Emotional Conflict and Neuroticism: Personality-Dependent Activation in the Amygdala and Subgenual Anterior Cingulate

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The amygdala and subgenual anterior cingulate (AC) have been associated with anxiety and mood disorders, for which trait neuroticism is a risk factor. Prior work has not related individual differences in amygdala or subgenual AC activation with neuroticism. Functional magnetic resonance imaging was used to investigate changes in blood oxygen level-dependent signal within the amygdala and subgenual AC associated with trait neuroticism in a nonclinical sample of 36 volunteers during an emotional conflict task. Neuroticism correlated positively with amygdala and subgenual AC activation during trials of high emotional conflict, compared with trials of low emotional conflict. The subscale of neuroticism that reflected the anxious form of neuroticism (N1) explained a greater proportion of variance within the observed clusters than the subscale of neuroticism that reflected the depressive form of neuroticism (N3). Using a task that is sensitive to individual differences in the detection of emotional conflict, the authors have provided a neural correlate of the link between neuroticism and anxiety and mood disorders. This effect was driven to a greater extent by the anxious relative to the depressive characteristics of neuroticism and may constitute vulnerability markers for anxiety-related disorders.

Keywords: amygdala, subgenual anterior cingulate, neuroticism, anxiety, personality

The amygdala (Drevets et al., 1992; Rauch, Shin, & Wright, 2003) and subgenual anterior cingulate (AC; Drevets et al., 1997; Rauch et al., 1994) have been associated with anxiety and mood disorders, for which neuroticism is a risk factor (Bienvenu et al., 2001; Durrett & Trull, 2005; Trull & Sher, 1994). Although prior work has reported significant correlations between neuroticism and brain activation in temporal, frontal, and caudal cingulate areas using affective (Canli et al., 2001) and cognitive (Eisenberger, Lieberman, & Satpute, 2005) task paradigms, it is currently unknown whether there is a significant association between neuroticism and activation within areas implicated in anxiety and mood disorders: the amygdala and the subgenual AC (Drevets, 2000; Gotlib et al., 2005; Rauch et al., 2003). Understanding the relationship between personality and brain function in regions implicated in psychopathology may provide insights into why some individuals are more vulnerable to certain psychological disorders rather than others. The current research used functional magnetic resonance imaging (fMRI) and an emotional conflict task to investigate the relationship between neuroticism and blood

oxygen level-dependent (BOLD) signal change within the amygdala and subgenual AC.

We used the Word-Face Stroop task (Haas, Omura, Constable, & Canli, 2006a) to investigate amygdala and subgenual AC activation in response to emotional conflict. In this task, subjects judge the valence of words that are either incongruent or congruent with a face that they are overlaid upon. This task combines several of the affective and attentional characteristics implicated in neuroticism (Suls & Martin, 2005; Wallace & Newman, 1998), as well as emotional attention and cognitive processes associated with amygdala (Zald, 2003) and cingulate (Bush, Luu, & Posner, 2000; Devinsky, Morrell, & Vogt, 1995) function. The amygdala is engaged in a wide variety of processes ranging from the modulation of perceptual and attentional recourses (Morris et al., 1998) to implicit face processing (Whalen et al., 1998), whereas the subgenual AC is engaged in negative mood (Phan, Wager, Taylor, & Liberzon, 2004) and attentionally demanding visuospatial processes (Drevets & Raichle, 1998). On the basis of findings that have associated high neuroticism with increased sensitivity to emotional experiences (Suls & Martin, 2005), discrepancy detection (Eisenberger et al., 2005), and psychopathology (Durrett & Trull, 2005), we predicted that neuroticism would be positively correlated with increased activation during trials of high emotional conflict, compared with trials of low emotional conflict, in the amygdala and subgenual AC.

We further sought to dissociate aspects of anxious and depressive types of neuroticism in this study. We therefore chose a personality instrument, the revised NEO Personality Inventory (NEO–PI–R; Costa & McCrae, 1992b), that conceptualizes neuroticism as the sum of various subfacets, two of which are anxious (N1, Anxiety) and depressive (N3, Depression) forms of neuroticism. Both the amygdala and subgenual AC have been shown to be

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hyperactive in anxiety (Drevets & Raichle, 1998; Rauch et al., 2003) and depression (Drevets, 2000). Attentional bias in response to affective information has also been associated with both anxiety and depressive disorders (Bar-Haim, Lamy, & Glickman, 2005; Gotlib, Krasnoperova, Yue, & Joormann, 2004), although it has been suggested that this effect may be more pronounced in anxiety than in depression (Mogg & Bradley, 2005). In the current study, involving a nonclinical population, we aimed to identify the unique contribution of anxious and depressive forms of neuroticism to individual differences in amygdala and subgenual AC response to emotional conflict.

Method and Materials

Subjects

Thirty-six right-handed subjects (20 men and 16 women) were recruited from Stony Brook University and Yale University and were scanned at the Magnetic Resonance Research Center at Yale University. The subjects' mean age was 24.2 years ($SD = 5.13$, range 18–44). Subjects had no history of brain injury, reported no substance abuse within the past six months, were not on any mood-altering medication, and had no physical limitations that prohibited them from participating in an fMRI study. This study was conducted with the approval of the institutional review boards of both Stony Brook University and Yale University. Informed consent was obtained from all subjects.

Prior to scanning, all subjects completed the NEO–PI–R (Costa & McCrae, 1992b) and the Profile of Mood States (McNair, Lorr, & Droppleman, 1971). Use of the NEO–PI–R allowed us to dissociate aspects of neuroticism, in particular to isolate the unique contributions of the Anxiety (N1) and Depression (N3) subscales. These two subscales are presumed to reflect the personality char-

acteristics associated with each of these disorders, respectively (Bagby, et al., 1997; Bienvenu et al., 2001; Costa & McCrae, 1992a, 1992b; Durrett & Trull, 2005).

Personality data were scored to represent T values, with the population mean defined as T = 50 and one standard deviation of T = 10. Thus, the sample scores for neuroticism ($M = 51.83$, $SD = 10.19$), anxiety (N1; $M = 49.58$, $SD = 9.83$) and depression (N3; $M = 56.42$, $SD = 10.80$) were well within the range of the normal nonclinical population. As expected, the intercorrelations between neuroticism and subscale scores, as well as between anxiety (N1) and depression (N3; $r = .62$, $p < .001$), were all highly significant. There were no significant positive intercorrelations between neuroticism and any of the other four personality traits (extraversion, openness, agreeableness, conscientiousness). In addition, there were no significant differences as a function of gender for trait neuroticism or scores on either of the subscales.

Task

In order to assess changes in brain function associated with emotional conflict, we used the Word-Face Stroop task (Haas et al., 2006a). In this task, subjects were asked to make emotional valence (positive, negative, or neutral) judgments of words overlaid upon faces. During trials of high emotional conflict the emotional valence of the word and face was incongruent, whereas during trials of low emotional conflict the emotional valence of the word and face was congruent.

Words were projected approximately across the noses of faces (see Figure 1). Groups of 30 positive, negative, and neutral emotionally valenced words were selected from a stimulus library, the Affective Norms for English Words set (Bradley & Lang, 1999). These three groups of words differed significantly in normed valence ratings, $F(2,$
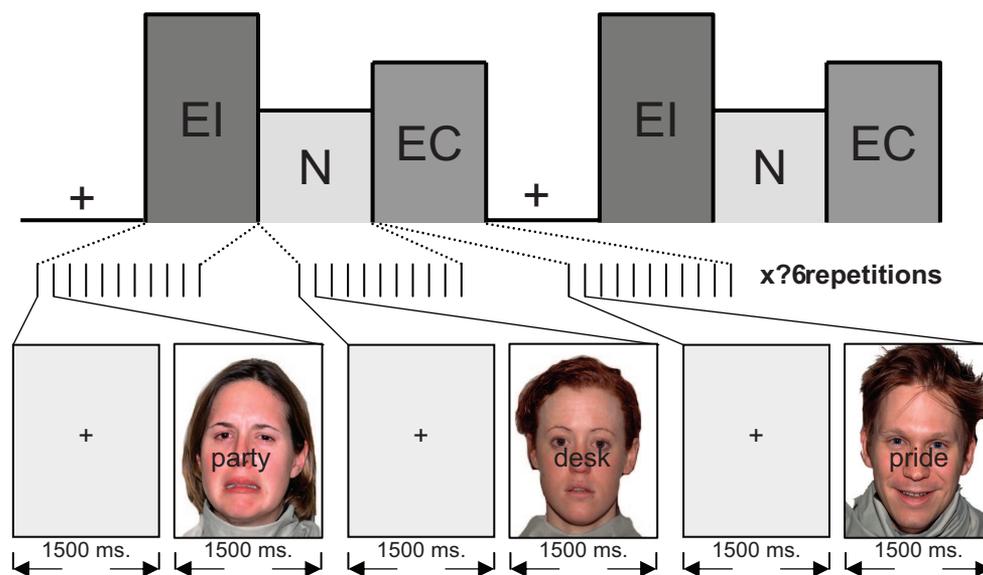
Figure 1. Schematic representation of an experimental paradigm. Stimuli were either emotionally incongruent (EI), neutral (N), or emotionally congruent (EC). Stimuli were presented in a total of 6 blocks of 10 trials for each condition. Each trial consisted of a 1,500 ms presentation of a fixation cross followed by a 1,500 ms presentation of the stimulus. Emotional conflict was assessed by comparing the blood oxygen level-dependent (BOLD) signal obtained during EI trials relative with the BOLD signal obtained during EC trials.

87) = 1,492.40, $p < .0001$, but did not differ in word length, $F(2, 87) = 0.47$, $p = .62$, or word frequency, $F(2, 87) = 0.05$, $p = .95$. Groups of 30 happy, sad, and neutral color faces were selected from the MacArthur–McDonnell face library (Tottenham, Borscheid, Ellertsen, Marcus, & Nelson, 2002). Each group of faces consisted of an equal number of males and females. By combining the words and faces, three groups of stimuli were created (emotionally incongruent [EI], emotionally congruent [EC], and neutral [N]). EI stimuli were made up of either positive or negative words selected at random and projected onto randomly selected faces of opposite valence (e.g., the word *party* on a sad face). EC stimuli were made up of either positive or negative words selected at random and projected onto randomly selected faces of like valence (e.g., the word *pride* on a happy face). N stimuli were made up of randomly selected neutral words selected at random and projected onto randomly selected neutral faces (e.g., the word *desk* on a neutral face). In order to assess changes in amygdala and subgenual AC activation in response to emotional conflict, we limited the following analysis to the EI and EC conditions.

Subjects were asked to judge the valence of each word (positive, negative, or neutral) as quickly and as accurately as possible via a button box. Stimuli were presented in counterbalanced blocks of 10 trials each for each condition (EI, EC, N, and fixation). Each trial consisted of a 1,500-ms fixation cross, followed by 1,500-ms presentation of the stimulus. Each subject viewed 24 blocks (6 per condition) and thus responded to 180 trials (60 per condition). Reaction time (RT) latencies and accuracy were recorded during the 1,500 ms following the onset of each stimulus. We performed a series of correlation analyses between RT and the neuroticism measures in order to investigate behavioral attentional bias relationships.

Image Acquisition

Whole-brain imaging data were acquired on a 3 Tesla Siemens Trio Scanner (Siemens Corporation, New York, NY). For structural whole-brain images, a three-dimensional high-resolution spoiled gradient scan and a T1 scan (24 slices, 5 mm thickness; oriented parallel to the line between the anterior and posterior commissure) were conducted. Functional images were acquired using a gradient echo T2*-weighted echoplanar imaging scan with a flip angle of 80°, a repetition time of 1.5 s, an echo time of 30 ms, and a field of view of a 220×220 mm matrix (voxel size: $2 \times 2 \times 2$ mm).

Image Analysis

Functional data were preprocessed and statistically analyzed using statistical parametric mapping (SPM2; Wellcome Department of Imaging Neuroscience, 2003). The images were temporally realigned (motion corrected) to the middle slice; spatially realigned to the first in the time series; and coregistered to the T1 volume image, which was segmented and normalized to the gray matter template. Spatial transformations derived from normalizing the segmented gray matter were then applied to all functional volumes, which were then spatially smoothed with an 8 mm full width at half maximum isotropic Gaussian filter. Fixed-effects models (Friston, 1994) were used at the individual subject level of analysis, and random effects models (Holmes &

Friston, 1998) were used for group-level analyses. At the individual level, models were created (general linear model) in order to represent all blocked conditions (EI, EC, N, and fixation), and all data were then high-pass filtered. To determine areas significantly activated in response to emotional conflict, a one-sample t test subtracting BOLD signal obtained during EC trials from BOLD signal obtained during EI trials was performed for each subject. We conducted group-level analyses to investigate brain activation in response to emotional conflict that was associated with neuroticism. We entered each subject's neuroticism score into a regression analysis that was based on contrast images between the EI and EC conditions. In order to control for negative mood, we also entered this factor as a covariate.

A second set of analyses was performed to isolate the individual and unique contributions of the anxious (N1) and depressive (N3) forms of neuroticism. This analysis was performed in order to investigate if the signal change associated with neuroticism in regions implicated in anxiety and mood disorders is more driven by anxious- or depressive-related personality characteristics. We therefore restricted this analysis to significant clusters obtained in the previous regression analysis and conducted two independent analysis approaches. The first analysis approach was conducted using statistical parametric mapping to quantify the number of voxels (within clusters that had previously been associated with neuroticism) that were uniquely associated with the anxious (N1) or depressive (N3) form of neuroticism. For this approach, we conducted a multiple regression analysis in which we entered simultaneously individuals' scores for both the anxious (N1) and depressive (N3) forms of neuroticism and set weights to identify voxels whose activation was significantly correlated with one subscale while partialing out the other. In the second analysis approach we used a statistical analysis program (SPSS) to quantify the percentage of observed variance that could be attributed to either the anxious (N1) or depressive (N3) form of neuroticism. For this approach, we extracted the mean percent signal change (in response to emotional conflict) from each of the clusters for each subject and simultaneously entered them into a multiple regression analysis predicted by each of the neuroticism subscales.

We initially performed a whole-brain analysis and then verified the anatomical location of observed clusters by using anatomical masks. These masks were created for the functional analyses that restricted observed activations to the a priori regions of interest (i.e., amygdala and subgenual AC). We used an automated method for generating the region of interest mask for the amygdala based on the Talairach Daemon database (Maldjian, Laurienti, Kraft, & Burdette, 2003). The region of interest mask for the subgenual AC was created by drawing a 20 mm radius sphere surrounding coordinates (Montreal Neurological Institute [MNI]: –2, 32, –2) previously implicated in subgenual AC function associated with anxiety (Drevets, Videen, Synder, MacLeod, & Raichle, 1994) and depression (Drevets et al., 1997). We confirmed the anatomical location of each cluster on a subject by subject basis by overlaying each cluster on each subject's normalized T1 scan. We applied a significance threshold of $p < .005$ (uncorrected) and 15 contiguous voxel spatial extent threshold, yielding a per-pixel appropriate false-discovery rate of $p < .00001$ (Forman et al., 1995) in order to identify clusters within these structures associated with neuroticism.

Results

Behavioral Measures

Consistent with our previous results (Haas et al., 2006a), RT latencies were greater during the EI condition ($M = 1,024.93$, $SD = 124.68$) than they were during the EC condition ($M = 989.59$, $SD = 125.89$), $t(1, 36) = 4.19$, $p < .001$. This significant difference in RT validates that the current task is effective in producing interference as a function of emotional conflict. Error rates were only slightly higher during EI trials (8.76%) relative to EC trials (7.58%). This difference did not reach statistical significance, $t(1, 36) = 1.38$, $p = .18$.

We next investigated the association of neuroticism with the behavioral interference measure of emotional conflict (EI–EC RT). This analysis revealed that, regarding neuroticism, anxiety (N1), and depression (N3), none were significantly associated with the RT difference between EI and EC trials. An analysis of RT in each condition independently revealed that anxiety (N1) was associated with increased RT during EI trials ($r = .70$, $t(34) = 2.22$, $p < .05$), whereas neuroticism ($r = .25$, $p = .14$) and depression (N3; $r = .14$, $p = .40$) were not. There were no significant relationships between any of the personality or mood measures and RT during EC trials. There were also no significant relationships found between error rates and neuroticism, anxiety (N1), or depression (N3) during any of the conditions.

Amygdala Activation as a Function of Neuroticism

Consistent with our predictions, amygdala activation during high-conflict EI trials, relative to low-conflict EC trials, correlated significantly with neuroticism. Figure 2 displays the activation within the left (MNI: –20, –8, –16; 34 voxels, $t(34) = 3.87$, $p < .001$) and right (MNI: 22, –8, –12; 17 voxels, $t(34) = 3.06$, $p < .005$) amygdala as a function of neu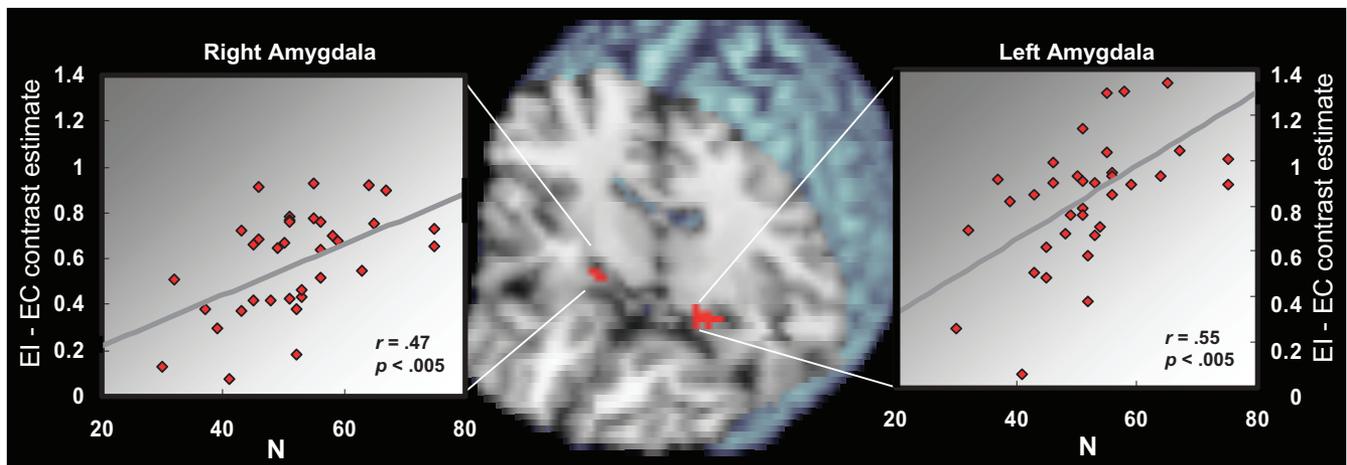roticism in response to emotional conflict: Higher scores of neuroticism were associated with a greater activation within the amygdala in each hemisphere during EI, relative to EC, trials (left: $r = .55$, $p < .001$; right: $r = .47$, $p < .005$).

We confirmed the discriminate validity of the observed association between neuroticism and amygdala activation by testing the specificity of each variable. We found that the correlation with amygdala activation was specific to neuroticism, because other measures of individual differences such as negative mood scores, RT differences between the two conditions, or differences in error rates between the two conditions yielded no significant correlations, even when we used a reduced statistical activation threshold ($p = .01$). Furthermore, we confirmed that the correlation with neuroticism was localized to the amygdala, because neuroticism did not correlate with activation in two neighboring structures, the hippocampus and parahippocampal gyrus, even when we used a reduced statistical activation threshold ($p = .01$).

Subgenual AC Activation as a Function of Neuroticism

Consistent with our predictions, subgenual AC activation during high-conflict (emotionally incongruent, EI) trials, relative to low-conflict (emotionally congruent, EC) trials, correlated significantly with neuroticism. Figure 3 displays the activation within the subgenual AC (MNI: 6, 42, –16; 75 voxels, $t(34) = 3.49$, $p = .001$) as a function of neuroticism in response to emotional conflict: Higher scores of neuroticism were associated with a greater activation within the subgenual AC during EI, relative to EC, trials ($r = .51$, $p < .005$). This cluster extended throughout both the left and right subgenual AC with the peak voxel (6, 42, –16) localized on the right subgenual AC.

As in the previous section, we confirmed the discriminate validity of the observed association between neuroticism and subgenual AC activation by testing the specificity of each variable. We found that the correlation with subgenual AC activation was

Figure 2. Changes in amygdala activation associated with neuroticism in response to emotional conflict. Areas of significant activation (red) are overlaid on a template brain, with areas shown in both the coronal ($y = -8$) and axial ($z = -14$) orientation. The cluster of activation within the left amygdala (–20, –8, –16; $p < .005$) encompassed 34 contiguous voxels, while the cluster of activation in the right amygdala (22, –8, –12; $p < .005$) encompassed 17 contiguous voxels. Data are plotted for both the left and right amygdalae, with the x-axis representing variation in neuroticism and the y-axis representing the signal change difference between emotionally incongruent (EI) and emotionally congruent (EC) trials.
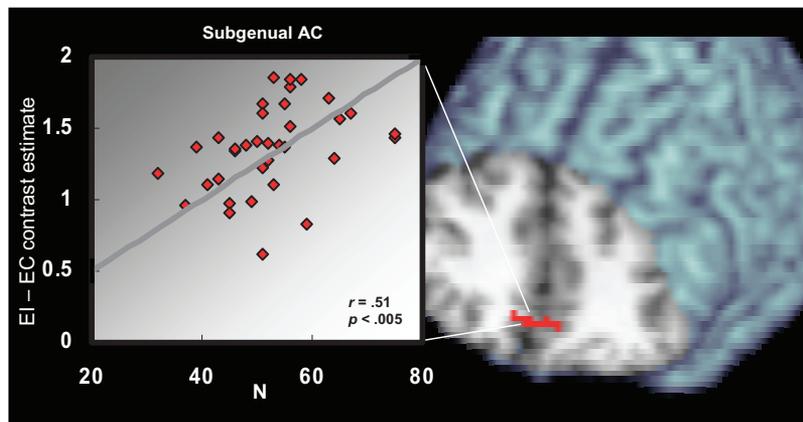
Figure 3. Changes in subgenual anterior cingulate (AC) activation associated with neuroticism in response to emotional conflict. Areas of significant activation (red) are overlaid on a template brain, with areas shown in both the coronal ($y = 38$) and axial ($z = -16$) orientation. The cluster of activation within the subgenual AC (6, 42, -16; $p < .005$) encompassed 75 contiguous voxels. Data are plotted for this cluster of activation with the *x*-axis representing variation in neuroticism and the *y*-axis representing the signal change difference between emotionally incongruent (EI) and emotionally congruent (EC) trials.

specific to neuroticism, because other measures of individual differences such as negative mood scores, RT differences between the two conditions, or differences in error rates between the two conditions yielded no significant correlations, even when we used a reduced statistical activation threshold ($p = .01$). Furthermore, we confirmed that the correlation with neuroticism was localized to the subgenual region of the AC, because neuroticism did not correlate with activation in any other region of the AC, even when we reduced the statistical activation threshold ($p = .01$). The signal change in the subgenual cingulate was significantly positively correlated with signal change in the left ($r = .47$, $p < .005$) but not the right amygdala. A whole-brain analysis using the same threshold identified one other cluster localized in the left superior temporal pole that was associated with neuroticism (MNI: -32, 12, -24; 17 voxels, $t(34) = 2.99$, $p = .003$).

Contribution of Anxious and Depressive Forms of Neuroticism to Amygdala and Subgenual AC Activation

We next identified the number of voxels that were uniquely and significantly associated with anxiety (N1) or depression (N3), respectively. When we correlated activation within the amygdala and subgenual AC with the depressive form of neuroticism (N3), controlling for the anxious form (N1), there were no significant voxels. However, when we correlated activation within the amygdala and subgenual AC with the anxious form of neuroticism (N1), controlling for the depressive form (N3), there were a number of significant voxels within both amygdalae (left: 25 voxels, $t = 2.63$, $p = .001$, right: 16 voxels, $t(33) = 2.38$, $p = .011$) and within the subgenual AC (75 voxels, $t(33) = 3.29$, $p = .001$). The absence of any unique contribution of the depressive form of neuroticism to these clusters could not be attributed to correlations that fell just short of the *a priori* statistical threshold, because there were no significant voxels even at a dramatically reduced statistical activation threshold ($p = .10$).

The second analysis identified the unique contribution of the anxious (N1) and depressive (N3) forms of neuroticism to the clusters identified in the preceding section. This analysis quantified the percentage of variance that was uniquely associated with anxiety (N1) and depression (N3), respectively. This analysis indicated that a greater percentage of variance in the activation of amygdala and subgenual AC was accounted for by the anxious form of neuroticism (N1) than by the depressive form of neuroticism (N3; see Table 1).

Discussion

Advancements in the fields of cognitive and affective neuroscience have facilitated investigations of the neural mechanisms underlying personality and other individual differences (Canli, 2004, 2006; Haas, Omura, Constable, & Canli, 2006b; Hamann & Canli, 2004). Because some personality traits render individuals vulnerable for psychopathology, this approach may help elucidate neurobiological vulnerability markers in otherwise healthy individuals. One candidate personality trait of interest is neuroticism, which renders individuals vulnerable for anxiety and mood disorders (del Barrio, Moreno-Rosset, López-Martínez, & Olmedo,

Table 1

Percentage of Variance Explained by the Anxiety (N1) and Depression (N3) Subscales of the Revised NEO Personality Inventory

Brain region	N1 (Anxiety)	N3 (Depression)
Left amygdala	.17*	.00
Right amygdala	.11*	.00
Subgenual AC	.20**	.00

Note. AC = anterior cingulate.
* $p < .05$. ** $p < .01$.

1997; Bienvenu et al., 2001; Durrett & Trull, 2005). Two structures that have been associated with these disorders are the amygdala and the subgenual AC (Drevets, 2000; Gotlib et al., 2005; Rauch et al., 2003). To our knowledge, there has been no previous demonstration of an association between neuroticism and activation in the amygdala or subgenual AC, although we have previously reported on an association between neuroticism and structural features of the amygdala (Omura, Constable, & Canli, 2005). The results of the current study indicate that activation within the amygdala and the subgenual AC was significantly correlated with neuroticism when participants responded to trials of high, relative to low, emotional conflict.

In light of the presumed link between neuroticism and psychopathology, we also investigated the relative contribution of two subscales of neuroticism thought to be associated with personality characteristics associated with anxiety and depression (Bagby et al., 1997; Bienvenu et al., 2001; Costa & McCrae, 1992a, 1992b; Durrett & Trull, 2005). This analysis demonstrated that the association between neuroticism and individual differences in amygdala and subgenual AC activation was driven by the anxious form of neuroticism but not by the depressive form of neuroticism. Such dissociative analysis strategies may be useful in future studies that seek to investigate how complex personality traits render individuals vulnerable to different psychopathologies.

We used the Word-Face Stroop task (Haas et al., 2006a) in order to assess changes in brain activity associated with emotional conflict. Our previous results demonstrated that the comparison of EI relative to EC trials is associated with increased caudal AC activation, a region previously implicated in conflict monitoring (Botvinick, Cohen, & Carter, 2004). In this sample of subjects, this effect was replicated: EI relative to EC trials were associated with increased caudal AC activation ($p < .005$).

Amygdala Function in Relation to Neuroticism

Changes in amygdala activation have been observed during conditions of negative emotional experience and during emotional arousal (Phelps & LeDoux, 2005). Previous research has also demonstrated that highly neurotic individuals tend to construe events in more negative ways (Gunthert, Cohen, & Armeli, 1999) and are more emotionally reactive (Suls & Martin, 2005) than less neurotic individuals. We offer two interpretations for the correlation between neuroticism and amygdala activation during trials of high emotional conflict. First, this correlation may indicate greater emotional arousal associated with subjective appraisal. Highly neurotic individuals may appraise the inconsistencies of valence during emotionally conflicting trials as more emotionally arousing relative to low neurotics and therefore display increased amygdala activation during this condition. This is consistent with research demonstrating that amygdala activation is associated with higher subjective ratings of arousal (Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000).

A second interpretation is that this correlation may indicate greater vigilance or attention toward emotional conflict. The amygdala is thought to be particularly involved in the modulation of vigilance in the face of threat or fear-related events in the environment (Whalen, 1998). The data presented here suggest that highly neurotic individuals are more vigilant when processing conflict, particularly emotional conflict. This is consistent with results of a recent study demonstrating an association between high neuroticism and increased sensitivity to discrepancy detection during a cognitive oddball task (Eisenberger et al., 2005).

Subgenual AC Function in Relation to Neuroticism

The ventral portions of the AC are implicated in a wide variety of emotional processes (Bush et al., 2000). One process thought to be specific to the subgenual area of the AC is the induction of negative mood states such as sadness (Phan et al., 2004). A propensity toward negative mood states is also a characteristic of highly neurotic individuals (Costa & McCrae, 1980). Indeed, our sample showed a significant correlation between neuroticism and negative mood ($r = .40, p < .05$). However, we found a significant correlation between N and subgenual AC activation even after correcting for mood state. This analysis suggests that subgenual AC response to EI, relative to EC, conditions is a traitlike feature. The behavioral phenotype of this trait could be that highly neurotic individuals are more reactive to emotional conflict than less neurotic individuals are.

The association between mood state and personality trait remains poorly understood. In previous work, we have reported that AC activation to negative, relative to neutral, words in an emotional Stroop task was associated with individual differences in negative mood but not neuroticism (Canli, Amin, Haas, Omura, & Constable, 2004). Our current study, using a different task (in which we collapsed across negative and positive stimuli) and a contrast condition that isolates conflict rather than emotional salience, suggests that subgenual AC activation is associated with individual differences in neuroticism but not negative mood. Thus, it appears that the moderating influence of mood state versus personality trait upon neural systems is highly sensitive to the choice of task paradigms and contrast conditions. This is clearly going to be a rich area for future studies, particularly for studies concerned with the clinical implications of state–trait relations in mood disorders.

An alternative interpretation of the data is that the correlation between N and subgenual AC activation may reflect individual differences in attention. Changes in subgenual AC activation have been observed in response to attentionally demanding visuospatial tasks (Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991; Drevets & Raichle, 1998). Research has also demonstrated that low neurotic individuals display less susceptibility to distraction during a cognitive attention task (Wallace & Newman, 1998). The differences observed in subgenual AC activation in response to emotional conflict may reflect individual differences in the ability to maintain attention when faced with distraction.

Clinical Implications

A number of studies have investigated the relationship between the Five-Factor Model of personality (Eysenck, 1967) and Axis 1 diagnosis and symptomatology (Bienvenu et al., 2001; del Barrio et al., 1997; Durrett & Trull, 2005; Fisher, 1993; Malouff, Thorsteinsson, & Schutte, 2005; Trull & Sher, 1994) and have associated neuroticism with anxiety and mood disorders. However, one conundrum facing clinicians is how to predict whether an individual who scores high in neuroticism is likely to develop psychopathology and which particular form of psychopathology this individual may develop. In the current study, we found that neuroticism correlated positively with activation in structures as-

sociated with anxiety and mood disorders. The observed differences in brain activation may be an underlying neural manifestation of a predisposition of these subjects to experience anxiety or mood disorder related symptoms throughout their lifetime.

Our observation that the anxious form of neuroticism is associated with amygdala and subgenual AC activation during emotional conflict is consistent with the existing clinical literature. Several recent reviews have suggested that anxiety-disordered patients exhibit increased amygdala activation in response to aversive types of conditions, compared with control subjects (Cannistraro & Rauch, 2003; Millan, 2003; Rauch et al., 2003). Other studies have reported that increased subgenual AC activation was associated with anxiety (Drevets & Raichle, 1998; Rauch et al., 1994). High anxiety is also associated with increased attentional bias for affective information (Bar-Haim et al., 2005). In our study, high levels of anxious neuroticism were associated with increased RTs to stimuli that were emotionally incongruent, indicating increased attentional bias, and the anxious form of neuroticism uniquely predicted amygdala and subgenual AC activation during these trials. This analysis was conducted by using a $p < .05$ uncorrected threshold and is therefore not as strong a statistical test as the analysis investigating the relationship between neuroticism alone and emotional conflict response. Further research investigating the relationship between anxiety-related personality measures and attention and vigilance tasks may clarify the nature of the behavioral relationship observed here.

Activation in the amygdala and subgenual AC is not limited to patients with anxiety disorders. Several studies have associated activation in these regions with depressive disorders (Canli et al., 2005; Gotlib et al., 2005). In particular, stimulation of the subgenual AC has been shown to be an effective treatment option for treatment-resistant depression (Mayberg et al., 2005). Major depressive disorder and generalized anxiety disorder are the most common type of anxiety-mood comorbidity (Gorwood, 2004). This task may be particularly applicable in order to assess changes in brain function between individuals diagnosed with anxiety and individuals diagnosed with both anxiety and depression.

We have shown that an emotional conflict task can dissociate neural activation that is unique to the anxious form of neuroticism. A clear prediction from this data is that individuals who show high levels of amygdala and subgenual AC activation during this task are more likely to develop anxiety-related psychopathology in the future than would individuals who show less activation. Future longitudinal studies using larger samples of at-risk individuals will need to test this prediction explicitly. The current paradigm is not sensitive in identifying neural activation that is unique to the depressive form of neuroticism. This may be primarily due to the conflict and attentional bias characteristics of this task. Both characteristics have been suggested to be particularly distinctive to anxiety (Mogg & Bradley, 2005; Shiloh & Melamed, 1988). One aim for future work consists of developing a task whereby a double dissociation can be made between these two forms of neuroticism.

In conclusion, the results presented here contribute to understanding of the neural mechanisms underlying individual differences in affective processing and shed new light on how a personality type such as neuroticism is implicated as a risk factor for anxiety and mood disorders. Future advancements in this type of research may result in the development of more effective preventative and diagnostic techniques.

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Correction to Haas, Omura, Constable, and Canli (2007)

In the article "Emotional Conflict and Neuroticism: Personality-Dependent Activation in the Amygdala and Subgenual Anterior Cingulate," by Brian W. Haas, Kazufumi Omura, R. Todd Constable, and Turhan Canli (*Behavioral Neuroscience,* 2007, Vol. 121, No. 2, pp. 249–256), there was an error in the text of Figure 1 on p. 250. Above the image of the third person, "x?6repetitions" should have appeared as "x 6 repetitions." See corrected figure below.
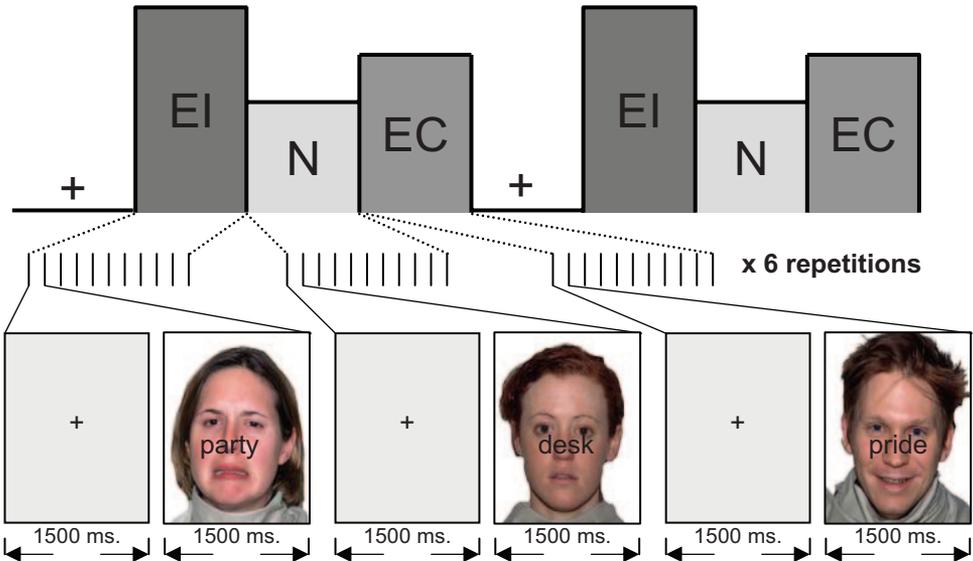

Figure 1. Schematic representation of an experimental paradigm. Stimuli were either emotionally incongruent (EI), neutral (N), or emotionally congruent (EC). Stimuli were presented in a total of 6 blocks of 10 trials for each condition. Each trial consisted of a 1,500 ms presentation of a fixation cross followed by a 1,500 ms presentation of the stimulus. Emotional conflict was assessed by comparing the blood oxygen level-dependent (BOLD) signal obtained during EI trials relative with the BOLD signal obtained during EC trials.