When psychopathy impairs moral judgments: neural responses during judgments about causing fear

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Psychopathy is a disorder characterized by antisocial behavior, reduced empathy and remorse, narcissism and impulsivity (Hare, 1991). Elevated psychopathic traits have been linked to atypical neural functioning in brain structures that include the amygdala, orbitofrontal cortex and striatum (Birbaumer et al., 2005; Rilling et al., 2007; Dolan and Fullam, 2009; Jones, et al., 2009; Harenaki, et al., 2010; Finger et al., 2011; Marsh et al., 2011a). These disruptions are thought to underlie emotion processing deficits in psychopathy, particularly dysfunctions in recognizing and responding to fear-relevant stimuli such as fearful facial expressions (Marsh and Blair, 2008). A lingering central question is how these neural and cognitive disruptions increase psychopathic individuals’ risk for engaging in antisocial behaviors such as threats, bullying and instrumental violence (Skem et al., 2011). The present study aimed to address this question using a novel task in which healthy adult participants varying in psychopathic traits underwent whole-brain fMRI scanning while they viewed statements that selectively evoke anger, disgust, fear, happiness or sadness. During scanning, participants judged whether it is morally acceptable to make each statement to another person. Psychopathy was associated with reduced activity in right amygdala during judgments of fear-evoking statements and with more lenient moral judgments about causing fear. No group differences in amygdala function or moral judgments emerged for other emotion categories. Psychopathy was also associated with increased activity in middle frontal gyrus (BA 10) during the task. These results implicate amygdala dysfunction in impaired judgments about causing distress in psychopathy and suggest that atypical amygdala responses to fear in psychopathy extend across multiple classes of stimuli.

Keywords: psychopathy; amygdala; moral judgment; fear; empathy; antisocial

Psychopathy is a disorder characterized by antisocial behavior, reduced empathy and remorse, narcissism and impulsivity (Hare, 1991). Elevated psychopathic traits have been linked to atypical neural functioning in brain structures that include the amygdala, orbitofrontal cortex and striatum (Birbaumer et al., 2005; Rilling et al., 2007; Dolan and Fullam, 2009; Jones, et al., 2009; Harenaki, et al., 2010; Finger et al., 2011; Marsh et al., 2011a). These disruptions are thought to underlie emotion processing deficits in psychopathy, particularly dysfunctions in recognizing and responding to fear-relevant stimuli such as fearful facial expressions (Marsh and Blair, 2008). A lingering central question is how these neural and cognitive disruptions increase psychopathic individuals’ risk for engaging in antisocial behaviors such as threats, bullying and instrumental violence (Skem et al., 2011). The present study aimed to address this question using a novel task in which healthy adult participants varying in psychopathic traits underwent whole-brain fMRI scanning while they viewed statements that selectively evoke anger, disgust, fear, happiness or sadness. During scanning, participants judged whether it is morally acceptable to make each statement to another person. Psychopathy was associated with reduced activity in right amygdala during judgments of fear-evoking statements and with more lenient moral judgments about causing fear. No group differences in amygdala function or moral judgments emerged for other emotion categories. Psychopathy was also associated with increased activity in middle frontal gyrus (BA 10) during the task. These results implicate amygdala dysfunction in impaired judgments about causing distress in psychopathy and suggest that atypical amygdala responses to fear in psychopathy extend across multiple classes of stimuli.

Psychopathy has long been known to impair recognition of and responses to fear-related stimuli. Fear-processing deficits in psychopathy include reduced electrodermal responses, potentiated startle, Pavlovian conditioning and passive avoidance to impending threats, like shock (Blair, 2005), as well as impaired fear conditioning, recognition of fear expressed by the face (Marsh and Blair, 2008; Sylvers, et al., 2011), voice (Blair et al., 2002, 2005) and body (Munoz, 2009). Psychopathy is also linked to reduced subjective experience of fear, which has been described anecdotally (Hare, 1993) as well as empirically (Aniskiewicz, 1979; Lykken, 1995; Birbaumer et al., 2005; Marsh et al., 2011b). Therefore, although many descriptions of psychopathy emphasize general poverty of affect, psychopathy appears to disrupt fear more than other emotions among both institutionalized and community samples.

Amygdala dysfunction has been identified as a likely source of deficient processing of fear-relevant stimuli in psychopathy (Kiehl et al., 2001; Blair, 2007; Shurtleff et al., 2009; Yang et al., 2009). The amygdala is essential for evaluating the emotional significance of stimuli, particularly fear-relevant stimuli (LeDoux, 2003; Whalen, 2007). Two meta-analyses of human neuroimaging data have shown that the amygdala responds preferentially to fear stimuli, including fearful facial expressions, relative to other categories of emotional stimuli (Phan et al., 2002; Murphy et al., 2003). Supporting the idea that the amygdala is critical for fear responding, amygdala lesions result in impaired fear conditioning, potentiated startle and recognition of fearful facial expressions (Adolphs et al., 1994; LaBar et al., 1995; Angrilli et al., 1996). Because psychopathy impairs performance in these same tasks, it is hypothesized that impaired fear responding in psychopathy results from amygdala dysfunction. Consistent with this, psychopathy is associated with reduced amygdala activity during exposure to fear-relevant stimuli like fearful facial expressions but not, for example, angry or neutral expressions (Marsh et al., 2008; Dolan and Fullam, 2009; Jones et al., 2009).

Is this ‘fear blindness’ relevant to the fact that individuals with psychopathic traits engage in behaviors that cause others fear, including threats, bullying and violence (Blair et al., 2006)? Research conducted by Marsh and Cardinale (2012) recently linked these features of psychopathy in a community sample. Paralleling prior findings that psychopathy impairs the ability to identify which emotional expressions convey fear, psychopathy was found to impair the ability to identify which of a series of emotionally evocative statements would cause fear in a listener. Moreover, psychopathy resulted in more lenient moral judgments about the acceptability of causing someone fear. And impaired recognition of the fear-evoking statements predicted leniency of judgments. These deficiencies were specific to fear; judgments of other types of transgressions were largely unrelated to psychopathy.

These data suggest that fear-processing deficits in psychopathy extend to judgments about causing others fear. The present study assesses the role of the amygdala in this feature of psychopathy.
We conducted whole-brain fMRI scanning in healthy adults who varied in psychopathic traits as they judged the moral acceptability of emotionally evocative statements. We first wished to determine whether high psychopathy scorers would show atypical amygdala activity only when evaluating the acceptability of causing fear. We predicted that low psychopathy scorers would show increased amygdala activation when judging fear-evoking statements and that high psychopathy scorers would not show this increase. Behaviorally, we predicted that reduced amygdala activation in psychopathy would be accompanied by more lenient moral judgments about causing fear. In keeping with prior findings, we also predicted that psychopathy would be associated with increased activity in dorsolateral prefrontal cortex across multiple categories of moral judgments (Rilling et al., 2007; Glenn et al., 2009a).

**METHODS**

**Participants**

Thirty-three adult volunteers (20 females, 13 males, $M_{age}=22.0$ years, s.d. = 1.8 years, range = 18–25 years) were recruited from the Georgetown University community and underwent fMRI scanning. High psychopathy scorers were oversampled using recruitment advertisements developed for use in psychopathy research (Widom, 1977). All participants were right-handed and free of psychotropic medications. Participants were screened for current diagnosis of and treatment for mental illness and no participant reported either current diagnosis or treatment. Consent in accordance with the Declaration of Helsinki. The study was approved by the Institutional Review Board at Georgetown University, and all participants provided informed written consent in accordance with the Declaration of Helsinki.

**Measurement of psychopathy and aggression**

Psychopathy was measured using the Psychopathic Personality Inventory-Revised (PPI-R; Lilienfeld and Widows, 2005). The PPI-R is a 154-item self-report measure designed to measure psychopathic traits in a dimensional manner, consistent with indications that psychopathy can be more accurately assessed dimensionally than taxonomically (Skeem et al., 2011). Items were developed in accordance with Cleckley's original characterization of psychopathy (Cleckley, 1988), and each item consists of a statement to which participants indicate how accurately it applies to them using a 4-point scale with response options ranging from ‘false’ to ‘true’. The PPI-R shows relations with psychopathy-relevant criterion measures that parallel those for other measures of psychopathy, notably the PCL-R, which is predominantly used to assess psychopathy in institutionalized samples (Poythress et al., 2010). The revised PPI and its predecessor have been successfully used in many previous neuroimaging explorations of cognitive and moral dysfunction in psychopathy (Gordon et al., 2004; Nunez et al., 2005; Fullam et al., 2009). Among the PPI-R subscales are Fearless Dominance, which indexes primarily affect and empathy deficits relevant to psychopathy (akin to Factor 1 of the PCL variants) and Self-Centered Impulsivity, which indexes impulsive antisociality (Factor 2). Participants were also administered the adult form of the Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al., 2006) a self-report measure that assesses aggressive behavior, including both reactive and proactive aggression.

**Emotionally evocative statements task**

The Emotionally Evocative Statements Task (EEST) was adapted from the task previously described by Marsh and Cardinale (2012). The task incorporates 100 emotionally evocative statements (see Table 1 for sample statements), which are an ecologically valid and sensitive means of eliciting specific emotional responses during neuroimaging (Blair et al., 2008). The statements were generated by young adult university students (comparable to the population tested in this study) in response to a request for statements a person could make to cause another to feel anger, disgust, fear, happiness or sadness. Statements were edited to clarify meaning and to equalize word count among the five emotion categories ($P > 0.10$). A separate sample of participants rated the emotional evocativeness of each statement, and the final statements selected as task stimuli were those that elicited maximal emotion category agreement across participants. Specifically, we calculated four t-values that compared the magnitude of each statement’s mean rating for the intended emotion compared to the other four emotions and selected the 20 statements from each category with the highest average t-values (mean t-values: anger = 6.78, disgust = 10.96, fear = 7.45, happiness = 52.11, sadness = 9.79). Overall agreement as to the emotions generated by the statements is high on average ($M$ all emotions = 81.5%; anger = 56%; disgust = 93%; fear = 75%; happiness = 99%; sadness = 84%) and comparable to picture-based emotion recognition tasks (Britton et al., 2006). Twenty affectively neutral statements were also included as a baseline condition for neuroimaging.

**fMRI scanning task**

During the scan session, four event-related fMRI runs were acquired from each participant, during which participants viewed emotionally evocative statements from the EEST that were projected onto a screen they viewed in a mirror in the MRI scanner. Participants rated the moral acceptability of making each statement to another person. Prior to the commencement of scanning, participants were placed in a light head restraint within the scanner to reduce movement and were instructed to read each statement that appeared on the screen and decide whether it would ever be morally acceptable to make that statement to another person. Responses were collected using response buttons held in the left and right hand, which participants pressed with their thumbs and corresponded to answers of ‘yes’ and ‘no’, respectively. Responses were therefore binary rather than ordinal points on a scale. The order in which the four runs of the task were completed was counterbalanced across participants.

During each of the runs, five statements from each of the six emotion categories were presented for 4000 ms each, during which time participants’ responses were collected. Each run used a separate stimulus set and the order in which the statements in each run were presented was randomized. Each statement slide was followed by a 1000-ms fixation cross. In addition, 10 fixation trials (jitters) appeared for 5000 ms at random intervals during each run of the task. Each run began and concluded with three 5000-ms baseline fixation trials. The task was programmed in E-Studio.
Psychopathy impairs moral judgments

T2* weighted images were collected during fMRI scanning using a 3.0 Tesla Siemens (Erlangen, Germany) TIM Trio whole-body MRI system with echo planar imaging capability (matrix 64 × 64; repetition time, 2500 ms; echo time, 30 ms; field of view, 192 mm; voxels, 3.0 × 3.0 × 3.0 mm). Functional images were acquired with a gradient echo-planar imaging (EPI) sequence (axial plane, 46 interleaved slices). High-resolution T1-weighted anatomical images were also acquired (3D Magnetization Prepared Rapid Acquisition Gradient Echo; 176 1.0-mm axial slices; 250 mm field of view; 1900 ms repetition time, 2.52 ms echo; 246 × 256 matrix).

Preprocessing of fMRI data
Data were analyzed within the framework of the general linear model using Analysis of Functional Neuroimages (AFNI; Cox, 1996). Both individual and group-level analyses were conducted. The first four volumes in each of the four scan collections, collected before magnetization equilibrium was reached, were discarded, leaving 88 TRs per run and 352 TRs total per participant. Data were then concatenated, despiked, motion corrected, spatially smoothed using a 6.0-mm full-width half-maximum Gaussian filter and activation outside the brain was masked. The time series was then normalized such that signal amplitude and regression coefficients represent a percent signal change from the mean. Regressors were created for each emotion category (anger, disgust, fear, happiness, neutral and sadness), and a regressor of no interest was created for trials in which participants did not provide a response. Fixation trials were modeled implicitly. The train of stimulus events was then convolved with a γ-variate hemodynamic response function to account for the slow hemodynamic response (Cohen, 1997). The baseline was modeled by a first-order function and motion artefacts were modeled using the six estimated rigid-body motion parameters. This produced a β-coefficient and associated t-statistic for each voxel and regressor. Participants’ anatomical scans were individually registered to the Talaraich and Tournoux Atlas (Talairach and Tournoux, 1988). Following normalization, voxels measured 3 mm³.

Statistical analysis
Participants were divided into high and low psychopathy scorers on the basis of a median split. For the analysis of behavioral data, participants’ judgments (which were dichotomous) were converted to ratios of unacceptable to acceptable ratings such that higher scores reflect less acceptance. Judgments were analyzed using a 2 (group: low psychopathy and high psychopathy) × 6 (emotion: anger, disgust, fear, happiness, neutral and sadness) repeated-measures ANOVA. Planned t-tests compared responses to each emotion category across high and low psychopathy scorers. Main effects and interactions are reported at P < 0.05, two tailed.

To identify whether activation in the hypothesized brain regions is differentially associated with judgments of the emotion categories as a function of psychopathy, we used two analytic strategies for fMRI data. Hypothesized areas of differential activation that survive both analytic strategies are reported. First, to identify whether a true group × emotion interaction existed in the hypothesized regions we conducted a whole-brain 2 (group: low psychopathy and high psychopathy) × 6 (emotion: anger, disgust, fear, happiness, neutral and sadness) random-effects ANOVA that paralleled our behavioral analysis. ANOVA results were reported that exceeded P < 0.005, uncorrected, for magnitude, with an extent threshold of 10 contiguous voxels, a joint thresholding procedure that balances the risk of type I and type II errors, (Lieberman and Cunningham, 2009), a critical consideration for omnibus interactions (Wahlsten, 1990). Small volume correction (SVC) was applied to clusters located in the amygdala. Mean parameter estimates were extracted from functionally defined regions of interest (ROIs) resulting from the ANOVA to determine the nature of the interaction and to assess the relationship between patterns of neural activation and patterns of behavior (aggression scores). Second, we performed focused tests of our hypothesis, calculated independently from the ANOVA, by conducting five whole-brain double-subtraction contrasts within AFNI (emotion – neutral, low psychopathy – high psychopathy). For these contrast tests, we applied a threshold that yielded a whole-brain false discovery rate (FDR) < 0.05 across all five contrasts (P < 0.0001, uncorrected, with an extent threshold of 10 voxels).

RESULTS
Psychopathy and aggression scores
Consistent with our goal to oversample psychopathic traits, the mean PPI-R score in our sample was 305.4 (s.d. = 41.21, range = 232–412), higher than would be anticipated in a typical community sample of 18- to 25-year-old females and males and higher than that reported in a male offender sample tested during development of the PPI-R (Lilienfeld and Widows, 2005). Following the median split, the mean score for high psychopathy scorers was 337.29 (s.d. = 33.39) and for low psychopathy scorers it was 275.45 (s.d. = 19.52). High psychopathy scorers reported engaging in more aggressive behavior (M = 10.27, s.d. = 6.03) than low scorers (M = 5.94, s.d. = 3.34), t(31) = 2.57, P < 0.05. No group differences emerged for distribution of sex, IQ or age across psychopathy groups (all Ps > 0.20).

Behavioral data
Participants’ moral acceptability judgments during the EEST varied across emotion categories, F(5, 135) = 16.598, P < 0.001. Participants judged causing others to feel fear (M = 4.105, s.d. = 4.31) and anger (M = 4.732, s.d. = 6.11) as least acceptable; ratings of these emotions were not significantly different, t(32) = 1.22, P = 0.233. These judgments were followed by judgments of statements that evoke disgust (M = 0.95, s.d. = 1.85), sadness (M = 0.77, s.d. = 0.91) and happiness (M = 0.02, s.d. = 0.05) and neutral statements (M = 0.02, s.d. = 0.07). There was no main effect of psychopathy group across emotion categories, F(1, 27) = 1.828, P = 0.187. The interaction between emotion and psychopathy group was marginally significant, F(5, 140) = 2.076, P = 0.072. To specifically test our behavioral hypothesis that only judgments of fear-evoking statements would differ across groups, we then collapsed participants’ responses to all non-fear statements and calculated a 2 (group) × 2 (fear statements, average of all other statements) ANOVA, the results of which yielded a similar interaction effect, F(1, 31) = 3.722, P = 0.063. The nature of the interaction was such that high psychopathy scorers judged causing others fear to be more acceptable (M = 2.51, s.d. = 1.67) than low psychopathy scorers did (M = 5.61, s.d. = 2.58), t(30) = 2.190, P < 0.05, whereas no group differences emerged for non-fear statements, t(30) = 1.333, P = 0.192, consistent with previous findings (Marsh and Cardinale, 2012) (Table 2).

fMRI data
The results of the group × emotion interaction identified a cluster of differential activation in right amygdala/parahippocampal gyrus, F(5, 135) = 3.498, P < 0.005, SVC (xyz = 14 8 −19). Analyses of mean parameter estimates within this functionally defined ROI confirmed that fear was also the only emotion for which low psychopathy scorers showed significantly greater activation than high scorers, t(31) = 3.738, P < 0.001 and that, for low psychopathy scorers, fear was the only emotion for which
Psychopathy has long been associated with an increased willingness to engage in behaviors that cause fear in victims, like threats, bullying and violence (Blair, 2005). The present study demonstrated that psychopathy is associated with aberrant amygdala responding during judgments about causing others fear. Participants in this study judged the moral acceptability of making fear-evoking statements, including threats such as ‘I could easily hurt you’ and ‘You can’t protect yourself from me’. Replicating previous results, participants with higher psychopathy scores judged making statements like these to be more acceptable (Marsh and Cardinale, 2012). This study extends these findings by demonstrating that, during their judgments, participants with high psychopathy scores showed reduced amygdala activity relative to participants with lower psychopathy scores.

These results are consistent with the modern construct of psychopathy, initially developed by Cleckley, who, in describing the prototypical psychopath, stated, ‘In the disaster he brings about he cannot estimate the affective reactions of others which are the substance of the disaster… the real psychopath seems to lack understanding of the nature and quality of the hurt and sorrow he brings to others’ (Cleckley, 1988, p. 322). Neurocognitive models of psychopathy developed more recently have built upon this idea. For example, Blair and colleagues have developed a model of violence inhibition in which impaired functioning in amygdala and associated structures (e.g. orbitofrontal cortex) disrupts appropriate emotional responding to victims’ distress, which increases the likelihood of causing ‘hurt and sorrow’ in future victims (Blair, 2005, 1997). This theory is consistent with the present findings, in which we observed reduced amygdala activation in high-psychopathy participants as they considered the (Figure 3).

The results of five independent double-subtraction analyses calculated in AFNI (such that differences in activation from the neutral condition for each type of emotion were compared across high and low psychopathy scorers) supported the specificity of this effect: fear was the only emotion category for which group differences emerged in the amygdala during the task (xyz = 14 − 4 − 19) (Figure 1b).

Our ANOVA also identified a group × emotion interaction effect in right middle frontal gyrus (BA 10, xyz = 35, 38, 20). Analyses of mean parameter estimates confirmed that, for high psychopathy scorers, activation in this region exceeded baseline for multiple emotion categories (Ps < 0.05 for all emotions except disgust, $P = 0.18$). By contrast, activation in this region did not significantly exceed baseline for any emotion in low psychopathy scorers (Figure 2a). The results of double-subtraction analyses computed in AFNI confirmed increased activation in this region in high psychopathy scorers across multiple emotions (Figure 2b).

The results of our omnibus ANOVA identified group × emotion interaction effects in only two regions in addition to right amygdala and right middle frontal gyrus: left and right anterior precuneus (BA 7, xyz = −7 to −61 47 and 2 to −55 44, Table 2). No other interaction effects or main effects of group were identified (main effects of emotion can be seen in Table 3).

To estimate the relationship between activation patterns during task performance and self-reported aggressive behavior, we calculated the relationship between activation in the functional ROIs defined by our group × emotion interaction and aggression, as defined by RPQ scores. Results indicated that aggression was most consistently associated with neural responding during the fear condition: more aggressive participants exhibited more activation in middle frontal gyrus and less activation in left precuneus and (at the trend level) amygdala during judgments of fear-evoking statements (Table 4).

Finally, we conducted post hoc analyses assessing the extent to which PPI-R factor scores related primarily to affect and empathy (Factor 1) or to impulsive antisociality (Factor 2) better predicted observed patterns of activation in the clusters generated by our group × emotion interaction.

### Table 2 Average acceptability judgments for high and low psychopathy scorers

<table>
<thead>
<tr>
<th>Emotion</th>
<th>Low psychopathy</th>
<th>High psychopathy</th>
<th>r value</th>
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<tbody>
<tr>
<td>Fear</td>
<td>5.61</td>
<td>2.51</td>
<td>2.19*</td>
</tr>
<tr>
<td>All non-fear</td>
<td>1.59</td>
<td>0.94</td>
<td>1.33</td>
</tr>
<tr>
<td>Anger</td>
<td>6.47</td>
<td>3.09</td>
<td>1.59</td>
</tr>
<tr>
<td>Disgust</td>
<td>0.91</td>
<td>0.99</td>
<td>0.14</td>
</tr>
<tr>
<td>Happiness</td>
<td>0.01</td>
<td>0.02</td>
<td>0.40</td>
</tr>
<tr>
<td>Neutral</td>
<td>0.00</td>
<td>0.03</td>
<td>1.45</td>
</tr>
<tr>
<td>Sadness</td>
<td>1.00</td>
<td>0.54</td>
<td>1.46</td>
</tr>
</tbody>
</table>

*P < 0.05 (two tailed).
Emotion Group low psychopathy scorers following double-subtraction analyses (fear Mean (SEM) percent signal changes extracted from functionally identified ROIs in the amygdala are presented at right (Adolphs et al., 1994) and vocal expressions (Scott et al., 2002; Murphy et al., 2011). Along the same lines, the present task stimuli consist of written statements for which visual attention is not obviously relevant to recognizing the emotional consequences of the statements or making moral judgments about them. No low-level stimulus features of fear-evoking statements distinguish them from other categories of statements. Yet the influence of psychopathy on responses to fear-evoking statements in the present task precisely mirrors its influence on responses to fearful facial expressions in community and institutionalized sample—both cases, specific deficits in acceptability of causing victims distress. In addition, high-psychopathy participants judged causing victims distress to be more acceptable and reported higher levels of actual aggressive and violent behavior.

The present results also extend previous findings on the role of the amygdala in processing fear-relevant stimuli. The results of two meta-analyses have shown that the amygdala is generally more active during the presentation of fear-relevant stimuli than other categories of emotional stimuli (Phan et al., 2002; Murphy et al., 2003) and that individuals with amygdala lesions show impairments in processing a variety of fear-relevant stimuli, including fearful facial expressions (Adolphs et al., 1994) and vocal expressions (Scott et al., 1997). But to our knowledge this is the first study to find that the amygdala is more sensitive to written statements that evoke fear relative to other emotions. In addition, it confirms that amygdala activation in response to fear-relevant stimuli increases only for individuals on the lower end of the psychopathy continuum.

Because it incorporates written stimuli linked to multiple specific emotions, the present paradigm may provide data relevant to ongoing debates about the role of the amygdala in processing fear-relevant stimuli and how amygdala dysfunction in psychopathy impairs fear processing. One prominent theory is that amygdala dysfunction impairs judgments about fearful facial expressions in psychopathy by reducing respondents’ attention to the eyes of fearful faces (Dadds et al., 2006; Han et al., 2012). This theory is supported by findings that, in both patients with amygdala lesions and in children with psychopathic traits, instructing participants to attend to the eyes of faces reduces fear recognition deficits (Adolphs et al., 2005; Dadd et al., 2008). It is argued that the eyes of fearful faces are most diagnostic features of the expression, and the amygdala helps direct attention to the most salient features of the face.

It is less clear how this theory can be extended to explain impairments in processing of other forms of fear-relevant stimuli. Amygdala lesions and psychopathy both also impair recognition of vocalized fear (Scott et al., 1997; Blair et al., 2002), auditory stimuli for which the relevance of attention directed to salient features is unclear. And a recent study shows that psychopathy also impairs pre-attentive recognition of fearful faces (Sylvén et al., 2011). Along the same lines, the present task stimuli consist of written statements for which visual attention is not obviously relevant to recognizing the emotional consequences of the statements or making moral judgments about them. No low-level stimulus features of fear-evoking statements distinguish them from other categories of statements. Yet the influence of psychopathy on responses to fear-evoking statements in the present task precisely mirrors its influence on responses to fearful facial expressions in community and institutionalized sample—both cases, specific deficits in

![Fig. 2](image) Clusters in which differential activation was observed in high psychopathy scorers (gray bars) and low psychopathy scorers (red bars) following a whole-brain 2 (group) × 6 (emotion category) ANOVA. Mean (SEM) percent signal changes extracted from functionally identified ROIs in the amygdala are presented at right (a). Clusters in which differential activation was observed in high psychopathy scorers and low psychopathy scorers following double-subtraction analyses (fear — neutral, low psychopathy — high psychopathy) across emotion categories in right amygdala (b).

<table>
<thead>
<tr>
<th>Table 3 Functionally defined ROIs identified by a group [high psychopathy, low psychopathy] × emotion (anger, disgust, fear, happiness and neutral sadness) ANOVA in AFNI</th>
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<tbody>
<tr>
<td>Cluster</td>
<td>BA</td>
<td>X</td>
<td>Y</td>
<td>Z</td>
<td>Voxels</td>
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<td>Group × emotion</td>
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</tr>
<tr>
<td>R middle frontal gyrus</td>
<td>10</td>
<td>+35</td>
<td>+38</td>
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<td>18</td>
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<td>R amygdala/parahippocampal gyrus</td>
<td>+14</td>
<td>+8</td>
<td>+9</td>
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<td>+2</td>
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<td>+45</td>
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<td>+38</td>
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<td>R cingulate gyrus</td>
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<td>+2</td>
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<td>+3</td>
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<tr>
<td>R precentral gyrus</td>
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<td>+63</td>
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<tr>
<td>R postcentral gyrus</td>
<td>3</td>
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<tr>
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<td>-26</td>
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<tr>
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</table>

XYZ Cluster BA/C151/C2 Group Functionally defined ROIs identified by a group [high psychopathy, low psychopathy] across emotion categories in right amygdala (b).
identifying fear-relevant stimuli are observed (Marsh and Blair, 2008). And the reduced amygdala response to fear-evoking stimuli in high-psychopathy participants mirrors previous findings of reduced amygdala responses to fearful faces in participants with psychopathic traits (Marsh et al., 2008; Dolan and Fullam, 2009; Jones et al., 2009).

This suggests the possibility of deeper emotional processing impairments that affect how fearful stimuli as a class are processed. One possibility is that correctly identifying fearful expressions requires the generation of an internal representation of fear, which relies on intact amygdala function (Goldman and Sripada, 2005). Amygdala dysfunction in psychopathy may impede the ability to generate an internal representation of fear, thereby impairing identification of others’ fear across contexts. So, for example, correctly identifying a statement like, ‘I could easily hurt you’, as one that would evoke fear may also require the ability to create an internal representation of the target’s likely emotional response. Given previous evidence that psychopathy impairs the ability to identify fear-evoking statements (Marsh and Cardinale, 2012), and current evidence for amygdala dysfunction when reading these statements in high psychopathy scorers, our present findings are consistent with the possibility that representation of the emotional outcome of fear-evoking statements is supported by activity in the amygdala. Our previous findings underscore the previously hypothesized link between awareness that a statement causes fear and judging it to be unacceptable, supporting the possibility that both capacities may rely on the internal representation of the statement’s emotional outcome (Nichols, 2001; Pfaff et al., 2008).

The pattern of activity we observed in bilateral anterior precuneus during the task also supports the possibility that identifying and judging fear-evoking statements requires the generation of an internal representation of fear. Because this effect was not hypothesized, these results are interpreted with some caution. The anterior precuneus, termed ‘the mind’s eye’ in early imaging studies (Fletcher et al., 1995), is thought to play a central role in a variety of tasks associated with the episodic retrieval of polymodal imagery and first-person perspective taking (Cavanna and Trimble, 2006). Activation in this region increases during tasks that require third-person perspective simulation or empathizing and may reflect the generation of vivid representations of the self to imagine the internal state of another person (Ochsner et al., 2004; Ruby and Decety, 2004; Farrow et al., 2005). The results of our analyses indicated that bilateral activation in this region was greatest in participants with lower psychopathy scores evaluating fear-evoking statements, whereas the opposite was true for high-psychopathy scorers. This is consistent with the possibility that the task requires participants to draw on stored representations involving episodic emotional memories. In typical individuals frightening memories are vivid and easily retrieved (McGaugh et al., 1996), whereas this may be less evident among individuals with psychopathic traits.

Participants with high psychopathy scores showed increased activation in middle frontal gyrus (dorsolateral prefrontal cortex) when judging multiple varieties of emotional statements. This is consistent

Table 4  Correspondence between aggression (RPQ score) and neural activity in functionally defined ROIs

<table>
<thead>
<tr>
<th>Region of interest</th>
<th>Anger</th>
<th>Disgust</th>
<th>Fear</th>
<th>Happiness</th>
<th>Neutral</th>
<th>Sadness</th>
</tr>
</thead>
<tbody>
<tr>
<td>R middle frontal gyrus</td>
<td>0.11</td>
<td>0.03</td>
<td>0.34*</td>
<td>0.07</td>
<td>0.20</td>
<td>0.01</td>
</tr>
<tr>
<td>R amygdala/parahipp.</td>
<td>−0.27</td>
<td>0.18</td>
<td>−0.28*</td>
<td>0.18</td>
<td>0.22</td>
<td>0.18</td>
</tr>
<tr>
<td>R precuneus</td>
<td>0.15</td>
<td>0.03</td>
<td>−0.27</td>
<td>0.34*</td>
<td>0.06</td>
<td>0.22</td>
</tr>
<tr>
<td>L precuneus</td>
<td>0.30</td>
<td>0.25</td>
<td>−0.37*</td>
<td>0.11</td>
<td>−0.04</td>
<td>0.24</td>
</tr>
</tbody>
</table>

*P < 0.05 (two-tailed).

Fig. 3  Clusters in which differential activation was observed in high psychopathy scorers (gray bars) and low psychopathy scorers (red bars) following a whole-brain 2 (group) × 6 (emotion category) ANOVA. Mean (SEM) percent signal changes extracted from functionally identified ROIs in dorsolateral prefrontal cortex is presented at right (b). Clusters in which differential activation was observed in high psychopathy scorers and low psychopathy scorers following double-subtraction analyses (fear − neutral, low psychopathy − high psychopathy) across emotion categories in right middle frontal gyrus (a).
Psychopathy has long been associated with two general domains of deficits: impoverished emotional responses, particularly to fear-relevant stimuli and increases in antisocial and aggressive behavior.
Accumulating neuroscience research has identified brain regions in which dysfunction accompanies these traits, such as the amygdala. The results of this study link these features of psychopathy, showing that psychopathy is associated with reduced amygdala activity when judging the acceptability of frightening others and impaired judgments about the acceptability of causing others fear. These findings extend research on fear deficits and amygdala dysfunction in psychopathy to a new class of emotional stimuli and illuminate the putative relationship between emotional impairments and antisocial behavior in individuals with psychopathic traits. By linking psychopathic traits to reduced amygdala responses during judgments of verbal statements that elicit fear, the present study helps explain why psychopathy may increase the belief that to incite fear in another person is a morally acceptable course of action.

Conflict of Interest
None declared.

REFERENCES
Psychopathy impairs moral judgments


