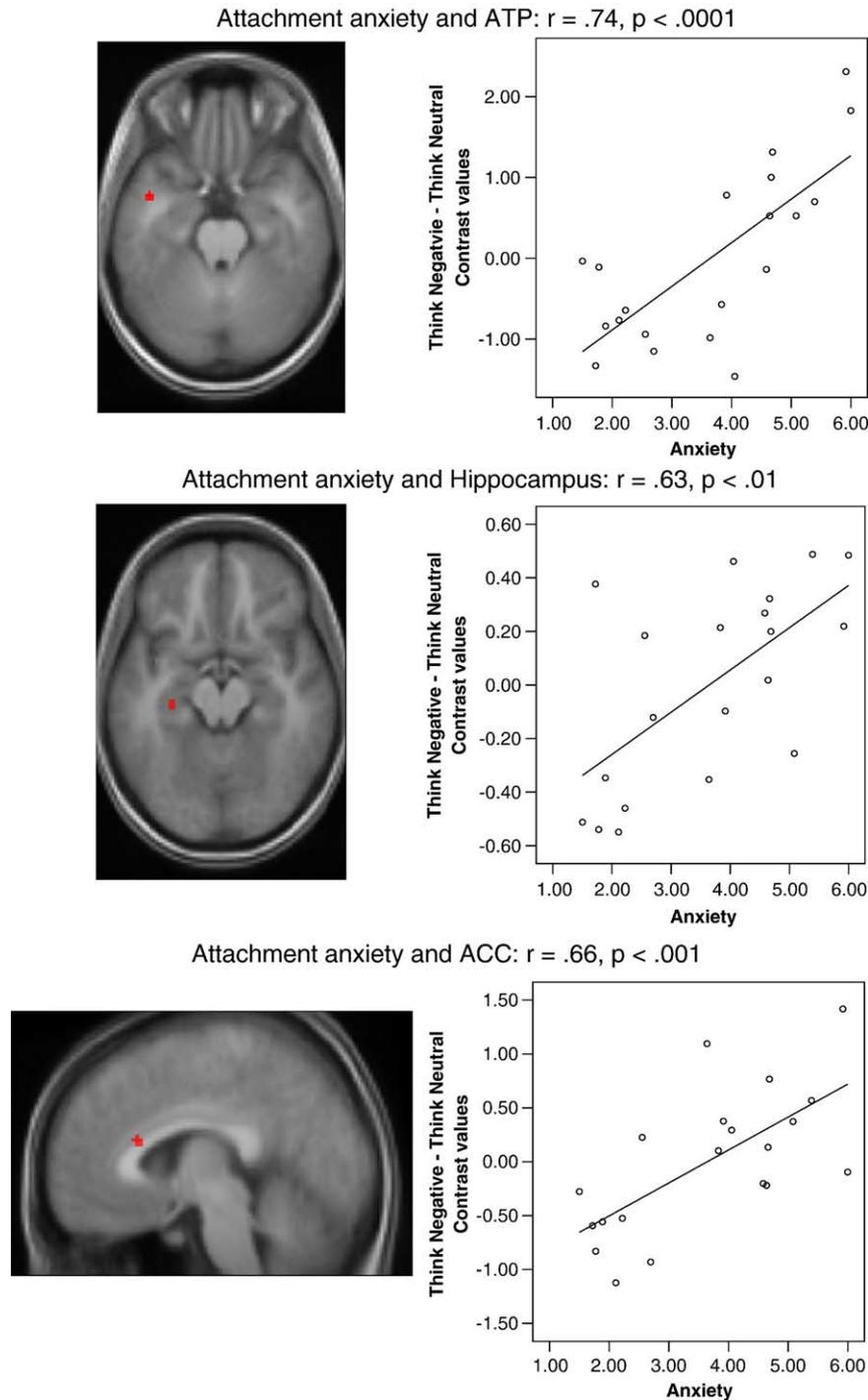


Fig. 4. Attachment anxiety and neural correlates of thinking negative thoughts. Across individuals, anxiety was associated with greater activation during negative thoughts (Think Negative > Think Neutral) in the ATP, hippocampus, and ACC.

(which allowed us to control for avoidance while considering the effects of anxiety). These analyses revealed that neuroticism and trait anxiety did not significantly predict activation in the regions of interest but that attachment anxiety was positively associated with relative activation during emotion-related thought (“Think Negative > Think Neutral”) in all three regions—ATP, hippocampus, and ACC—even when neuroticism, trait anxiety, and avoidance were respectively controlled (the  $\beta$  for anxiety predicting activation

in the ATP was  $0.91, t_{(4,15)} = 7.05, P < 0.0001$ ; the  $\beta$  for anxiety when predicting activation in the hippocampus was  $0.55, t_{(4,15)} = 2.57, P < 0.05$ ; the  $\beta$  for anxiety when predicting activation in the ACC was  $0.79, t_{(4,15)} = 3.78, P = 0.002$ ). We conducted similar analyses in which we used the residual attachment anxiety score, after statistically controlling for avoidance, neuroticism, and general anxiety, in our initial search for correlated regions of activation, and the same set of regions was identified.

In a regression analysis performed to examine the same contrast (“Think Negative > Think Neutral”) with respect to the left OFC (Fig. 5a), the relative degree of activation while thinking negative thoughts was negatively correlated with attachment anxiety, even when neuroticism, trait anxiety, and avoidance were controlled ( $\beta = -0.64$ ,  $t_{(4,15)} = -2.95$ ,  $P = 0.01$ ), revealing that more anxious participants failed to activate OFC as robustly as their less anxious counterparts. This finding led us to consider the possibility that activation in the ATP, which we interpret as indicating emotional arousal, was negatively correlated with activation in the OFC. The OFC is known to regulate emotion via its anatomical connections with the ATP (e.g., Cavada et al., 2000; Lévesque et al., 2003; Mesulam and Mufson, 1982). As predicted, there was a negative correlation between OFC and ATP activation ( $r = -0.57$ ,  $P = 0.009$ ) (Fig. 5b). To explore the possibility that attachment anxiety is related to these regions, we computed the correlation again while partialing attachment anxiety scores. The previously significant correlation became nonsignificant ( $r = -0.26$ , ns). In contrast, the correlation remained significant when neuroticism and trait anxiety were partialled ( $r = -0.57$ ,  $P = 0.01$ ;  $r = -0.56$ ,  $P = 0.01$ ), suggesting that the effects of attachment anxiety on activation in the ATP and relative deactivation in the OFC are specific and not due to a more generic form of anxiety.

Finally, as discussed in the Materials and methods section, since our study was exploratory, we examined a relatively large number of ROIs (e.g., amygdala and LPFC), nine in all. There were null effects for all of the ROIs not reported above. Furthermore, although not directly related to the goals of the present study, we examined the neural correlates of general anxiety and neuroticism when participants were thinking negative thoughts because these correlates might be important both for understanding the underlying mechanisms of general anxiety and for distinguishing general anxiety from attachment-specific anxiety. The relevant correlations are shown in Table 2. In general, different brain regions were identified for the three different constructs (attachment anxiety, general anxiety, and neuroticism), providing further support for the claim that they are different.

## Discussion

In general, we replicated previous findings (e.g., Wyland et al., 2003) associating thought suppression with higher activation in the ACC and MPFC. Based on prior behavioral research on adult attachment style, we predicted that people high on attachment anxiety would show greater activation in emotion-related brain regions when thinking about negative attachment-related events (conflicts, breakups, death of partner). In line with our predictions, attachment anxiety was associated both with higher activation in the ATP, a region associated with sadness (Lévesque et al., 2003), and with lower activation in the OFC, a region associated with emotion regulation (Beer et al., 2004; Lévesque et al., 2003). The ATP and OFC are known to be anatomically connected (Cavada et al., 2000; Lévesque et al., 2003; Mesulam and Mufson, 1982), so it is possible that the OFC suppresses sadness through top-down modulation of activity in the ATP. Consistent with this possibility, we found that the two regions were negatively correlated across participants, such that those who exhibited the lowest levels of OFC activation tended to exhibit the highest levels of ATP activation. This correlation was reduced to insignificance when scores on attachment anxiety were statistically controlled because attachment anxiety was associated with high ATP activation and low OFC activation. It seems possible, therefore, that people’s self-reports of high attachment anxiety are based on repeatedly experiencing high emotional arousal and low ability to down-regulate it.

We also expected participants high on attachment anxiety to show greater activation in memory-related regions when thinking about negative attachment-related events. In line with previous studies in which highly attachment-anxious individuals had greater access to negative memories (Mikulincer and Orbach, 1995), anxiety was associated with higher activation in the hippocampus, which is known to be important for memory retrieval (for reviews, see Eichenbaum, 2004). Although we cannot know for sure that *negative* memories were being activated since the hippocampus plays a general role in memory retrieval, we infer, based on previous behavioral findings in the attachment literature, that

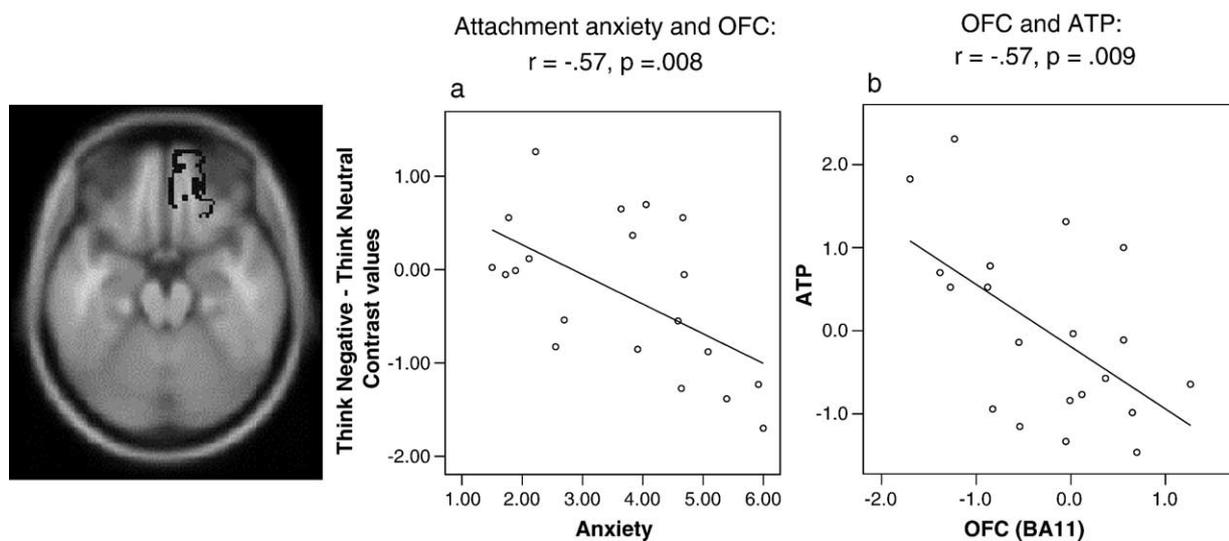


Fig. 5. Attachment anxiety was negatively correlated with negative thoughts (Think Negative > Think Neutral) in the OFC (BA 11) (a). OFC activation was negatively correlated with ATP activation (b).

Table 2  
Whole-brain contrasts of interest that are correlated with neuroticism or general anxiety

Region	~BA	Coordinates of peak activity	<i>P</i> value	<i>t</i>	<i>r</i>
<i>(A) Regions in which the difference in activation between Think Negative and Think Neutral was correlated with general anxiety</i>					
R parahippocampal gyrus <sup>a</sup>	37	32 -52 2	0.001	3.56	0.64
R caudate (tail) <sup>a</sup>		20 -42 14	0.001	3.52	0.64
L visual association cortex <sup>a</sup>	31/18	-20 -72 18	0.002	3.24	0.61
R lateral orbitofrontal cortex <sup>a</sup>	11	36 46 -12	0.004	2.99	0.58
<i>(B) Regions in which the difference in activation between Think Negative and Think Neutral was correlated with neuroticism</i>					
R parahippocampal gyrus	19/39	32 -52 2	0.002	3.24	0.61
R parahippocampal gyrus, precuneus	30	18 -42 8	0.003	3.06	0.58
R caudate (tail)		28 -40 16	0.003	3.16	0.60
R posterior cingulate gyrus	31	22 -28 42	0.003	3.05	0.58
R lateral orbitofrontal cortex	11	38 46 -12	0.0001	4.96	0.76
L lateral orbitofrontal cortex	11/47	-40 38 -16	0.002	3.37	0.62
R inferior frontal gyrus, orbital	47	36 38 -6	0.0001	4.18	0.70
L inferior frontal gyrus, orbital	47	-42 40 -8	0.001	3.61	0.65
L inferior frontal gyrus, orbital	47	-50 34 -4	0.001	3.48	0.63
R rostromedial prefrontal cortex	10	36 50 8	0.0001	3.96	0.68
L rostromedial prefrontal cortex	10	-26 50 -4	0.0001	4.33	0.71
L rostromedial prefrontal cortex	10	-24 50 6	0.001	3.78	0.67
L medial orbitofrontal cortex	10	-4 58 -6	0.001	3.63	0.65
R superior/middle frontal gyrus	6	32 28 58	0.002	3.25	0.61
L anterior insula		-22 26 16	0.001	3.76	0.66

<sup>a</sup> Regression analyses were conducted using a statistical threshold of  $P < 0.005$ , uncorrected.

activation in the hippocampus while thinking about negative social experiences was related to recalling negative memories. This inference should be tested more rigorously in future studies.

Finally, anxiety was associated with activation in the dorsal ACC, a region that has been associated with the subjective distress of physical pain (Rainville et al., 1997), social rejection (Eisenberger et al., 2003), and neuroticism (Eisenberger and Lieberman, 2004; Eisenberger et al., in press). It has also been associated with performance monitoring and detection of mental conflict (Bush et al., 2000). In our study, one region in dACC [-4, 24, 20] was positively correlated with anxiety during negative thoughts, whereas another region in dACC [-14, 20, 24] was activated in general when participants were asked not to think about a particular topic. Activation in this latter region was uncorrelated with anxiety. Further investigations are needed to determine the significance of these two distinct regions in dACC. Taken together, our results provide further support for the idea that anxious

individuals experience negative emotions intensely, have greater access to a web of negative memories, and find it difficult to suppress negative feelings. This higher activation and greater memory accessibility may be one reason for their inability to inhibit the spread of activation across negative thoughts, memories, and feelings.

Also based on behavioral research on adult attachment style, we expected avoidant people to use a somewhat different strategy for “not thinking” as compared with non-avoidant people. In line with this expectation, we found that more avoidant people maintained stable levels of activation in regions where less avoidant people showed greater deactivation when not thinking about particular scenarios. The decreases in the SCC and LPFC (BA 9) among the less avoidant participants resembled the regional decreases in blood flow noticed in other prefrontal regions in previous studies and labeled “task-induced deactivation” (Binder et al., 1999; Mazoyer et al., 2001; Shulman et al., 1997).

Hester et al. (2004) suggested that deactivation in the left medial frontal gyrus (BA 9) is related to successful inhibition in a task requiring inhibition. Furthermore, successful inhibition involves both activation of task-relevant areas and deactivation of task-irrelevant ones (Hester et al., 2004). In the Hester et al. study, when cues signaled a forthcoming need for cognitive control (inhibition of a response), increased activation occurred in frontal, parietal, and cingulate regions (such as the dorsal ACC), and deactivation occurred in regions not required for cognitive control. Such deactivation occurred in the left medial frontal gyrus (but also in the left insula, caudate, and precuneus/posterior cingulate). The left medial frontal region was also significantly deactivated when Hester et al. (2004) compared the cue period prior to successful inhibitions with the cue period prior to failures. In other words, failure to decrease activation in the left medial frontal region in concert with “attentional” increases in other areas was directly related to poor performance. Hester et al. suggested that cueing provided an opportunity for top-down control to decrease the resources devoted to monitoring internal states (Gusnard and Raichle, 2001; Gusnard et al., 2001; Raichle et al., 2001), while at the same time increasing attentional resources devoted to processing external stimuli. In our study, this kind of coordinated process, although occurring in different brain regions (SCC and LPFC), was evident only among non-avoidant participants.

Since the LPFC is involved in the integration of emotion and cognition, and in the regulation of emotion (Gray et al., 2002; Lévesque et al., 2004), including emotions related to approach or withdrawal (Davidson, 1995; Harmon-Jones and Sigelman, 2001), it makes sense that the contrasts between “Don’t Think Relationship–Think Relationship” and “Don’t Think Negative–Think Negative” resulted in differential avoidance-related deactivation in the LPFC, whereas more general, less specifically emotional, contrasts produced avoidance-related differences in the SCC. In both cases, more avoidant individuals showed less differential deactivation during suppression, but the regions in which the difference occurred differed somewhat based on what was being suppressed.

Our finding that more avoidant people failed to fully deactivate certain regions while suppressing thoughts and emotions seems compatible with previous finding from behavioral studies (e.g., Mikulincer et al., 2000, 2004). In those studies, avoidant people failed to maintain suppression when a cognitive load was added to a suppression task, something that did not happen with less avoidant people. Future imaging studies could determine what happens in the brain when avoidant people become unable to

suppress certain mental contents while operating under a cognitive load and whether the failure to deactivate regions that are directly related to the suppression task is related to the disruptive effects of the load.

Although preliminary, our study yielded promising results. The questions under investigation are rooted in a well-established theory, and the experimental procedures employed have been used in previous cognitive and behavioral experiments. Moreover, the inclusion of individual differences enabled us to look beyond seemingly general processes that are likely to be clouded by such differences. For example, another interpretation of Eugene et al.'s (2003) study, described in the introduction, is that their first group of participants included a relatively high proportion of anxious individuals, whereas their second group included a relatively high proportion of avoidant individuals. Many supposedly general mental processes documented by social cognition researchers have turned out to be moderated by attachment style (for a review, see Mikulincer and Shaver, 2005). Using brain imaging techniques to study such social cognitive processes as they are moderated by attachment style should reveal where and how the differences arise. This new methodology may clarify individual differences that could previously be studied only behaviorally or via self-reports and interviews.

Although we view this study as encouraging, it is important to consider some of its limitations. First, we studied only women to eliminate gender-related variance in the issues under investigation. Second, our sample size was small for an individual-differences study, and we therefore included relatively extreme scorers on the attachment scales. While doing this, we discovered that it was easier to recruit secure and anxious participants for a study of “close relationships and the brain” than to recruit avoidant participants. We ended up excluding many secure and anxious volunteers while working hard to attract avoidant ones. This might have been related to our requirement that participants be involved in a serious long-term relationship, which many avoidant college students are not. Whatever the reasons, we were able to represent the anxiety dimension of attachment style more completely than the avoidance dimension, making it possible that more or clearer findings related to avoidance will be obtained in future studies with larger, more representative samples. This is especially important in light of the liberal threshold ( $P < 0.005$ ) we used for identifying the differences between avoidant and non-avoidant participants.

We were not able to include all relevant control conditions in our experiment. Inclusion of a non-attachment-related negative emotion condition will be important in future studies because we cannot yet be sure how many of our findings can be attributed to attachment-related negative emotions alone and how many might be replicated when we include non-attachment-related stimuli and situations. Finally, it is difficult to infer, based on our findings, what psychological process is associated with ATP activation. Although some investigators have linked ATP activation to affective processing, as we are inclined to do here, others have linked it to semantic processing (e.g., Noppeney and Price, 2002; Vandenberghe et al., 2002). Thus, further investigation of the ATP activations in relation to attachment anxiety is necessary.

## Conclusion

Despite these limitations, the study revealed associations between self-reported attachment style and brain activation in

regions associated with emotion, memory, and emotion regulation. The results fit well with other neuroscientific studies of emotion regulation (e.g., Ochsner and Gross, 2004), which suggests that emotional processes are modulated by top-down control from the OFC and PFC and bottom-up processes in the anterior temporal pole and hippocampus. Our findings indicate that individual differences play an important role in emotion regulation, suggesting that future studies should include relevant individual-difference measures. Many psychological processes related to people's immersion in a social environment are likely to be moderated by such individual differences.

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