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Emotional-stimulus processing in trait anxiety is modulated by stimulus valence during neuroimaging of a working-memory task

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Recent neuroimaging studies have examined the effects of anxiety on cognitive processing in the presence of emotional distractors. However, when the target stimuli themselves are emotional, it is unclear whether emotion acts as a distracting or enhancing influence. We predicted that anxiety levels would modulate the effect of emotion on neural activity in a valence-specific manner. In the current experiment, we used functional magnetic resonance imaging (fMRI) to examine activity in brain regions associated with cognitive and affective control. Twenty-nine healthy adults, rated for trait anxiety, performed blocks of a 2-back working-memory task (using faces) in which facial expressions were either entirely neutral, mixed neutral and fearful, or mixed neutral and happy. Behavioural results showed no effects of anxiety on either accuracy or response time for any stimulus type. In event-related analyses, dorsal prefrontal regions tended to show reduced activation for emotional faces relative to neutral, suggesting possible neural facilitation, while the amygdala and ventrolateral regions linked to affective-interference showed increased activation to emotional faces. Moreover, in the left inferior frontal gyrus (Brodmann area 45), anxiety discriminated between the response to happy trials and fear trials. The higher the anxiety score, the greater the increase in activation for fear faces versus neutral. By contrast, the *lower* the anxiety score, the greater the increase in activation to happy faces. These results suggest that emotional content in target stimuli can both enhance and interfere with neural processing, and these effects may depend on emotional valence and participants' anxiety levels.

Keywords: Anxiety; Working memory; Cognitive efficiency; Emotional stimuli; Emotional interference; Neural efficiency.

INTRODUCTION

In recent years, cognitive neuroscience researchers have conducted multiple studies investigating the effects of anxiety on working memory (WM) and cognitive control. This research has often focused on cognitive inefficiency and reduced cognitive control in anxious individuals, and has provided

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helpful insights in understanding the cognitive underpinnings of state and trait anxiety (Bishop, Duncan, Brett, & Lawrence, 2004; Derakshan & Eysenck, 2009; Eysenck & Calvo, 1992). However, an important limitation of this research is that it has primarily approached processing differences in anxiety from the standpoint of situations that might engender cognitive conflict. In such studies, participants perform cognitive tasks while trying to ignore sources of negative arousal such as threat-related distractors. In life, there are many situations in which the stimuli that are the focus of attention are themselves a potential source of negative (or positive) affect. However, the influence of anxiety on neural and cognitive processing in such situations has not yet been studied. This is an important omission for anxiety research because in many cognitive domains, emotional stimuli have been shown to facilitate performance rather than impair it. Thus, it is important to determine whether trait anxiety modulates the facilitatory effects of emotion-related processing, and whether any such facilitation depends on the valence of the stimulus. As such, the goal of the current study was to examine the relationship between individual differences in trait anxiety and performance and neural activation on a WM task that involved emotionally valenced target stimuli.

In order to generate predictions about the effects of anxiety on WM for emotionally valenced stimuli, we must first understand how such stimuli normally influence WM performance and associated brain activity. In terms of behaviour, emotional stimuli capture attention more easily than neutral stimuli (Fox, Derakshan, & Shoker, 2008; Fox, Griggs, & Mouchlianitis, 2007; Fox et al., 2000; Koster, Crombez, Van Damme, Verschuere, & De Houwer, 2004; Mogg & Bradley, 1999; Nummenmaa, Hyönä, & Calvo, 2006) and facilitate long-term memory effects (Labar & Cabeza, 2006). In particular, both improved WM accuracy (Jackson, Wolf, Johnston, Raymond, & Linden, 2008; Langeslag, Morgan, Jackson, Linden, & Van Strien, 2009), and reduced proactive interference (Levens & Phelps, 2008) have been reported for emotional versus neutral stimuli. In addition to WM tasks, valence facilitation has also been seen for oddball paradigms (Delplanque, Lavoie, Hot, Silvert, & Sequeira, 2004), attentional blink paradigms (Keil & Ihssen, 2004) and saccade tasks (Kissler & Keil, 2008). These studies suggest that relative to neutral stimuli, WM for emotional stimuli is likely to be enhanced (regardless of anxiety level). This would occur if emotional valence of the stimulus resulted in more robust perceptual processing, which in turn might strengthen maintenance of WM contents over a delay, and enhance recognition processes when a later probe item must be compared to these contents. Thus, in the current study, we might expect to see better accuracy for emotional stimuli, and/or faster response times overall.

In view of these data on enhanced performance for WM with emotional stimuli, such stimuli might also be expected to facilitate (i.e., decrease) neural activity in WM-related brain regions—such as lateral prefrontal cortex (PFC) relative to neutral stimuli. This is because decreased activation in lateral PFC has been found with easier versions of WM tasks. For example, when task difficulty or WM load is manipulated, activation in dorsolateral prefrontal cortex (DLPFC) has been shown to increase or decrease with difficulty level (Braver et al., 1997; Manoach et al., 1997; Rypma, Berger, & D'Esposito, 2002). Thus, enhanced WM performance associated with emotional stimuli (potentially because of enhanced perceptual processing or encoding) might result from easier maintenance of these items in WM and thus reduced activation of lateral PFC regions associated with WM. Surprisingly, however, there have been few neuroimaging studies of WM for emotional stimuli in healthy adults. Moreover, available findings suggest that the nature of emotion-related effects may depend on whether one focuses on ventral- versus dorsolateral-PFC regions, as well as the specific valence involved (positive or negative). For example, several studies have found *increased* activation in ventral-PFC regions for negative as compared to neutral stimuli, with no difference in activation between neutral and positive stimuli (Jackson et al., 2008;

LoPresti et al., 2008) or for emotional compared to scrambled faces (Beneventi, Barndon, Ersland, & Hugdahl, 2007). On the other hand, Perlstein and colleagues (Perlstein, Elbert, & Stenger, 2002) found *decreased* activation in right DLPFC for WM with negative pictures, but increased activation for positive pictures. These findings suggest that dorsal and ventral regions of lateral PFC may both respond to emotional valence, but be differentially sensitive depending on the specific valence and WM load. Ventral-PFC regions have been linked in previous research to the resolution of cognitive or affective interference (D'Esposito, Postle, Jonides, & Smith, 1999b; Dolcos & McCarthy, 2006; Jha, Fabian, & Aguirre, 2004), and thus negative stimuli that are the memoranda in WM tasks may elicit increased activation in these regions even though they also facilitate performance. Dorsal-PFC regions have been linked to the manipulation and maintenance of information in WM, and negative stimuli may reduce activation in such regions, potentially through the enhanced encoding processes discussed above.

These data regarding the influence of emotional WM stimuli on brain activity in non-anxious individuals can help us generate predictions as to what we might expect in high-anxious individuals. People high in anxiety are known to show enhanced behavioural responses toward negative material (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007), and in some instances also attentional avoidance of positive stimuli (Hunt, Keogh, & French, 2006; Keogh, Dillon, Georgiou, & Hunt, 2001). Such attentional biases have been demonstrated using emotional Stroop paradigms, spatial cuing paradigms, and dot-probe tasks, using subliminally or explicitly presented emotionally valenced stimuli. These studies have shown that anxious individuals are often faster to attend to threat-related stimuli and slower to disengage attention from them (Bar-Haim et al., 2007). These threat-bias paradigms typically focus on attentional-selection tasks, and do not directly speak to WM performance in anxiety. However, recent models of attentional bias in anxiety (Bishop, 2007; Derakshan & Eysenck, 2009; Eysenck, Derakshan, Santos, & Calvo, 2007; Pessoa, 2009) suggest that the mechanisms underlying these biases should also be relevant for WM performance. These models propose that both bottom-up and top-down attentional processes may be altered in anxious individuals, with amygdala hyper-responsiveness to threat altering early perceptual processing, and hypoactivity in lateral PFC reducing the efficiency of later attentional-control mechanisms such as inhibition or shifting (Derakshan & Eysenck, 2009; Eysenck et al., 2007). If true, these alterations would be likely to have an impact on the correct functioning of WM processes. An over-response to threat-related stimuli in the amygdala is likely to enhance perceptual processing of such negative stimuli, possibly rendering them easier to encode and maintain in WM after the stimuli are no longer present. If so, then anxious individuals might actually show more benefit in WM performance than low-anxious individuals for negative stimuli. On the other hand, amygdala activity has been suggested to have a "reciprocal relationship" with activity in cognitive-control regions in lateral PFC (Drevets & Raichle, 1998), such that increased activity in the amygdala is associated with decreased activity in lateral PFC. Thus any increased response in the amygdala to negative stimuli might reduce recruitment of cognitive-control regions that have been linked to competent WM maintenance and manipulation (D'Esposito, Postle, Ballard, & Lease, 1999a). Highly anxious individuals might be particularly vulnerable to this effect, as Bishop (2009) has produced evidence that in trait-anxious individuals, recruitment of lateral PFC may be reduced or less efficient even in the absence of external stimuli likely to trigger amygdala responses. Thus, an alternative hypothesis is that high-anxious individuals will experience more disruption in lateral-PFC function during WM tasks when the stimuli to be remembered have negative emotional valence.

In the current study, to test the effects of anxiety on emotional-stimulus processing, we used functional magnetic resonance imaging

(fMRI) to examine brain activity in healthy adults with varying levels of trait anxiety as they performed a WM task. The task was a 2-back task using faces with neutral, negative (fearful), and positive (happy) expressions. The findings reviewed above suggest that in such a task, anxious people might be expected to show enhanced behavioural response to negative stimuli relative to non-anxious people, with little or no enhancement for positive stimuli. Thus we predicted that for behavioural performance, improvements in accuracy (or faster response times) for negative stimuli relative to neutral would be enhanced for participants with higher levels of anxiety.

For neural activity, previous studies suggest we should see increased neural activity in ventrolateral prefrontal cortex (VLPFC) and decreased activation in DLPFC (as suggested by Perlstein et al., 2002) for negative stimuli versus neutral. To examine brain activation for this task, we focused on two sets of cognitive-control regions in the brain, as well as the amygdala. The first set of cognitive control regions is the canonical WM-related network, where increased activation during WM tasks has been repeatedly documented (Owen, McMillan, Laird, & Bullmore, 2005; Wager & Smith, 2003). These regions include areas in lateral prefrontal cortex (dorsal and ventral), dorsal anterior cingulate, and posterior parietal cortex. Our specific predictions were that activation levels might be reduced in DLPFC for the fear-face condition, and this reduction would be enhanced in participants with higher anxiety. By contrast, we predicted that DLPFC activation might increase for happy-face conditions, but potentially less so in individuals with higher anxiety.

The second set of cognitive control regions we investigated included more inferior prefrontal regions that have been linked in previous research to resolution of emotional interference (Bishop et al., 2004; Dolcos & McCarthy, 2006; Kim et al., 2004; Mitchell, Banaji, & Macrae, 2005; Ochsner, Hughes, Robertson, Cooper, & Gabrieli, 2009). In line with the Jackson, LoPresti, Beneventi and Perlstein papers (Beneventi et al., 2007; Jackson et al., 2008; LoPresti et al., 2008; Perlstein et al., 2002), we predicted that regions in inferior PFC would increase activation in the fear-face condition relative to neutral, and that in regions where increases were seen, the increases would be greater for those with higher levels of trait anxiety. We also predicted that theses increases would not be seen for the happy-face condition. Increases in the amygdala for fearful faces were also predicted to be greater in those with higher anxiety.

METHOD

Participants

Participants (Table 1) were recruited through the Conte Center for the Neuroscience of Mental Disorders (CCNMD) at Washington University in St Louis, and included 33 healthy participants. Exclusion criteria included: (a) substance abuse or dependence within the past three months; (b) the presence of any neurological, psychiatric or other

Table 1. *Sample demographics*

Characteristics	
Age (years)	35.5 (10.9)
Gender	
Female	10 (34.5%)
Male	19 (65.5%)
Ethnicity	
African American	14 (48.3%)
Caucasian	15 (51.7%)
Education (years)	15.6 (4.35)
Mood & Personality	
BDI	4.0 (4.3)
BAI	25.4 (3.8)
BIS	18.6 (4.0)
EPQ-Neuroticism	5.9 (4.1)
MTQ-MA	56.7 (12.1)
Overall Anxiety (mean *z*-score)	0.0 (2.14)
Overall Anxiety (*z*-score range)	−3.6 to +5.3

Notes: $N = 29$. Results given as Mean (*SD*) or *n* (%). BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; Behavioural Inhibition System scale; EPQ = Eysenck Personality Questionnaire; MTQ-MA = Motivational Anxiety subscale of the Motivational Trait Questionnaire.

medical disorder; (c) present or past head injury with documented neurological sequelae, and/or causing loss of consciousness; (d) mental retardation (meeting DSM-IV criteria for mild severity or greater); (e) presence of any first-degree family member with a lifetime history of any DSM-IV disorder; and (f) pregnancy, history of claustrophobia, any metallic object in the body, history of heart rhythm abnormalities or presence of a heart pacemaker. Of the original 33 participants, four were dropped because of scanner or computer failure ($n = 3$) or the presence of significant depression (Beck Depression Inventory score > 21, $n = 1$), leaving a total of 29 participants in the study. The resulting sample included 19 males and 10 females, with mean age of 35.5 years ($SD = 10.9$, range = 19–52) and mean education of 15.6 years ($SD = 4.4$, range = 12–32). Racial composition was 14 African Americans and 15 Caucasian.

Task and materials

Anxiety, mood, and cognitive assessments. Four anxiety assessments and one depression scale were administered (see Table 1). Measures related to anxiety included the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988), the Behavioural Inhibition System scale (BIS; Carver & White, 1994), the Neuroticism subscale of the Eysenck Personality Questionnaire (EPQ-Neuroticism; Eysenck & Eysenck, 1991), and the Motivational Anxiety subscale of the Motivational Trait Questionnaire (MTQ-MA; Kanfer & Heggestad, 1999). These measures were combined to arrive at a single measure of trait anxiety. Mood state for the prior one week was assessed using the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Finally, basic cognitive abilities were measured using the vocabulary and matrix reasoning subscales of the Wechsler Adult Intelligence Scales III (WAIS-III; Wechsler, 1997).

Working-memory task. While in the MRI scanner, participants performed six runs of a "2-back" version of the "n-back" task: three consecutive runs using face stimuli and three using word stimuli. The current study focuses on the face stimuli as these represent socially salient stimuli that are known to elicit robust amygdala responses, especially in anxious individuals (Somerville, Kim, Johnstone, Alexander, & Whalen, 2004; Wright & Liu, 2006). The order of the verbal versus non-verbal runs was counterbalanced. A single run consisted of four initial fixation trials (not analysed), three "task" blocks, and four "fixation" blocks in alternating order. Stimuli for the three task blocks were manipulated to create three valence conditions: (1) all neutral faces (neutral block); (2) mixed fearful and neutral faces (fear block); and (3) mixed happy and neutral faces (happy block). (This arrangement of trials for the blocks allowed us to conduct both blocked and event-related analyses.) Stimuli appeared one at a time on a screen projected over the participant's head, and for each face, participants were instructed to press a "target" button if the current face was the same as the face seen two trials previously, or a "non-target" button if it was any other face. "Target" faces always matched the face two back in both identity and emotional expression; no faces were ever presented with more than a single valence with the same block. All face stimuli were matched in lighting, location, distance, exposure, and arousal ratings (by valence). The faces used as stimuli were colour photos of people showing three different emotional expressions (fearful, happy, neutral). The photos were taken from the set developed by Gur and colleagues (Gur et al., 2002), which were generated and tested for normative response to each expression.

Each task block consisted of 32 trials, including 16 neutral faces and 16 emotional faces with the relevant valence (neutral, fearful, or happy). Target and non-target responses were distributed evenly over both the neutral and the emotional faces. Each face was displayed for 2.5 seconds (regardless of the participant's response time), and was followed by a fixed 500 ms inter-stimulus interval. Thus trials were not jittered, but the critical emotional faces were pseudo-randomly interleaved with the neutral faces so as to ensure

disambiguation using rapid event-related analysis. During fixation blocks (30 trials at 3.0 seconds each, a cross-hair appeared continuously, and subjects were told to fixate on it. Visual stimuli were generated by a G3 Macintosh computer and presented using PsyScope experimental software (Cohen, MacWhinney, Flatt, & Provost, 1993). Images were projected onto a computer screen behind the subject's head within the imaging chamber. Participants saw the screen through a mirror positioned approximately 8 cm above their face. A fibre-optic, light-sensitive key press interfaced with the PsyScope button box was used to record subject's behavioural responses.

Procedure

fMRI imaging and analysis

Image acquisition. Functional scanning was performed on a 3T Siemens Allegra head-dedicated system at the Research Imaging Center of the Mallinckrodt Institute of Radiology at the Washington University Medical School. First, a low-resolution 3D sagittal T1-weighted MP-RAGE acquisition image was obtained (TE = 2.9 ms, TR = 6.6 ms, flip angle = 8°, 96 × 128 acquisition matrix, 1 acquisition, 80 slices, 2 × 2.67 × 2 mm voxels). This MP-RAGE was then warped to Talairach space. A T2 image was subsequently acquired in the same position as the functional images (TE = 96 ms, TR = 5 s, 189 × 256 acquisition matrix, 48 slices, 1.02 × 1 × 3 mm voxels), and used as a bridge to facilitate the registration of the T1-weighted images acquired during the structural imaging session and the functional (T2*-weighted) images. The slice locations for functional images were placed based on the results of the computerised slice pre-registration. The functional images were collected in runs using an asymmetric spin-echo echo-planar sequence sensitive to blood oxygenation level-dependent (BOLD) contrast (T2*) (TR = 3000 ms, TE = 25 ms, FOV = 205 mm, flip = 90°). During each functional run, sets of 40 contiguous axial images with isotropic voxels (3.2 mm^3) were acquired parallel to the anterior–posterior commissure plane.

fMRI processing. MR data were reconstructed into images, and normalised across runs by scaling whole-brain signal intensity to a fixed value (mode of 1000), and removing the linear slope on a voxel-by-voxel basis to counteract effects of drift. The MR data were aligned to correct for head motion using 6-parameter rigid-body rotation and translation correction algorithms interference (Friston, Williams, Howard, Frackowiak, & Turner, 1996; Snyder, 1996; Woods, Cherry, & Mazziotta, 1992). MR data was registered to a common space (Talairach & Tournoux, 1988) using 12-parameter linear (affine) transformations of the participant's average MP-RAGE structural images into a target image in Talairach atlas space, and then using the T2 images to align the T2* and T1 images. The fMRI images were spatially smoothed with a 9 mm FWHM Gaussian kernel.

Statistical analysis. We used in-house software to carry out *t*-tests, analyses of variance (ANOVAs), and correlations to identify regions significantly activated for various conditions. For each participant, a General Linear Model (GLM) was defined to estimate activation magnitudes for blocked and event-related effects. Blocked GLMs allowed for the separate estimation of neural activation for neutral, happy, and fear blocks. Event-related GLMs were defined to model different trial types: fear and neutral trials from the fear blocks, and happy and neutral trials from the positive blocks. All individual-subject estimates were then entered into second-level analyses that treated subject as a random factor. We chose to conduct both blocked and event-related analyses because of their varying sensitivity to different types of effects. In particular block-related analyses are more likely to detect context effects in a block (for example, global responses to emotional stimuli) that are not detectable using event-related analyses. By contrast, the event-related results were expected to give more precise picture of trial-specific effects of emotion.

We used an a priori region of interest (ROI) approach to test our hypotheses. To identify areas of significant activation, we imposed the follow-

ing restrictions: a voxel-wise threshold criterion of $p < .005$, a region-wise criterion of $p < .01$, and a required cluster size of at least 9 contiguous voxels. Additionally, all voxels thus identified had to be located somewhere within our a priori ROI masks, as described below. For *t*-tests and ANOVAs, voxels were considered significant if they met the above criteria for the given statistic, as well as significant task-related activity in at least one emotional condition. For correlational analyses, voxels were required to meet the thresholds described but not the task-related activity.

To look for non-predicted effects in regions outside our a priori ROI masks, we conducted a whole-brain three-way ANOVA. The ANOVA results were thresholded to obtain a whole brain false positive rate of .05 ($p < .0001$ and a minimum-cluster extent of 30 or more contiguous voxels). As this ANOVA revealed no significant anxiety-related effects, the whole-brain analysis was not pursued further.

A priori ROI selection. For the current study, we used two sets of a priori-defined regions (see Figure 1), which were combined into two masks defining what voxels were "eligible" to show significant effects. Thus, we report any regions showing significant activity for the contrast of interest (as described above) that also fell within one of these masks. The first mask consisted of regions linked to WM task performance in previous studies. These included regions in lateral PFC, dorsal anterior cingulate cortex (DACC) and posterior parietal regions. We created this mask using neuroanatomical co-ordinates described in a meta-analysis of n-back neuroimaging results (Owen et al., 2005), as well as those for working-memory tasks more generally (Wager & Smith, 2003). Our focus for this network was mainly on lateral prefrontal regions previously linked to attentional control and interference resolution. However, we chose to include other cortical WM regions previously shown to support WM processes (D'Esposito et al., 1999a; Wager & Smith, 2003), since neural alterations (facilitation or disruption) could potentially influence regions in this dorsal control network collectively. We omitted co-ordinates for subcortical locations. For the chosen regions, we created spherical ROIs of 20 mm in diameter. The second mask consisted of regions linked to emotional-interference resolution (in bilateral inferior PFC and the insula).

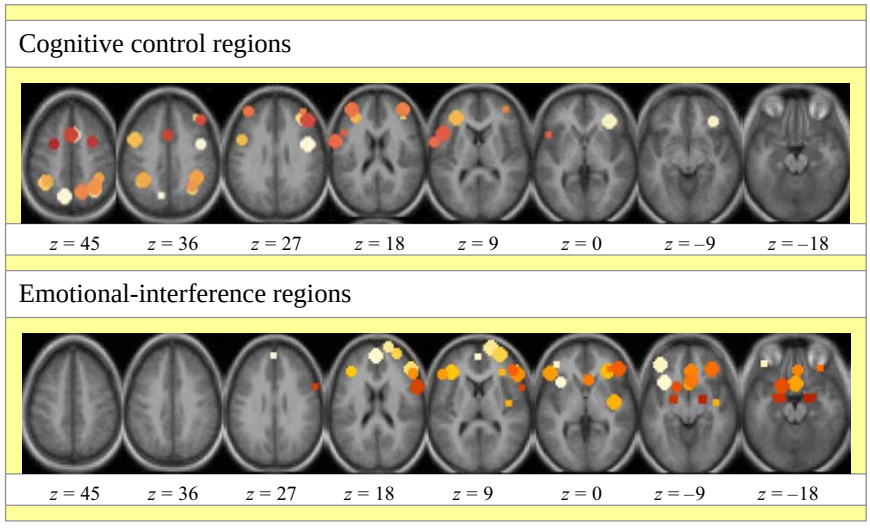

Figure 1. *A priori region-of-interest masks for: (top) working-memory/cognitive-control regions and (bottom) emotional-interference regions. [To view this figure in colour, please visit the online version of this journal].*

In addition we were interested in activity in the amygdala, and thus included right and left amygdala. To generate this mask, we again created 20 mm spheres around peak locations identified in several studies of emotional-interference processing (Bishop et al., 2004; Dolcos & McCarthy, 2006; Kim et al., 2004; Mitchell et al., 2005; Ochsner et al., 2009). For the amygdala ROIs, we hand-drew the mask using Talairach co-ordinate maps (Talairach & Tournoux, 1988). It is important to note that the two ROI masks thus generated contained some overlapping voxels—mostly in the inferior prefrontal gyrus (PFG), since previous WM studies frequently found significant activity in regions linked to emotional-interference processing (see appendix, Tables A1 and A2).

Neuroimaging data analysis

For both blocked and event-related analysis, we were primarily interested in anxiety-related effects of emotional stimuli. Thus, we used two methods to identify brain regions the activity of which was associated with anxiety level. First, we identified regions in the whole sample showing the effects described below and asked whether activity in these regions correlated with anxiety level. Second, we conducted whole-brain correlations between anxiety scores and the contrasts described below to find brain regions the activity of which might be associated with anxiety even when group-level effects were not present.

Since we were interested in the influence of anxiety on emotion-related effects in our WM task, we focused on the contrast of fear stimuli versus neutral, and secondarily on the contrast of happy stimuli versus neutral stimuli. The blocked analysis used contrasts comparing block activation (i.e., fear-block activation vs. the neutral block; happy block vs. the neutral block), while the event-related analysis contrasted emotional-face versus neutral-face trials (e.g., fear-face trials minus neutral-face trials from the fear block). Moreover, because highly anxious individuals sometimes show effects of both comparisons (oversensitivity to negative stimuli and reduced sensitivity to positive stimuli), we also examined the difference between fear and happy contrasts directly. For event-related analysis, the resulting contrast (fear-minus-neutral contrast minus the happy-minus-neutral contrast) allowed us to look for regions showing interaction effects that depended on both valence (fear, happy) and emotionality (emotional, neutral).

RESULTS

Behavioural results

Anxiety and depression measures. The BAI scores for all 29 participants had a mean value of $M = 24.5$, $SD = 3.8$, with a range from 21 to 33. The BIS scores had a mean value of $M = 18.6$, $SD = 4.0$, with a range of 11 to 28. The Neuroticism scores had a mean value of $M = 5.9$, $SD = 4.1$, with a range of 1 to 14. The Motivational Anxiety (MTQ-MA) scores had a mean value of $M = 56.7$, $SD = 12.1$, with a range of 36 to 77. To generate a single measure of anxiety, we *z*-scored each measure and computed a principal component analysis. From this analysis the eigenvector for the first principal component, which captures the greatest portion of the variance for a set of measures, was used to weight scores on individual measures as follows: Anxiety index = .616 (Z-BAI) + .813 (Z-BIS) + .879 (Z-MTQ-MA) + .657 (Z-Neuro). The range of composite anxiety scores for all 29 participants was – 3.61 to 5.31, with a mean of 0 and *SD* of 2.14. Participants were excluded ($n = 1$) if their BDI depression scores exceeded 25, and remaining BDI scores ($M = 4.0$, $SD = 4.3$) did not correlate significantly with either the individual or composite anxiety scores. (See Table 1.)

Task performance: Block-type effects. Percent accuracy and reaction time (RT) data were analysed using an ANCOVA, with Block Valence (fear, neutral, happy) as a within-subjects factor, and Anxiety Scores as a continuous covariate. See Figure 2, which depicts participants divided into high- and low-anxiety groupings (simple median split). All RT data are reported for correct

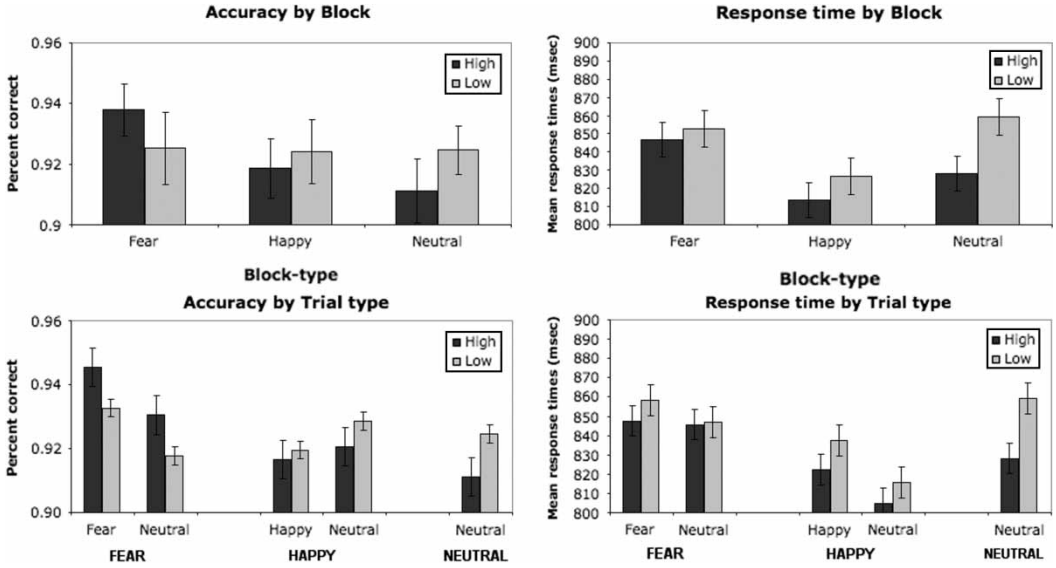

Figure 2. *Behavioural performance (mean percent correct and response time) for the three emotion conditions: (a) Performance by Block; (b) Performance by Trial-type within the Fear and Happy blocks. For the ANCOVA, anxiety was a continuously varying measure, but for visualisation purposes, effects are depicted for participants divided into high- and low-anxiety subgroups (median split). Dark grey: high anxiety scores, Light grey: low anxiety. Error bars indicate standard errors of the mean.*

trials only. For accuracy, the main effect of Block Valence was not significant, $F(2, 26) = 2.55$, $p > .05$, although there was a trend for fear-block performance to be more accurate than neutral-block trials, $F(2, 27) = 3.16$, $p = .087$. Anxiety Score did not modulate these accuracy effects ($F < 1.0$), nor did it correlate with difference scores (fear-block or happy-block accuracy minus neutral-block accuracy). Mean RTs did, however, show a main effect of Block Valence, $F(2, 26) = 6.88$, $p < .01$, with RTs for the happy block significantly faster than for the neutral blocks, $F(1, 27) = 6.01$, $p < .05$, or for the fear blocks, $F(1, 27) = 11.38$, $p < .01$. Anxiety Scores again did not modulate these effects or RT difference scores (fear-block or happy-block RT minus neutral-block RT).

Task performance: Trial-type effects. We used a three-factor ANCOVA (see Figure 2), with Block Valence (fear vs. happy) and Emotion-Presence (emotional vs. neutral stimuli) as within-subject factors, and Anxiety Scores as a continuous covariate. For accuracy, there was again a trend for a main effect of Block Valence, $F(1, 27) = 3.89$, $p = .059$, with fear-block trials more accurate than happy-block trials. There was also a trend for a Block Valence × Emotion-Presence interaction, $F(1, 27) = 3.44$, $p = .075$, such that in the fear blocks, the emotional-stimuli trials were more accurate than the neutral-stimuli trials, while in the happy blocks, the emotional-stimuli trials were *less* accurate than the neutral trials. Anxiety Score did not modulate any of these accuracy effects, nor did it correlate with difference scores (fear trials minus neutral trials in the fear block, or corresponding differences in the happy block). For mean RTs, however, there was a significant main effect of Block Valence, $F(1, 27) = 11.38$, $p < .01$, with times for the happy-block trials significantly faster than for the fear-block trials. There was also a significant main effect of Emotion-Presence, $F(1, 27) = 8.88$, $p < .01$, with emotional trials being *slower* than neutral trials. (This contrasts with the block-level comparison, where

happy-block RTs are faster than neutral-block RTs.) There were no significant interactions. Anxiety scores again did not modulate any of these RT effects, or the corresponding RT difference scores (fear trials minus neutral trials within the fear block, or happy trials minus neutral trials within the happy block).

Neuroimaging results

Blocked analysis. Results of the blocked analyses are summarised in Table 2.

Emotion-related contrasts. A *t*-test of fear- versus neutral-task blocks revealed a significant difference in activation only in the right amygdala. Contrary to expectations, BOLD activity was *lower* in the fear block than in the neutral one. Anxiety did not modulate this effect. A *t*-test of positive- versus neutral-block activation found several regions where BOLD activity was lower in the happy blocks than in the neutral ones, and no regions showing the reverse effect. The regions found included cognitive-control regions—bilateral DLPFC, left dorsal anterior cingulate and left supramarginal gyrus—as well as emotional-interference regions—bilateral inferior frontal gyrus (IFG). Anxiety did not modulate these effects. A *t*-test of valence effects (fear-block activation minus happy-block activation) revealed several regions lateralised to the left hemisphere. In all of these regions—superior and inferior

Table 2. *Significant effects for blocked analysis*

Region	*Brodmann*	*X*	*Y*	*Z*	*Voxs*	*Mask*	*Z-value*	*p-value*	*Effect*	*Anx-correl*
Negative minus neutral blocks										
R-Amyg		21	−10	−10	15	EI	−2.97	.003	Neutr > Neg	*ns*
Positive minus neutral blocks										
L-IFG/MFG	45,46	−37	25	21	29	CC	−3.75	.000	Neutr > Pos	*ns*
R-MFG	9,46	43	30	26	20	CC	−3.52	.000	Neutr > Pos	*ns*
L-IFG/Precentral Gyrus	6,44	−51	2	21	64	CC	−3.37	.001	Neutr > Pos	*ns*
R-IFG/Ant. Insula	47	43	15	0	21	CC	−3.35	.001	Neutr > Pos	*ns*
R-Precentral Gyrus	4	41	−4	46	44	CC	−3.20	.001	Neutr > Pos	*ns*
L-Ant. Insula		−34	13	3	95	CC	−2.71	.007	Neutr > Pos	*ns*
L-IPL	40	−36	−49	44	91	CC	−2.65	.008	Neutr > Pos	*ns*
L-MFG/Precentral Gyrus	4,6	−36	−5	56	14	CC	−2.63	.008	Neutr > Pos	*ns*
L-Medial PFC/ACC	6	−2	10	51	11	CC	−2.63	.009	Neutr > Pos	*ns*
R-IFG	44,45	34	10	21	37	CC	−2.94	.003	Neutr > Pos	*ns*
R-Precentral Gyrus	6,44	58	7	10	29	EI	−2.66	.008	Neutr > Pos	*ns*
Negative minus positive blocks										
L-Ant. Insula		−27	17	4	93	CC	3.01	.003	Neg > Pos	*ns*
L-IFG (Broca)	44	−46	6	33	185	CC	2.72	.007	Neg > Pos	*ns*
L-MFG/SFG	10	−30	49	23	12	CC	2.59	.010	Neg > Pos	*ns*
L-Precentral Gyrus	6	−62	1	21	15	CC	3.27	.001	Neg > Pos	*ns*
L-IPL	40	−38	−50	40	91	CC	2.75	.006	Neg > Pos	*ns*
L-Ant. Insula		−29	16	−1	21	EI	2.86	.004	Neg > Pos	*ns*
Correlations of anxiety with negative—minus—neutral contrast										
R-MFG	6	31	11	53	19	CC	−2.92	.004		$r = -.5238$
L-Amyg		−18	−5	−12	21	EI	4.03	.000		$r = .6768$
Correlations of anxiety with positive—minus—neutral contrast										
L-MFG/Precentral Gyrus	6	−34	−6	58	11	CC	−2.82	.005		$r = -.5097$
L-Amyg		−16	−1	−13	28	EI	3.65	.000		$r = .6287$

Notes: Mask types are denoted as "CC" (working memory mask) and "EI" (emotional interference mask). L = Left; R = Right; Amyg = Amygdala; IFG = Inferior Frontal Gyrus; MFG = Middle Frontal Gyrus; SFG = Superior Frontal Gyrus; ACC = Anterior Cingulate Cortex; IPL = Inferior Parietal Lobe; Ant. = Anterior.

frontal cortex, anterior insula, and inferior parietal lobe (IPL)—neural activity was significantly greater during the fear blocks than the happy blocks. Anxiety did not modulate these effects.

Correlations with anxiety. We correlated anxiety scores with contrasts of (1) fear versus neutral, (2) happy versus neutral, or (3) fear versus happy block activation. For the fear-versus-neutral contrast, two regions were found where the activity difference between fear and neutral blocks was significantly associated with anxiety: the left amygdala, and right superior frontal gyrus (SFG). The change in activity between neutral and fear blocks in left amygdala was positively correlated with anxiety ($r = .68$, $p < .001$). This relationship was largely driven by the fact that anxiety was positively related to fear-block activation ($r = .66$, $p < .001$) in the amygdala, while anxiety showed no association with neutral-block activity. In the right SFG, anxiety correlated negatively with fear-minus-neutral contrast activity ($r = -.52$, $p < .01$). Specifically, anxiety showed a trend toward a negative association with fear-block activity in right SFG ($r = -.36$, $p = .058$), but no relationship with neutral block activity in this region.

For the contrast of happy versus neutral blocks, anxiety was again related to activity in a slightly more inferior region of the left amygdala. Activity change in this region was positively correlated with anxiety ($r = .57$, $p < .01$), but this time the relationship was driven by a significant negative relationship between anxiety and neutral block activity ($r = -.54$, $p < .01$). No association between anxiety and happy-block activity was found.

For the valence contrast (fear-versus-happy blocks), we found two regions whose activity was significantly associated with anxiety. In the left amygdala, anxiety was positively correlated with the fear-versus-happy contrast ($r = .63$, $p < .001$). The higher the anxiety score, the greater the activity for the fear block relative to the happy block. This relationship was driven by the fact that anxiety was significantly positively related to the fear-block activity level ($r = .53$, $p < .01$), but not to activity in the happy block. In the second region—left middle frontal gyrus (MFG)—anxiety was negatively related to the difference between fear and happy blocks ($r = -.51$, $p < .01$), with higher anxiety scores associated with a smaller increase in fear-block activation relative to happy. There was a trend toward a significant positive correlation between anxiety and happy-block activity ($r = .35$, $p = .060$), but no relationship to activity in the fear block.

Event-related analysis. Results for event-related analyses are summarised in Table 3.

Fear versus neutral trials. Neural response to fear faces versus neutral faces was found in a number of cognitive-control regions—bilateral MFG, bilateral IPL, right dorsal ACC, supplementary motor area (SMA) and precuneus—all of which showed reduced activity in fear trials relative to neutral trials. By contrast, significant increases in activity in response to fear faces (relative to neutral) were found in the left amygdala and several emotional-interference regions (bilateral IFG, and polar MFG). Activity in these regions was not significantly modulated by anxiety.

Happy versus neutral trials. For happy-face trials relative to neutral, reductions in neural activity were found in the dorsal cingulate, left IPL, and the right precuneus. In contrast, an emotional-interference region overlapping the left IFG and left insula showed the reverse pattern: significantly increased activity to happy faces, relative to neutral. Moreover, this pattern was modulated by anxiety: the higher the anxiety score, the smaller the increase in neural activity to happy faces, relative to neutral ($r = -.46$, $p < .05$).

Main effect of emotion. To examine the effects of emotion with greater power, we examined activation for all emotional-face trials (average of fear faces and happy faces) as compared to activation for neutral trials from both fear and happy blocks. Working memory-related regions (bilateral DLPFC, bilateral IPL, bilateral precuneus and

dorsal anterior cingulate) were all less active for emotional trials than for neutral trials. By contrast, the left amygdala and emotional-interference regions (bilateral IFG and left polar SFG) all showed greater activation in response to emotional trials than to neutral trials. Anxiety scores were positively related to amygdala activity for this contrast (greater activation for higher anxiety in response to emotional trials relative to neutral; $r = .38$, $p < .05$).

Table 3. *Significant effects for event-related analysis*

Region	*Brodmann*	*X*	*Y*	*Z*	*Voxs*	*Mask*	*Z-value*	*p-value*	*Effect*	*Anx–correl*
Negative minus neutral trials										
R-MFG	46	33	34	28	68	CC	−2.81	.005	Neutr > Neg	*ns*
D-ACC	32	6	28	33	27	CC	−2.76	.006	Neutr > Neg	*ns*
R-IPL	40	42	−51	41	88	CC	−3.23	.001	Neutr > Neg	*ns*
PCU	7	0	−70	47	130	CC	−2.83	.005	Neutr > Neg	*ns*
Mid-Cing	6	23	−2	56	89	CC	−3.11	.002	Neutr > Neg	*ns*
Mid-Cing	6	−29	−4	57	71	CC	−3.10	.002	Neutr > Neg	*ns*
L-IFG/MFG	45,46	−29	30	18	32	CC	−3.00	.003	Neutr > Neg	*ns*
L-IFG/MFG	45,46	−30	30	15	9	EI	−2.61	.009	Neutr > Neg	*ns*
R-IFG	47	36	29	−11	9	EI	3.20	.001	Neg > Neutr	*ns*
L-IFG	47,10	−47	30	−1	53	EI	2.99	.003	Neg > Neutr	*ns*
R-IFG	46	49	32	12	19	EI	2.69	.007	Neg > Neutr	*ns*
L-Medial SFG	10	−3	57	18	27	EI	2.84	.005	Neg > Neutr	*ns*
L-Amyg		−22	−7	−14	63	EI	4.14	.000	Neg > Neutr	*ns*
Positive minus neutral trials										
D-ACC	6,32	0	11	43	151	CC	−3.19	.001	Neutr > Pos	*ns*
L-IPL	40	−38	−51	43	108	CC	−2.75	.006	Neutr > Pos	*ns*
R-PCU	7	14	−65	46	271	CC	−3.95	.000	Neutr > Pos	*ns*
R-PCU	7	12	−58	53	14	CC	−2.72	.007	Neutr > Pos	*ns*
L-IFG/Insula	45	−29	26	1	11	CC	2.61	.009	Pos > Neutr	$r = -.46^*$
L-IFG/Insula	47	−32	22	−7	16	EI	2.71	.007	Pos > Neutr	*ns*
Emotional trials minus neutral trials										
R-PCU	7	4	−69	52	125	CC	−4.57	.000	Neut > Emot	*ns*
R-IPL	40	45	−51	56	51	CC	−3.58	.000	Neut > Emot	*ns*
R-PCU	7	32	−75	43	60	CC	−2.99	.003	Neut > Emot	*ns*
L-PCU/Cuneus	7,19	−15	−77	41	52	CC	−3.79	.000	Neut > Emot	*ns*
R-DACC	32	3	29	31	50	CC	−2.65	.008	Neut > Emot	*ns*
R-MFG	9	32	31	37	45	CC	−2.65	.008	Neut > Emot	*ns*
L-IPL	40	−40	−50	47	155	CC	−3.00	.003	Neut > Emot	*ns*
R-MFG/Precentral G	6	40	−6	56	49	CC	−2.81	.005	Neut > Emot	*ns*
L-MFG	9,46	−42	41	27	11	CC	−2.73	.006	Neut > Emot	*ns*
L-MFG	6	−33	−9	61	40	CC	−2.59	.010	Neut > Emot	*ns*
L-SFG	9	−12	55	27	27	EI	2.82	.005	Emot greater	*ns*
L-IFG	47	−34	32	−6	30	EI	3.77	.000	Emot greater	*ns*
R-IFG	47	42	25	−15	10	EI	3.62	.000	Emot greater	*ns*
L-Amyg		−30	−5	−19	29	EI	3.44	.001	Emot greater	$r = .38^*$
Negative response minus positive response (negative–minus–neutral) minus (positive–minus–neutral)										
L-MFG	46,10	−28	31	18	19	CC	2.57	.010	(Fear-Neu) > (Pos-Neu)	*ns*
R-IFG	46	48	33	11	16	EI	2.82	.005	(Fear-Neu) > (Pos-Neu)	*ns*
L-Amyg		−13	1	−15	9	EI	3.06	.002	(Fear-Neu) > (Pos-Neu)	$r = .457^*$
Correlation of anxiety with (negative–minus–neutral) minus (positive–minus–neutral) contrast										
L-IFG/MFG	45,46	−43	22	21	40	CC	3.39	.001		$r = .593$

Notes: Mask types are denoted as "CC" (working memory mask) and "EI" (emotional interference mask). L = Left; R = Right; D=Dorsal; Amyg = Amygdala; IFG = Inferior Frontal Gyrus; MFG = Middle Frontal Gyrus; SFG = Superior Frontal Gyrus; ACC = Anterior Cingulate Cortex; IPL = Inferior Parietal Lobe; PCU = Precuneus; Cing = Cingulate Gyrus.

Interaction of valence and emotion. The interaction contrast (fear-minus-neutral contrast minus the positive-minus-neutral contrast) revealed three regions showing significant effects. In the left DLPFC, there was reduced activation for fear faces relative to neutral, while in the happy block, no difference was found between happy and neutral faces. Activation in this region showed a trend toward modulation by anxiety, $F(1, 27) = 3.54$, $p = .071$ ($r = .34$). The higher the anxiety score, the greater the difference between fear-related reductions and happy-related ones. A region in right inferior PFC showed a different pattern: significantly *increased* activation for fear faces relative to neutral, while the happy block again showed no difference between happy and neutral faces (anxiety did not modulate this relationship). Finally, in the left amygdala, fear-face trials showed significantly increased activation relative to neutral, while activation for happy faces was significantly reduced, relative to neutral. This interaction did depend on anxiety scores: the higher the anxiety score, the larger the overall difference between emotion-related activation changes for fear versus happy trials ($r = .46$, $p < .05$).

Correlational analyses. Whole-brain correlations were conducted to find regions where the effects of our contrasts (fear-minus-neutral, happy-minus-neutral, and the difference between the two) were significantly associated with anxiety regardless of whether there were group level differences between conditions. Neither the activation found for fear-minus-neutral faces nor the activation for happy-minus-neutral faces was related to anxiety scores at the threshold used (see methods). Notably, however, the third contrast (expressing the interaction of valence × trial-type) showed a strong association with anxiety in a single region that overlapped the left middle and left inferior frontal gyri (Figure 3). This region showed an interaction of all three factors such that the higher the anxiety score, the greater the difference between response patterns in the fear block versus the happy block. Specifically, higher anxiety was associated with increased activation to fear faces (relative to neutral), and reduced activation to happy faces. By contrast, people with lower anxiety showed increased response to happy-face trials (relative to neutral) and little change in activation to fear-faces. Figure 3 depicts this interaction using a three-way split of participants into subgroups with anxiety scores within or beyond one standard deviation from the mean.

In order to characterise this interaction in more detail, we examined the correlation between anxiety scores and the main effect of trial-type for each valence separately. Anxiety showed a significant inverse relationship with the contrast of happy-minus-neutral faces, $r = -.47$, $p < .05$, while there was only a trend toward a significant relationship between anxiety and the fear-minus-neutral contrast, $r = .31$, $p = .104$. For the three-way split of participants into subgroups (mentioned above; see Figure 3), we found that for the lowest-anxiety group, the valence × trial-type interaction showed a trend toward significance ($p = .084$), as did the paired t-test comparing activation levels for happy-minus-neutral trials ($p = .059$), while the fear-minus-neutral comparison was non-significant. For the middle-anxiety group, the valence × trial-type interaction was non-significant.

By contrast, for the highest anxiety group, the valence × trial-type interaction was significant ($p < .01$), with the simple effect of fear-minus-neutral trials being significant ($p < .01$), but not happy-minus-neutral trials. Thus the three-way interaction (valence × trial-type × anxiety) in left IFG appears to be driven by both the relationship of anxiety with the happy-face condition and its relationship with the fear-face condition.

Influence of other factors. We examined the possibility that other factors rather than anxiety might explain the results described here. All of the reported anxiety-related effects (in particular the three-way interaction found for activation in left IFG) remained significant when controlling for depression scores. They also remained significant (with the exception of one effect reduced to trend level) after controlling for intelligence-related cognitive measures such as the WAIS-III vocabulary or

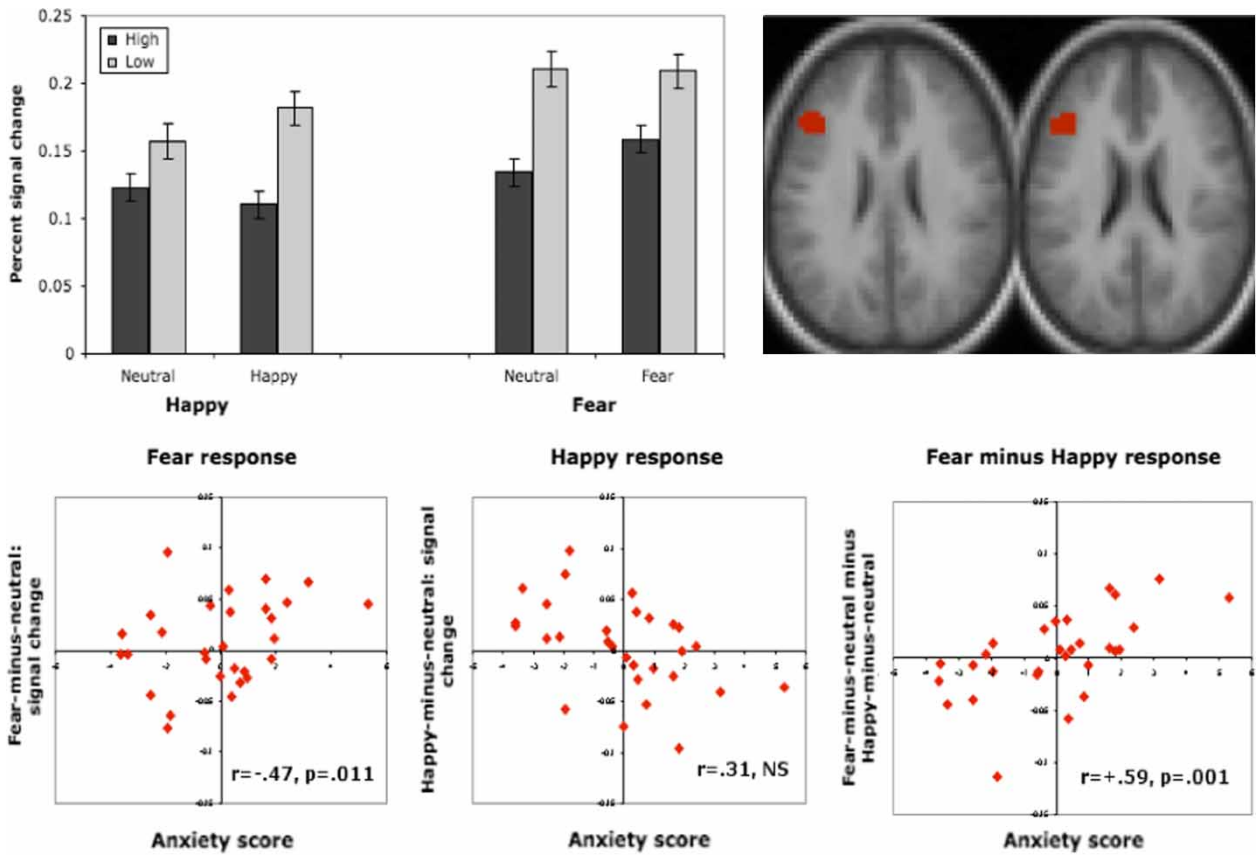

Figure 3. *Significant three-way interaction (Valence × Trial-type × Anxiety) effect on neural activity in left inferior frontal gyrus (Talairach co-ordinates − 43, 22, 21). For the ANCOVA, anxiety was a continuously varying measure, but for visualisation purposes, effects are depicted for participants divided into high- and low-anxiety subgroups (median split). Dark grey: high anxiety scores, Light grey: low anxiety. Error bars indicate standard errors of the mean. [To view this figure in colour, please visit the online version of this journal].*

matrix-reasoning scores. Finally, we also examined whether the faster response times found for happy versus neutral blocks could explain the reduced activation levels found when comparing the happy blocks versus the neutral. For all regions showing a main effect of positive emotion (both blocked and event-related), the activation differences remained significant (or in one case, at trend-level significance) when controlling for RT differences between the conditions. Thus time-on-task effects cannot explain the activation differences found for happy versus neutral conditions.

DISCUSSION

The current study examined anxiety effects on performance and neural activity during WM tasks including emotional target stimuli. We asked whether anxiety would modulate the effect of emotional stimuli on behavioural performance and neural activity, and whether this modulation would depend on stimulus valence. We predicted that high-anxious participants might show enhanced behavioural response to fear faces relative to neutral, while showing little or no facilitation for happy faces. Additionally we questioned whether anxious individuals would show exaggerated activation increases in VLPFC in response to fear-related stimuli, as well as possible decreases in DLPFC.

Anxiety-independent effects of emotional stimuli

In terms of behavioural performance, we found enhanced accuracy for blocks with fearful stimuli, but faster RTs for blocks with happy stimuli. These results are at least partially consistent with prior work showing enhanced behavioural performance for emotional valenced stimuli in WM (Jackson et al., 2008; Langeslag et al., 2009). In terms of neural activity, the blocked analyses revealed significantly reduced activation in a range of cognitive-control (CC) and emotional-interference (EI) regions for happy blocks compared to neutral blocks, with little significant change (except in the amygdala) for the fear blocks.

A more nuanced picture emerged when comparing trial-type effects within a block. For behavioural performance at the trial-type level we again found enhanced accuracy for fear-face versus neutral trials, but slower response times for emotional-face trials versus neutral. In terms of neural activity, the event-related analysis revealed a disjunction in activity patterns for dorsal regions linked to WM versus ventral areas linked to emotional interference. When comparing emotional versus neutral trials, dorsal WM regions showed reduced activation for both fear-face and happy-face trials (relative to neutral trials), suggesting that for emotional trials, participants may have recruited less executive control than for neutral trials. These findings for fearful stimuli are consistent with the results of Perlstein and colleagues (Perlstein et al., 2002), who also found reduced DLPFC activity for negative stimuli during a WM task. In contrast to Perlstein et al., however, we did not find significantly increased activation in DLPFC for positive stimuli. However, as with Perlstein et al., and other research groups (Beneventi et al., 2007; Jackson et al., 2008; LoPresti et al., 2008), we found increased activation in inferior frontal regions for both fearful and happy faces versus neutral. In general, our findings suggest that while emotional valence may indeed facilitate processing in dorsal control regions, it co-occurs with increased recruitment in ventral emotion-processing regions.

An important question for this study is the extent to which our emotional stimuli genuinely facilitated cognitive processing versus interfered with it. On the one hand, the emotion on a face might be entirely independent of making a same–different judgement about it, and hence could function as interference. The increased activation found in emotional-interference regions would seem to support this. On the other hand, emotional valence is known to enhance memory and sensory processes (Labar & Cabeza, 2006; Phelps, Ling, & Carrasco, 2006; Schupp, Junghöfer, Weike, & Hamm, 2003), and the

reduced activation found in more dorsal cognitive-control regions may reflect either greater ease of processing in these cases or on the contrary, reduced ability to mobilise executive control. The improved behavioural performance in emotional versus neutral blocks would seem to support a facilitation interpretation, but the slower response times at the trial-type level suggests possible interference. The current results argue that both kinds of mechanism may be at work for emotional stimuli.

Effects of anxiety on emotional-stimulus processing

In the current study, anxiety notably failed to modulate any performance measures as a function of emotion. Nevertheless, anxiety-related effects were found in the underlying neural activity. In both blocked and event-related analyses, there was a prominent positive relationship between anxiety scores and increases in the left amygdala for fear faces versus neutral. Increased amygdala activation for fear faces is consistent with a wealth of data supporting a role for this region in fear-related emotional processing (LeDoux, 1996), and in the processing of motivationally salient stimuli such as human faces (Adolphs, Tranel, Damasio, & Damasio, 1994; Anderson & Phelps, 2001). The enhancement of this response in higher-anxiety participants adds to the evidence for greater reactivity in such individuals to negative stimuli (Fox & Georgiou, 2005; Mogg, Bradley, & Williams, 1995).

Blocked analyses also found a negative relationship between anxiety scores and activation in the MFG (Brodmann area 6), revealing a pattern consistent with laterality effects found in previous research (Canli, Desmond, Zhao, Glover, & Gabrieli, 1998). Specifically, higher anxiety was associated with smaller activation increases for happy blocks in the left hemisphere, and with smaller increases for fear blocks in the right hemisphere. Thus, activation increases in these regions depended on both anxiety scores and stimulus valence, with hemispheric involvement consistent with other research linking negative-emotion processing to the right and positive-emotion processing to the left (Davidson & Fox, 1982; Wager, Phan, Liberzon, & Taylor, 2003). These effects, however, were not found in the event-related analyses, suggesting that individual differences in anxiety may play a role in context effects. Independent of stimulus valence, higher-anxiety participants recruited these dorsal PFC regions less for emotional stimuli than participants with lower anxiety. This finding is not consistent with our prediction that anxiety-related effects in lateral PFC would be greater for fear stimuli and less (or non-existent) for happy stimuli. Instead, this finding is consistent with the work of Bishop (2007, 2009), which posits overall lower recruitment of lateral PFC at higher trait anxiety levels, even in the absence of any emotional stimuli at all.

Anxiety effects for event-related contrasts were limited to regions linked to emotional-interference processing, and these effects mostly depended on stimulus valence. As mentioned above, higher anxiety levels were associated with enhanced amygdala response to fear faces (relative to neutral), and also with decreased response in IFG regions for happy-face trials. Notably, contrary to our predictions, activation increases found in inferior frontal regions for fear-face trials were not significantly related to anxiety. The reduced activation in VLPFC in the happy-face trials suggests that participants with greater trait anxiety recruited less emotional-interference processing for these trials than people with lower levels of anxiety. Ventrolateral PFC activity has been linked to interference resolution in emotional-distractor paradigms (Dolcos & McCarthy, 2006; Mitchell et al., 2008; Ochsner et al., 2009) and to emotional suppression in emotion-regulation studies (Ochsner et al., 2004; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). Thus, our current finding in this region may reflect either: (1) reduced availability of interference-processing resources in higher anxiety (a reduced cognitive control argument); or (2) reduced need for interference processing. The former interpretation may be unlikely, given that we did not find this effect for the fear-face condition. On the other

hand, the latter interpretation could play a role for example if high-anxious individuals are less susceptible to emotional arousal by positive stimuli, or perhaps experience them as less distracting than low-anxiety participants do.

In line with this interpretation, we also found a single region in left IFG (Brodmann area 45, overlapping cognitive-control and emotional-interference masks) that showed different activation patterns depending on both stimulus valence and anxiety score. Less-anxious participants increased activation in this region for happy faces (relative to neutral faces), but not for fear faces in the fear blocks. By contrast, more-anxious participants increased activation for fear faces (relative to neutral), but not for happy faces in the happy blocks. This result hints at a possible neural correlate for mood-congruent processing biases. Decades of research have documented a bias in anxious individuals to attend to negative material (Fox & Georgiou, 2005; Mogg et al., 1995), and often also a bias against attending to positive material (Hunt et al., 2006; Keogh et al., 2001). Given our lack of anxiety-related performance differences, the current data cannot directly support this notion, but the activity found for this lateral IFG region would be consistent with such findings. For example, participants might show greater recruitment of this region to the extent that each individual finds particular trials more difficult. The low-anxious, who perhaps engage more automatically with happy facial expressions, could find these faces either more arousing or at least more distracting from the task of making a same–different judgement. By contrast, the high-anxious may respond more automatically to the fearful faces, and thus recruit lateral IFG more for fear trials than for neutral, but show no such difference for the happy block. This pattern is consistent with other studies linking lateral inferior frontal regions to resolution of emotional interference (Mitchell et al., 2008). Moreover, it is noteworthy that this result seems equally driven by both the happy- and fear-emotion conditions, since Mitchell et al. (2008) found that this region responds mainly to negatively valenced interference. The current result raises the possibility, though speculative, that activity in this region may be sensitive to individual differences in processing challenge, where such challenges arise from the combination of stimulus valence and individual levels of trait emotion or arousal.

The current findings resonate somewhat with the recent "attentional control theory" (Eysenck et al., 2007), which recasts the notion of anxiety effects on attentional control as a shift in the balance between top-down and bottom-up control systems. This theory proposes that whereas non-anxious individuals might exert robust top-down (executive) control of processing, anxiety is likely to reduce top-down control in favour of more bottom-up influences. Top-down cognitive control is usually considered to be mediated by ACC and DLPFC (Botvinick, Braver, Barch, Carter, & Cohen, 2001). In the current study, we have seen that anxiety was negatively related to activity increases in DLPFC (Brodmann area 6) in response to emotional faces, which may be a sign of loss of top-down control. These effects were not trial-type specific, and may reflect more global context effects of intermittent emotional arousal. By contrast, the current findings also show a positive relationship between amygdala response and anxiety (at least in response to the fear faces). This finding supports the notion that anxiety increases responsiveness to bottom-up emotional influences. By definition, bottom-up control may be viewed as a form of stimulus-driven, reactive cognitive control (Braver, Gray, & Burgess, 2007) that tightens attentional selection as needed when distractions present themselves. While the n-back task is not considered a test of attentional selection, we argue that in the context of emotional stimuli, emotional arousal may in fact impose attentional conflict. The pattern of anxiety-specific results found in the left IFG suggests that both high- and low-anxious individuals may have utilised reactive (bottom-up) control mechanisms when confronted with the types of emotional arousal to which each is uniquely vulnerable.

The use of the combined blocked and event-related design for this study enabled us to detect

effects with each analysis that were not present in the other. The blocked analysis found several possible context-related effects, including the reduced activation for higher-anxious individuals in bilateral Brodmann area 6 and an unusual decrease in activation in right amygdala (which was lower in the fear block than in the neutral block). Although amygdala activation almost always increases in response to threat-related stimuli, deactivations in the right amygdala have been occasionally reported (Geday & Gjedde, 2009; Simpson, Drevets, Snyder, Gusnard, & Raichle, 2001). These latter deactivations were reported where performance on harder versus easier tasks was compared. We speculate that such situations may incur sustained increases in performance anxiety over a block of trials, with the right-amygdala deactivation perhaps reflecting a form of ongoing arousal regulation. Finally, in terms of performance, the disparity in results for happy-minus-neutral response times at the block-level versus trial-type level again suggests the presence of context effects, in that emotional facilitation of performance may depend more on emotional tone for a current block than for particular trials.

An important limitation of this study is the composition of the sample. Participants with high levels of depression were excluded from the study, a restriction likely to reduce the anxiety scores of remaining participants, given the known overlap between depressive and anxious traits (Brown, Campbell-Sills, Grisham, & Mancill, 2001). As a result, the study may have had limited power to detect anxiety-related differences in behavioural performance or in neural efficiency if they existed. Nevertheless, we found a number of significant effects of anxiety, including increased amygdala response and reduced response in several cognitive-control and emotional-interference regions on emotional trials. In particular, the valence × emotion × anxiety interaction found in the left IFG is quite significant, and perhaps more meaningful for being found in a group of otherwise healthy (non-clinical) adults. It remains for a future study to examine the effects of emotional stimuli on patients with clinical levels of anxiety.

Conclusion

The current study examined the effects of anxiety on processing of emotionally valenced stimuli in a WM task. The study investigated the possibility that such stimuli might lead to enhanced cognitive or neural effects specifically in the mixed-fear blocks for participants with higher anxiety scores. Neural effects were predicted to be enhanced response to emotional interference in VLPFC, or potentially decreased response in DLPFC. These effects were found for the sample as a whole and thus were not anxiety specific. However, anxiety did increase activation in the left amygdala for fear stimuli relative to neutral, and reduce it in a bilateral dorsal PFC region for both fear and happy stimuli. Moreover, in one area of left IFC, higher anxiety was associated with increased activation for fear stimuli, while lower anxiety was associated with increased activation for happy stimuli. Overall, the current results are consistent with the notion that anxiety effects can be conceptualised as a shift toward greater susceptibility to bottom-up influence of emotional arousal for negative stimuli, as suggested by the Attentional Control Theory of Eysenck and colleagues (Derakshan & Eysenck, 2009; Eysenck et al., 2007).

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APPENDIX

Table A1. Centres of mass for Working-Memory/Cognitive-Control regions of interest (ROIs) used for the mask the neuroimaging data (see method section for details)

Brain region	*X*	*Y*	*Z*	*mm*^{3}
Owen et al. (2005)				
Lateral premotor (6)	28	0	52	20 mm sphere
	−26	2	52	20 mm sphere
Dorsal cingulate/SMA (32,6)	−2	12	42	20 mm sphere
Dorsolateral PFC (46,9)	42	32	30	20 mm sphere
Ventrolateral PFC (44)	−50	12	8	20 mm sphere
	−62	0	14	20 mm sphere
Frontal pole (10)	−38	44	20	20 mm sphere
	36	46	18	20 mm sphere
Medial posterior parietal (7)	12	−64	48	20 mm sphere
Inferior parietal lobe (40)	30	−58	42	20 mm sphere
	38	−46	38	20 mm sphere
	−34	−48	38	20 mm sphere
Wager et al. (2003)				
BA 10,9,46,47	−32	34	22	20 mm sphere
BA 9,6	−45	7	32	20 mm sphere
BA 40,39,7	−37	−51	41	20 mm sphere
BA 9,10,46	36	36	28	20 mm sphere
BA 7,40	31	−59	43	20 mm sphere
BA 47,10,11,13	34	31	−4	20 mm sphere
BA 7	−12	−70	46	20 mm sphere
BA 6,32,8	0	11	49	20 mm sphere
BA 6	27	0	56	20 mm sphere
BA 6,9,44	45	1	29	20 mm sphere
BA 6	−28	−4	56	20 mm sphere

Note: BA = Brodmann area. SMA = Supplementary Motor Area; PFC = Prefrontal Cortex.

Table A2. Centres of mass for the Emotional-Interference regions of interest (ROIs) used to mask the neuroimaging data (see method section for details)

	X	*Y*	*Z*	*mm*^{3}
Anticevic et al. (in press)				
Middle frontal gyrus (BA 10)	28	53	13	20 mm sphere
IFG/MFG (BA 45,46)	51	32	16	20 mm sphere
Bishop et al. (2004)				
Medial PFC (BA 9,10)	−2	50	18	20 mm sphere
Ventrolateral PFC/Insula	−36	16	−6	20 mm sphere
Dolcos & McCarthy (2006)				
IFG (BA 45,46)	53	28	12	20 mm sphere
IFG (BA 45)	−50	27	4	20 mm sphere
Subgenual ACC (BA 25)	2	13	−16	20 mm sphere
Insula	40	−12	−1	20 mm sphere
Fales et al. (2008)				
Amygdala	21	−7	−15	Talairach
Amygdala	−21	−7	−15	Talairach
Kim et al. (2004)				
IFG (BA 44,45)	59	8	18	20 mm sphere
OFC (BA 47,25)	−15	8	−15	20 mm sphere
IFG (BA 45)	47	32	4	20 mm sphere
IFG (BA 47)	34	32	−8	20 mm sphere
Subgenual ACC (BA 25,32)	5	19	−6	20 mm sphere
Ventromedial PFC (BA 11)	4	29	−11	20 mm sphere
Mitchell et al. (2008)				
IFG (BA 47)	32	29	−1	20 mm sphere
IFG (BA 45,46)	−37	29	12	20 mm sphere
Ochsner et al. (2009)				
SFG (polar BA 10)	16	62	12	20 mm sphere
IFG (BA 47)	−42	40	−8	20 mm sphere

Note: BA = Brodmann area. IFG = Inferior Frontal Gyrus; MFG = Middle Frontal Gyrus; SFG = Superior Frontal Gyrus; PFC = Prefrontal Cortex; ACC = Anterior Cingulate Cortex; OFC = Orbitofrontal Cortex.