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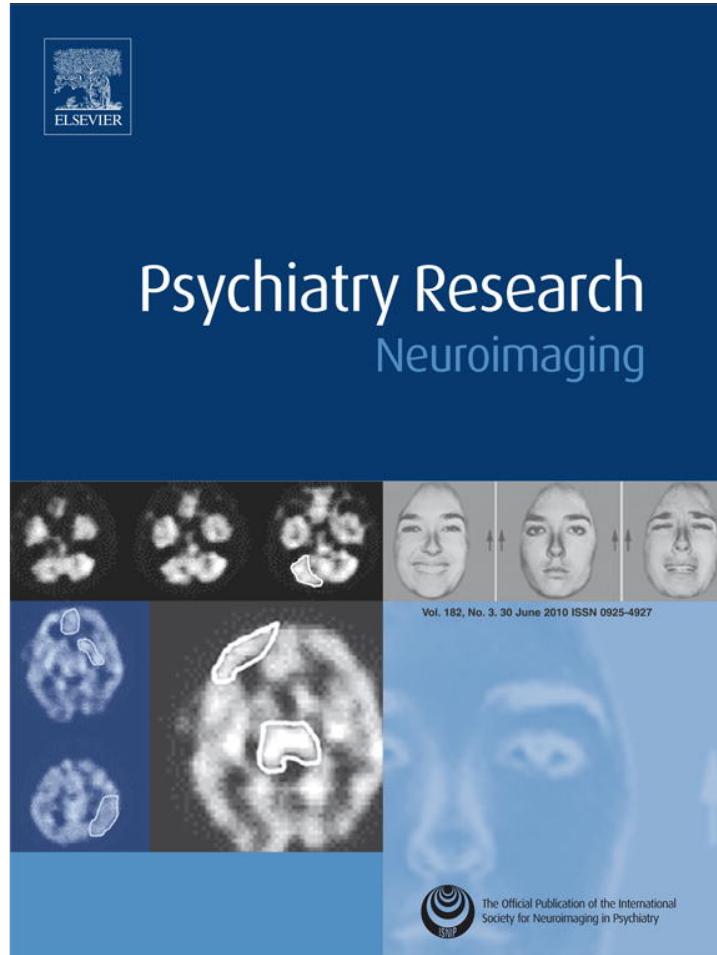
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Psychiatry Research: Neuroimagingjournal homepage: www.elsevier.com/locate/psychresns**Brief report****Blind rage? Heightened anger is associated with altered amygdala responses to masked and unmasked fearful faces**

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ABSTRACT

We investigated anger-related variability in the BOLD fMRI response to crude/masked and detailed/unmasked fearful faces. Anger expression positively covaried with amygdala activation to crude fear, while trait anger negatively covaried with amygdala responses to detailed fear. This differential processing may trigger aggression without the subsequent inhibition associated with distress cues.

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1. Introduction

Given the high incidences of aggression and violence that stem from the expression of anger, it is important to understand the functional neuroanatomy underlying individual differences within this variable. The amygdala is critically involved in the recognition of fearful facial expressions (Adolphs et al., 1994), which are salient threat and distress cues that are thought to reduce aggression/anger in healthy populations, thereby stabilizing social interactions (Marsh and Blair, 2008). Aggression-related traits are associated with impairments in recognizing fearful faces and hypoactive amygdala responses to fearful faces (Gordon et al., 2004; Marsh and Blair, 2008). In addition to emotion recognition, the amygdala is also involved in modulating the expression of emotions, including anger (LeDoux, 1996). Yet, the relationship between amygdala reactivity and the disposition for aggressive behavior has not been examined.

Evidence from animal (LeDoux, 1996) and neuroimaging (Liddell et al., 2005; Vuilleumier and Pourtois, 2007) research indicates that threat information is relayed to the amygdala through two pathways. These include a subcortical route (via the thalamus) for rapid responses to crude threats and a slower cortical route (including the fusiform gyrus for visual stimuli) for more discriminative/detailed responses. Here we investigated how amygdalar reactivity to both crude (initial sensory processing interrupted and restricted by backward masking) and detailed (unmasked) facial cues is associated

with trait anger (i.e., disposition to feel angry) and anger expression (i.e., disposition for aggressive behavior).

2. Methods**2.1. Participants**

Fifteen (female = 7; 19–48 years old, $M = 26.60$, S.D. = 7.41) healthy consenting adults participated in the study. Thirteen reported being right-handed and two left-handed. Participants completed the State-Trait Anger Expression Inventory-2 (Spielberger, 1999) and the State-Trait Anxiety Inventory (Spielberger et al., 1970). Participants' Trait Anger (TA; 11–23, $M = 14.8$, S.D. = 3.21), Anger Expression-Out (AE-O; 9–24, $M = 13.87$, S.D. = 4.09), and Trait Anxiety (20–41, $M = 32.33$, S.D. = 5.73) scores were within the normal range. TA and AE-O were significantly correlated with each other ($r = 0.66$, $P = 0.008$), but not with age or Trait Anxiety ($P > 0.10$).

2.2. Experimental setup and procedure

The experiment was programmed and run with E-prime (Psychology Software Tools, Pittsburgh, PA, USA). An MRI-compatible 60-Hz projector with a 1024×768 resolution reflected stimuli onto a mirror attached to the head coil. Facial stimuli (Gur et al., 2002) were grey-scaled and cropped to eliminate hair and other extraneous features. Each trial started with a 2300-ms fixation cue (+) centered on a black background. Next, the initial face was briefly (33 ms) presented and then immediately masked by a new face for 167 ms. Finally, a jittered intertrial interval ($M = 5.5$ s, 2.5–17.5 s) followed the face pairs. Trial types were determined by the order and expression of the initial face-mask face pairing, where masked fearful = fearful-neutral (FN),

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unmasked fearful = neutral-fearful (NF), and neutral = neutral-neutral (NN). There were a total of 132 trials (44 of each type) presented pseudorandomly in a 12-min run. The mask face was offset from the initial face by approximately 1° of visual angle on either the Y- or X-axis to reduce apparent motion (Liddell et al., 2005). We do not claim that backward masking rendered the initial image subliminal per se, but it did restrict fearful face processing during FN relative to NF trials. Participants were instructed to always maintain fixation in the center of the screen and to pay close attention to the faces.

2.3. Functional image acquisition and analysis

A 3 T Philips whole body scanner was used to acquire 288 T2*-weighted scans with an EPI sequence using the following parameters: Repetition Time = 2500 ms, Echo Time = 22 ms, Flip Angle = 83°, Matrix Dimensions = 96 × 96, Field of View = 224 × 224 mm, Slices = 36, Slice Thickness = 3.5 mm, Gap = 0. Standard preprocessing procedures were performed in SPM5, including image realignment corrections for movement, slice timing corrections, normalization to standard 2 × 2 × 2 mm Montreal Neurological Institute space, and spatial smoothing with a Gaussian full-width-at-half-maximum 6-mm filter. First-level single subject SPMs were created for each condition (FN, NF, and NN). Second-level analyses of FN vs. NN and NF vs. NN with TA and AE-O regressors were created. Bilateral amygdala, thalamus, and posterior fusiform gyrus ($y \leq -36$) region-of-interest (ROI) analyses were performed in SPM5 using the Masks for ROI Analysis (Walter et al., 2003) with a cluster-level search volume corrected (SVC) $\alpha = 0.05$ and extent

thresholds of 10 and 20 continuous voxels for subcortical and cortical regions, respectively.

3. Results

As displayed in Fig. 1, the results of the ROI analyses revealed that for masked (FN > NN) fearful faces AE-O positively covaried with the left amygdala (-26, 0, and -16, $t(13) = 2.92$, $P_{\text{corrected}} = 0.006$, $k = 40$), while for unmasked (NF > NN) fearful faces TA negatively covaried with the right amygdala (28, 0, and -26, $t(13) = 2.20$, $P_{\text{corrected}} = 0.023$, $k = 21$). These associations remained significant ($P < 0.05$) in partial correlations controlling for age, gender, and anxiety. There were no significant associations in the thalamus or posterior fusiform gyrus.

4. Discussion

To our knowledge, we provide the first evidence that individuals high in anger expression have an amplified left amygdala response to crude (i.e., backward masked) representations of fearful faces. Additionally, we found that higher levels of trait anger coincided with decreased right amygdala reactivity during unrestricted/unmasked fearful face processing, which is consistent with previous findings of hypoactive amygdala responses and impaired fearful face recognition in antisocial and aggressive populations (Gordon et al., 2004; Marsh and Blair, 2008). Anger expression and trait anger were not associated with activity in perceptual areas (i.e., thalamus and fusiform gyrus), but only in the fear/emotion processing amygdala.

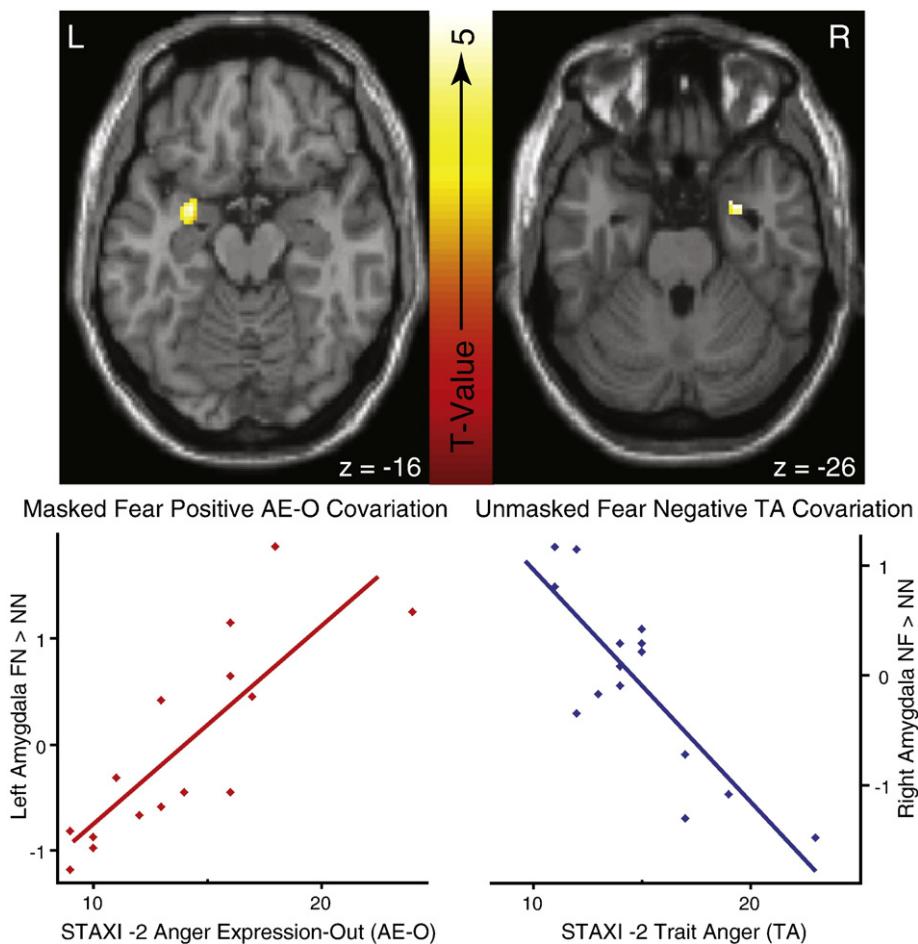


Fig. 1. The left amygdala positively covaried with anger expression during crude/masked fearful face processing (left panels). The right amygdala negatively covaried with trait anger during detailed/unmasked fearful face processing (right panels).

The observed hyperactive left amygdala response to crude fear expressions in individuals with higher levels of anger expression may reflect a mechanism that triggers aggressive responses, while the hypoactive right amygdala response to detailed fear expressions may reflect deficits in fearful face processing, which result in dismissal of these distress cues. In extreme cases, this differential amygdala reactivity may lead to "blind rage" or aggressive behavior without appropriate distress processing and subsequent withdrawal. Interestingly, the observed amygdala asymmetries are consistent with models of affective asymmetry where the left hemisphere is thought to be involved in approach-related behaviors (e.g., lashing out), whereas the right hemisphere is associated with withdrawal behaviors and negatively valenced perceptual processing (Demaree et al., 2005). Given our sample size and lack of a recognition test, future research in this area is needed. Nevertheless, the current findings lead us to speculate that two processes are associated with aggression—a rapid reactivity to crude threat/distress that facilitates the aggressive response and a deficit in processing detailed threat/distress cues that maintains it.

Acknowledgements

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