doi:10.1093/scan/nsr092 SCAN (2013) 8, 235–246

Emotion disrupts neural activity during selective attention in psychopathy

Naomi Sadeh,[1] Jeffrey M. Spielberg,[1,2] Wendy Heller,[1,2] John D. Herrington,[1,2,3] Anna S. Engels,[1,2,4] Stacie L. Warren,[1,2] Laura D. Crocker,[1,2] Bradley P. Sutton,[2] and Gregory A. Miller[1,2]

[1]Department of Psychology, University of Illinois at Urbana-Champaign, Champaign, IL, [2]Beckman Institute Biomedical Imaging Center, University of Illinois at Urbana-Champaign, Champaign, IL, [3]Center for Autism Research, Children's Hospital of Philadelphia, Philadelphia, PA and [4]Social, Life, and Engineering Sciences Imaging Center, Pennsylvania State University, University Park, PA

Dimensions of psychopathy are theorized to be associated with distinct cognitive and emotional abnormalities that may represent unique neurobiological risk factors for the disorder. This hypothesis was investigated by examining whether the psychopathic personality dimensions of fearless-dominance and impulsive-antisociality moderated neural activity and behavioral responses associated with selective attention and emotional processing during an emotion-word Stroop task in 49 adults. As predicted, the dimensions evidenced divergent selective-attention deficits and sensitivity to emotional distraction. Fearless-dominance was associated with disrupted attentional control to positive words, and activation in right superior frontal gyrus mediated the relationship between fearless-dominance and errors to positive words. In contrast, impulsive-antisociality evidenced increased behavioral interference to both positive and negative words and correlated positively with recruitment of regions associated with motivational salience (amygdala, orbitofrontal cortex, insula), emotion regulation (temporal cortex, superior frontal gyrus) and attentional control (dorsal anterior cingulate cortex). Individuals high on both dimensions had increased recruitment of regions related to attentional control (temporal cortex, rostral anterior cingulate cortex), response preparation (pre-/post-central gyri) and motivational value (orbitofrontal cortex) in response to negative words. These findings provide evidence that the psychopathy dimensions represent dual sets of risk factors characterized by divergent dysfunction in cognitive and affective processes.

Keywords: psychopathy; fearless-dominance; impulsive-antisociality; emotional distraction; fMRI

INTRODUCTION

There is increasing interest in neural processes associated with violent and antisocial behavior, partly as a means of identifying different neurobiological processes associated with these harmful behaviors (Blair, 2003; Patrick and Bernat, 2009). Research has begun to examine the neural mechanisms of psychopathy, a particularly virulent constellation of personality traits that confers risk for repeated engagement in violent crimes (Hare *et al.*, 2000). However, the vast majority of neuroimaging research has not attended to heterogeneity in the deficits present among individuals who manifest these traits, despite evidence that at least two distinct sets of deficits contribute to psychopathy (Fowles and Dindo, 2009; Patrick and Bernat, 2009). Consequently, the present study used functional magnetic resonance imaging (fMRI) to examine whether two dimensions of psychopathy exhibit patterns of neural activation consistent with the theory that they index distinct risk factors for the disorder.

Research indicates that psychopathy is characterized by two primary personality dimensions (Harpur *et al.*, 1988). The affective-interpersonal dimension is marked by a fearless and low-anxious temperament coupled with a socially dominant and grandiose interpersonal style. In contrast, the social-deviance dimension is characterized by an irresponsible, impulsive and antisocial lifestyle (Harpur *et al.*, 1989). Although elevated scores on both dimensions are needed to qualify as 'psychopathic', the unique variance associated with each dimension relates differentially to external criterion variables (Benning *et al.*, 2003; Ross *et al.*, 2009), which has led researchers to speculate that the dimensions index separate sets of risk factors that can lead individuals to engage in serious antisocial behavior and look phenotypically similar on measures of psychopathy. The two-process theory proposes that these psychopathy dimensions represent dual etiological processes instantiated in separable neurobiological systems (Patrick and Bernat, 2009), which is consistent with research that suggests each dimension is associated with distinct emotional and cognitive deficits. For example, research has linked the affective-interpersonal dimension with deficient emotional reactivity and the social-deviance dimension with emotional dysregulation and enhanced emotional processing (Patrick *et al.*, 1993; Patrick *et al.*, 1994; Patrick and Zempolich, 1998; Raine *et al.*, 1998; Sprague and Verona, 2010). Research also indicates that the affective-interpersonal dimension is associated with abnormal early selective attention (Sadeh and Verona,

Received 15 April 2011; Accepted 27 November 2011
Advance Access publication 29 December 2011
National Institute of Mental Health National Research Service Award grant (F31 MH086178 to N.S.). This work was also supported by the National Institute of Drug Abuse (R21 DA14111) and National Institute of Mental Health (R01 MH61358, T32 MH19554, P50 MH079485).

Correspondence should be addressed to Naomi Sadeh, Department of Psychology, University of Illinois at Urbana–Champaign, 603 East Daniel Street, Champaign, IL 61801, USA. E-mail: naomisadeh@gmail.com

2008; Newman *et al.*, 2010; Baskin-Sommers *et al.*, 2011), whereas the social-deviance dimension is associated with deficits in working memory, including difficulty suppressing the effects on distractors on behavior (Sellbom and Verona, 2007; Sadeh and Verona, 2008).

Based on this research, the psychopathy dimensions should evidence distinct patterns of neural activation that are consistent with the abnormal emotional and cognitive processes proposed to be associated with each dimension. This hypothesis has not been thoroughly examined, however, because the majority of fMRI research in this area has examined psychopathy as a unitary construct in small samples ($\leq$10) using primarily emotional tasks (Kiehl *et al.*, 2001; Muller *et al.*, 2003; Birbaumer *et al.*, 2005; Deeley *et al.*, 2006; Muller *et al.*, 2008). These studies indicate that psychopaths typically show decreased activation in affect-related brain areas and networks during emotional processing, including the amygdala, ventromedial prefrontal cortex, orbitofrontal cortex and broader paralimbic system (Kiehl *et al.*, 2001; Muller *et al.*, 2003; Gordon *et al.*, 2004; Birbaumer *et al.*, 2005; Deeley *et al.*, 2006; Finger *et al.*, 2008; Marsh *et al.*, 2008) and increased activation in fronto-temporal regions (Kiehl *et al.*, 2001; Soderstrom *et al.*, 2002; Muller *et al.*, 2003).

A small set of studies have attempted to parse the differential effects of the psychopathy dimensions on emotional processing, reward anticipation and moral reasoning. These studies provide evidence consistent with the hypothesis that the affective-interpersonal dimension is associated with deficient emotional reactivity, in that it is associated with decreased activation in affect-related regions during emotional processing, including the amygdala and media frontal cortex (Gordon *et al.*, 2004). They also suggest that the social-deviance dimension is associated with increased neural activation in regions related to emotional processing and reward anticipation, including the amygdala and nucleus accumbens (Gordon *et al.*, 2004; Buckholtz *et al.*, 2010) and regions involved in selective attention (Rilling *et al.*, 2007). These studies used primarily emotional manipulations and did not investigate the cognitive deficits purportedly associated with the psychopathy dimensions. Therefore, the hypothesis that the psychopathy dimensions are differentially associated with dysfunction in regions instantiating both emotional processes and cognitive processes remains untested. The present study tested this hypothesis by investigating whether the psychopathy dimensions moderate neural activity during an emotion-word Stroop task (Williams, Mathews, and MacLeod, 1996) that included both a selective attention and an emotional processing manipulation.

Increasingly, research aimed at identifying etiological mechanisms in psychopathy is being conducted with non-forensic samples (Gordon *et al.*, 2004; Benning *et al.*, 2005; Rilling *et al.*, 2007), given evidence that psychopathy can be conceptualized as a constellation of extreme manifestations of normal personality traits (Widiger and Lynam, 1998; Miller *et al.*, 2001) and is dimensional rather than taxonic in nature (Edens *et al.*, 2006; Murrie *et al.*, 2007). Further, use of non-incarcerated samples allows for the study of neural processes without the potential confounds associated with extreme levels of psychopathy, such as histories of violence-related head trauma, heavy illegal substance consumption and long-term incarceration. Consequently, this study recruited adults from the community to examine the influence of trait psychopathy on selective attention and emotional processing.

An emotion-word Stroop task was used to test the hypotheses that (i) the affective-interpersonal dimension would be associated with diminished emotional reactivity and (ii) the social-deviance dimension would be related to heightened emotional processing and poor suppression of distractor effects. Thus, the affective-interpersonal dimension was expected to be associated with decreased activation in brain regions associated with processing of emotional, particularly threatening, stimuli (e.g. amygdala, paralimbic system). In contrast, the social-deviance dimension was hypothesized to be associated with increased neural activity in affect-related brain regions (e.g. amygdala, orbitofrontal cortex) and regions involved in attentional control (e.g. anterior cingulate cortex, dorsolateral prefrontal cortex) to compensate for enhanced sensitivity to emotional distractors. The interaction of the psychopathy dimensions was also examined, because it is unclear how the distinct cognitive and affective processes associated with each dimension operate in individuals who are high on both psychopathy dimensions. Hypotheses for the interaction analysis were based on research that examined individuals scoring high in psychopathy measured as a unitary construct. Thus, individuals high on both psychopathy dimensions were expected to show decreased activation in amygdala, orbitofrontal and broader paralimbic regions over and above the effect of the affective-interpersonal dimension, and increased activation in fronto-temporal areas associated with the processing of emotional, particularly threatening, distracting stimuli.

METHODS

Participants

Forty-nine adults (30 women, 61%) ages 19–51 years ($M = 33.7$, s.d. $= 9.0$) participated in exchange for money. Individuals recruited from the community through advertisements were asked to participate if they did not meet any of the following exclusion criteria: metal in body, claustrophobia, left-handedness, psychosis, mania and current substance disorder determined using the Structured Clinical Interview for DSM-IV-TR (First *et al.*, 2002). Participant data were excluded if the individual (i) moved more than 3.3 mm relative to the registration volume or more than 2 mm relative to the previous volume, (ii) committed errors on 15% or more of trials or (iii) exhibited reaction

times >3 s.d. from the mean across subjects. The 49 participants passed all of these criteria.

Estimated psychopathy facets

The 60-item NEO-Five Factor Inventory (NEO-FFI; Costa and McCrae, 1992) is a well-validated measure of the Big Five dimensions of personality, which were used to estimate psychopathy dimensions in the present study. Each participant rated the extent to which he/she generally behaves in the manner described in each item on a scale from 1 ('Strongly Disagree') to 5 ('Strongly Agree'). Scores were summed to form the dimensions of Neuroticism, Extraversion, Openness, Agreeableness and Conscientiousness. Based on work demonstrating that psychopathy dimensions can be estimated from general personality traits (Benning *et al.*, 2005; Ross *et al.*, 2009), the psychopathy facets of fearless-dominance (FearDom) and impulsive-antisociality (ImpAnti) were computed using standardized scores on the NEO-FFI personality dimensions to represent the affective-interpersonal and social-deviance dimensions of psychopathic traits, respectively. The psychopathy dimensions ($r = -0.21$, $P > 0.13$) were estimated from regression equations generated in a separate sample of undergraduate and incarcerated adults (for beta weights, see Ross *et al.*, 2009), which found that fearless-dominance was marked primarily by low Neuroticism, high Extraversion and low Agreeableness, whereas impulsive-antisociality was marked primarily by low Agreeableness and low Conscientiousness.

Experimental design

Participants completed an emotion-word Stroop while functional magnetic resonance data were collected. The task consisted of 256 trials presented in 16 blocks (four positive, four negative and eight neutral), using a variable intertrial interval (2000 ± 225 ms). Trials began with the presentation of a word in one of four equally occurring ink colors (red, yellow, green, blue) for 1500 ms, followed by a fixation cross for an average of 500 ms. Blocks of positive or negative words alternated with blocks of neutral words, and block presentation order was counterbalanced. There were four fixation blocks and five rest blocks.

The word stimuli were selected from the Affective Norms for English Words set (Bradley and Lang, 1998). Sixty-four were positive (e.g. ecstasy, laughter), 64 were negative (e.g. suicide, war, victim) and 128 were neutral (e.g. hydrant, carpet). Words were selected on the basis of established norms for valence, arousal, frequency of usage in the English language (Bradley and Lang, 1998) and number of letters. Words ranged from three to eight letters (visual angle 6–16 degrees). Participants responded with their index and middle fingers using a four-button response box (James Long Company).

Behavioral data analysis

Mean reaction time and error frequency across trials were analyzed using repeated—measures MANOVAs with Emotion (positive, neutral, negative) as the within-subject factor, continuous scores on FearDom, ImpAnti, and their interaction as between-subjects predictors, and block presentation order as a covariate. Planned contrasts of positive *vs* neutral (PosvNeu) and negative *vs* neutral (NegvNeu) were used to interpret Emotion effects. Although not a focus of the present study, the moderating effects of gender were tested, and gender was not found to influence the behavioral results reported.

fMRI data collection and processing

fMRI data were 370 3D images acquired using a gradient-echo echo-planar imaging sequence (TR 2000 ms, TE 25 ms, flip angle 80°, FOV = 220 cm) on a Siemens Allegra 3 T scanner. Each image consisted of 38 axial slices (slice thickness 3 mm, 0.3 mm gap, in-plane resolution 3.4375×3.4375 mm) acquired parallel to the anterior and posterior commissures. After the fMRI acquisition, a 160-slice MPRAGE structural sequence was acquired (spatial resolution $1\,\text{mm} \times 1\,\text{mm} \times 1\,\text{mm}$) and used to warp functional data into standard space.

Image processing and statistical analysis were performed primarily using FEAT v5.98. The first three time points were discarded to allow the MR signal to reach a steady state. Functional data were motion-corrected using FMRIB's linear registration tool, MCFLIRT (Jenkinson *et al.*, 2003), intensity-normalized, temporally filtered with a non-linear high-pass filter and spatially smoothed using a 3D Gaussian kernel (FWHM = 5 mm). Temporal low-pass filtering was carried out using AFNI's 3dDespike tool (http://afni.nimh.nih.gov/).

Within-subject regression analyses were performed on the processed functional time series of each participant using FILM (Woolrich *et al.*, 2001). Four predictors, one for each word-type block (positive, neutral, negative) and one modeling the rest condition, were included in the regression model (fixation was unmodeled). For each explanatory variable, the vector of assigned weights corresponding to word type was convolved with a gamma function to better approximate the temporal course of the blood-oxygen-dependent hemodynamic response function. Each explanatory variable yielded a per-voxel effect size parameter estimate (β) map representing the magnitude of activation associated with that explanatory variable.

To create comparisons of interest, regression β-values for the conditions were contrasted. A positive affect comparison was created by contrasting the positive condition with the neutral condition (PosvNeu). A negative affect comparison was created by contrasting the negative condition with the neutral condition (NegvNeu). For each participant, these functional activation maps were warped into a common

stereotaxic space (2009 MNI symmetrical $1 \times 1 \times 1$ mm^3, Fonov *et al.*, 2009), using FNIRT.

Four mixed effects multiple regression analyses were conducted using FLAME, two with the PosvNeu contrast and two with the NegvNeu contrast as the dependent variable. Three covariates were included in all regressions to account for variance associated with the block presentation order, which was not correlated with the psychopathy variables (Miller and Chapman, 2001). For each dependent variable, the first regression contained FearDom and ImpAnti as predictor variables, to determine the main effects of these predictors. In the second regression, the interaction between FearDom and ImpAnti was added. Each predictor produced a beta map corresponding to the unique variance associated with that predictor. *T*-tests were conducted on the beta maps and then converted to *z*-scores. A gray-matter mask was used to limit the number of voxels under consideration. In addition, ventral prefrontal cortex (VPFC) and amygdala masks were used to examine a priori hypotheses for these areas.

Monte Carlo simulations via AFNI's AlphaSim program were used to estimate the overall significance level for thresholding the 3D functional *z*-map image (Ward, 2000). These simulations provided the appropriate cluster sizes for an individual *z*-threshold of 2.3263 that would give an overall two-tailed family-wise error rate of 0.05. The minimum cluster sizes for the three masks were: gray-matter = 1170 mm^3, VPFC = 507 mm^3, amygdala = 234 mm^3.

Analysis of brain activation and behavior relationships

To assess the potential effect of neural activity related to the psychopathy dimensions on behavioral performance, a score for each cluster identified in earlier analyses was created by averaging β-values across voxels in each cluster for each participant. Partial correlations between cluster scores and RT interference/error interference were calculated, with the variance associated with the three covariates related to block presentation order (included in the fMRI regressions) partialled out. Potential moderating effects of gender were analyzed for the fMRI results, and no moderating effects of gender were found.

Mediation analyses

Mediation analyses were conducted to determine whether brain activation in clusters correlated with behavior (RT or Error interference) mediated the observed effects of FearDom/ImpAnti on behavior. Following the recommendations of MacKinnon (2008), Sobel (1982) tests were conducted to determine whether significant mediation was present (one-tailed tests were used). Effect sizes were calculated by dividing the indirect effect by the total effect of the relevant psychopathy dimension on behavior, providing the proportion of the total effect that was mediated by brain activation. Standardized *ab* values were calculated by standardizing the *a* and *b* coefficients and calculating the product.

Analyses to detect potential confounds

To ensure that findings were not driven by outliers, fMRI analyses were rerun using FSL's outlier de-weighting (Woolrich, 2008) procedure. Findings were virtually identical, indicating that findings were not due to outliers. In addition, to ensure that fMRI findings were not due to differences in structural gray matter, voxel-based morphometry was carried out using FSL-VBM, with FearDom, ImpAnti and the interaction as predictors. No gray-matter differences were observed in the areas where fMRI activation was associated with FearDom, ImpAnti or the interaction, indicating that present fMRI findings were not due to structural differences.

RESULTS

Behavioral responses

An effect of Emotion emerged for reaction time, $F(2,44) = 17.35$, $P < 0.001$, such that participants responded more slowly to both positive, $F(1,45) = 4.06$, $P = 0.05$, and negative, $F(1,45) = 35.48$, $P < 0.001$, words than neutral words (Positive: $M = 692.9$, s.d. = 83.7; Neutral: $M = 685.8$, s.d. = 92.1; Negative: $M = 714.1$, s.d. = 96.7). Analysis of error frequency also revealed an effect of Emotion, $F(2,44) = 6.81$, $P = 0.003$, with participants committing more errors to both positive, $F(1,45) = 10.02$, $P = 0.003$, and negative, $F(1,45) = 8.97$, $P = 0.004$, relative to neutral words (Positive: $M = 2.9$, s.d. = 1.9; Neutral: $M = 2.0$, s.d. = 1.7; Negative: $M = 2.8$, s.d. = 1.7).

The behavioral interference induced by emotion was moderated by NEO-estimated psychopathy facets. The Emotion effect on reaction time was modulated selectively by scores on ImpAnti, $F(2,42) = 4.86$, $P = 0.013$. Contrasts revealed that ImpAnti was positively associated with response interference (reaction time slowing) for the PosvNeu contrast, $F(1,43) = 8.57$, $P = 0.005$, and a similar trend emerged for the NegvNeu contrast, $F(1,43) = 3.74$, $P = 0.06$. The effect of Emotion on error frequency was moderated by FearDom, $F(2,42) = 3.56$, $P = 0.037$, such that scores on this dimension were positively and selectively related to the commission of errors for the PosvNeu contrast, $F(1,43) = 4.23$, $P = 0.046$. Overall, findings suggest that individuals high in ImpAnti were more distracted by the arousing properties of the emotional stimuli than individuals low on this psychopathy dimension. In contrast, positive stimuli were particularly distracting to individuals high in FearDom.

Moderation of neural activation by fearless-dominance

As shown in Table 1 and Figure 1, FearDom correlated positively with PosvNeu activation in one cluster in right superior frontal gyrus (SFG) and one in left Supramarginal Gyrus (SG)/Inferior Parietal Lobule (IPL). FearDom did not moderate NegvNeu activation.

Table 1 Brain areas moderated by NEO-FFI estimated psychopathy dimensions and correlations with behavior

Region	Cluster size (mm^3)	Direction of relationship	Mean *z*-value	Location			RT	Errors
				x	*y*	*z*		
Fearless-dominance								
Positive *vs* neutral								
R SFG[a] (BA 6)	1423	Positive	2.75	11	18	66	.20	0.42*
L SG/IPL[a] (BA 40)	1603	Positive	2.68	−65	−46	32	0.16	0.21
Impulsive-Antisociality								
Positive *vs* neutral								
L ITG/MTG/STG/IFG/OFC[a] (BA 13/20/21/38/47)	10 069	Positive	2.80	−45	8	−24	0.49*	0.15
L ITG/MTG[a] (BA 21/20)	1533	Positive	2.68	−59	−31	−12	0.04	−0.04
L MTG/STG/SG/AG/Precuneus/IPL[a] (BA 19/39/40)	4244	Positive	2.76	−47	−63	35	0.49*	0.21
M and L Frontal Pole[a] (BA 10)	1997	Positive	2.64	−5	65	−5	0.38*	−.16
L SFG[a] (BA 6/8)	1492	Positive	2.64	−15	29	53	0.37*	0.10
L Amygdala[c]	481	Positive	2.76	−27	−8	−23	0.33*	−0.29
Negative *vs* Neutral								
M Cuneus[a] (BA 18)	1971	Positive	2.56	−5	−80	28	0.20	−0.12
R Pre-central/Post-central Gyri[a] (BA 3/4)	1351	Positive	2.57	41	−21	50	0.17	−0.08
L Pre-central/Post-central Gyri[a] (BA 3/4)	1605	Positive	2.70	−20	−23	67	0.39*	−.20
M dACC/MeFG[a] (BA 6/24)	3354	Positive	2.70	−1	−10	51	0.13	−0.08
R Insula/Putamen[a] (BA 13)	1243	Positive	2.57	36	−17	7	0.26	−0.23
R Insula/OFC/IFG[b] (BA 13/47)	807	Positive	2.72	30	19	−17	0.18	0.11

Note. L = left. R = right. M = medial. BA = Brodmann's Area. IFG, MeFG, SFG = Inferior, medial and superior frontal gyrus. ITG, MTG and STG = Inferior, middle and superior temporal gyrus. IPL = Inferior parietal lobule. AG = Angular gyrus. SG = Supramarginal gyrus. OFC = Orbitofrontal cortex. dACC = Dorsal anterior cingulate cortex. Location = Coordinates are for the center-of-mass in MNI152 2009a symmetrical space. RT = Reaction time interference. Errors = Error interference. Correlations for RT and Errors are Pearson product-moment and Spearman rank-order correlations, respectively.
[a]Correction for all gray-matter voxels.
[b]Correction only for ventral prefrontal cortex gray matter voxels.
[c]Correction for only amygdala voxels. * = $P < 0.05$.

Moderation of neural activation by impulsive-antisociality

ImpAnti correlated positively with PosvNeu activation in six clusters and NegvNeu activation in six clusters (Table 1 and Figure 1). For PosvNeu, three clusters emerged in left temporal or temporal/parietal cortex, one in left SFG, one in medial and left frontal pole and one in left amygdala. For NegvNeu, two clusters emerged in bilateral pre-/post-central gyri, one in medial cuneus and one in right anterior insula/posterior OFC/IFG. In addition, one cluster emerged in posterior insula, extending into putamen, and another cluster in medial dorsal anterior cingulate (dACC)/medial frontal gyrus (MeFG).

Moderation of neural activation by fearless-dominance and impulsive-antisociality interaction

As shown in Table 2 and depicted in Figure 2, the interaction of FearDom and ImpAnti moderated activation in seven clusters. Three clusters emerged in left temporal and temporal/parietal cortex, with increased activation in these regions evident among individuals high in both ImpAnti and FearDom relative to one psychopathy dimension (Figure 2A). Similarly, two clusters in bilateral pre-/post-central gyri (Figure 2B) and one in rostral anterior cingulate cortex (rACC)/MeFG/SFG (Figure 2C) evidenced greater activation for individuals scoring high on both psychopathy dimensions relative to those scoring high on one dimension. One cluster also emerged in anterior, medial OFC/frontal pole, and depiction of the interaction in Figure 2D illustrates that activation in this cluster increased as scores on ImpAnti increased only among individuals who were low on FearDom. Interaction of FearDom and ImpAnti did not moderate PosvNeu activation.

Correlations between brain activation and behavior

As presented in Table 1, the right SFG cluster associated with FearDom exhibited a positive correlation with error interference for PosvNeu stimuli, suggesting that activation in this brain region increased as participants had more difficulty ignoring positive distractors. Activation in several brain regions related to ImpAnti correlated positively with reaction time to emotional *vs* neutral words, including clusters in temporal cortex, OFC, medial and left frontal pole, SFG, precentral and post-central gyri and left amygdala. With regard to the clusters associated with the FearDom × ImpAnti interaction, two out of the three clusters located in temporal/parietal cortices exhibited positive

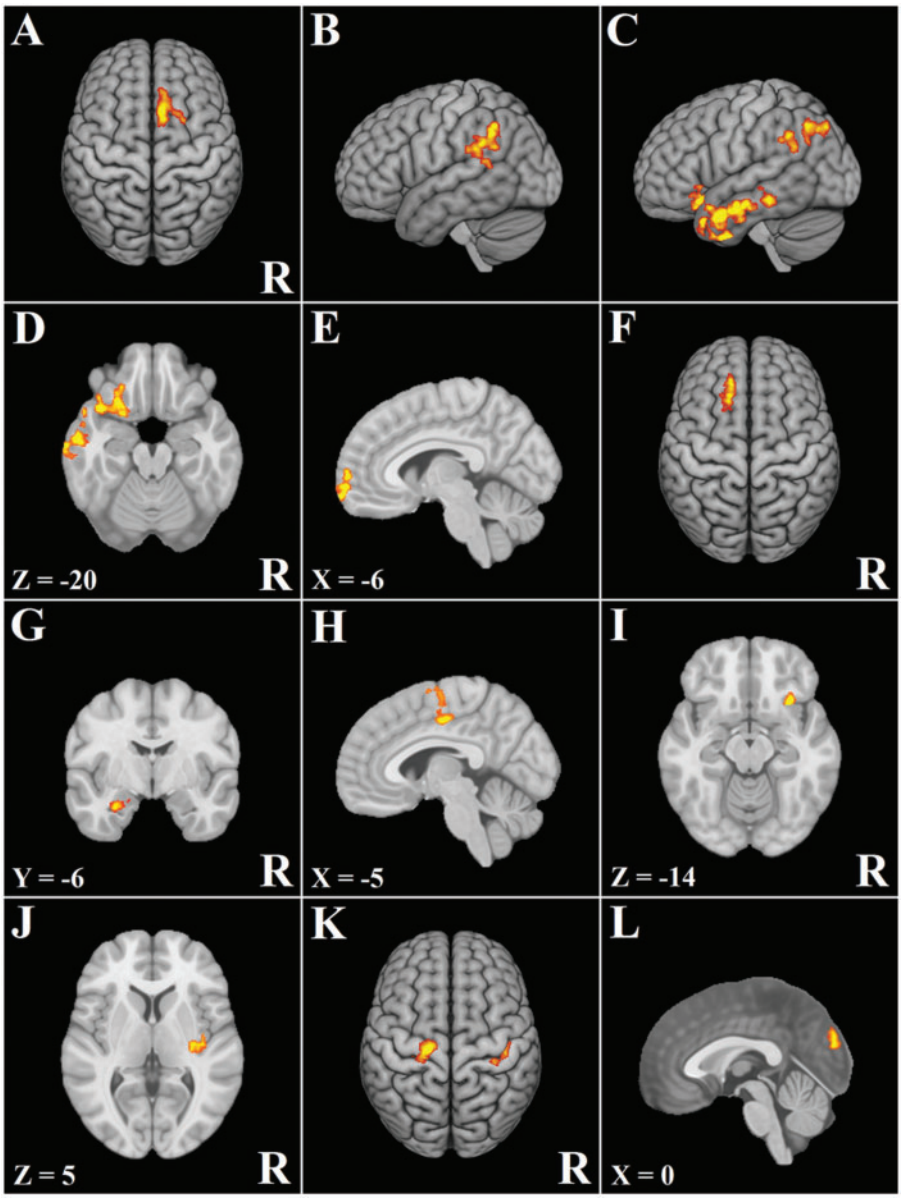

Fig. 1 Brain areas moderated by NEO-FFI estimated psychopathy dimensions. Red/yellow = positive correlation between brain activation (positive–neutral or negative–neutral) and psychopathy dimension (Fearless-Dominance or Impulsive-Antisociality). R = right. (**A**–**B**) Positive correlations between FearDom and PosvNeu activation. Clusters emerged in: (A) right superior frontal gyrus (SFG); and (B) left supramarginal gyrus/inferior parietal lobule (IPL). (**C**–**G**) Positive correlations between ImpAnti and PosvNeu activation. Clusters emerged in: (C) left temporal and temporal–parietal cortex; (D) left temporal cortex, extending into orbitofrontal cortex (OFC) and inferior frontal gyrus (IFG); (E) medial and lateral frontal pole; (F) left SFG; (G) left amygdala. (**H**–**L**) Positive correlations between ImpAnti and NegvNeu activation. Clusters emerged in: (H) dorsal anterior cingulate/medial frontal gyrus; (I) right insula/OFC/IFG; (J) right insula/putamen; (K) bilateral pre-central/post-central gyri; (L) medial cuneus.

correlations with RT interference to NegvNeu stimuli, as did the rACC/MeFG/SFG cluster (Table 2).

Mediation analyses

The right SFG cluster associated with FearDom mediated the effect of FearDom on PosvNeu errors ($ab = 0.17$, $P = 0.05$), and the indirect effect accounted for 36% of the total effect of FearDom on errors. Activation in two of the left temporal/parietal clusters for PosvNeu also mediated the relationship between ImpAnti and RT interference. First, the left temporal cluster that extended into OFC and IFG mediated the effect of ImpAnti on PosvNeu RT interference ($ab = 0.19$, $P = 0.02$), and the indirect effect accounted for 62% of the total effect of ImpAnti on RT. Second, the medial temporal gyrus (MTG)/superior temporal gyrus (STG)/IPL/SG cluster mediated the effect of ImpAnti on PosvNeu RT interference ($ab = 0.17$, $P = 0.02$), and the indirect effect accounted for 56% of the total effect of ImpAnti on RT. No other mediation analyses were significant at $P < 0.05$.

Table 2 Brain areas associated with the interaction of NEO-FFI estimated fearless-dominance and impulsive-antisociality

Region	Cluster size (mm^3)	Direction of relationship	Mean *z*-value	Location			RT	Errors
				x	*y*	*z*		
Negative *vs* neutral								
L MTG/STG[a] (BA 21/22)	2826	Positive	2.94	−61	−21	−10	0.47*	−0.17
L MTG/STG/SG[a] (BA 21/22/39)	1892	Positive	2.72	−53	−44	3	0.48*	−0.43*
L STG/IPL/SPL/SG/Precuneus[a] (BA 7/39/40)	3683	Positive	2.66	−41	−53	42	0.47*	−0.16
R Pre-central/Post-central Gyri[a] (BA 4/6/43)	1763	Positive	2.76	58	−7	21	0.24	−0.28
L Pre-central/Post-central Gyri[a] (BA 4/6/43)	2570	Positive	2.73	−51	−9	30	0.21	−0.31*
M rACC/MeFG/SFG[a] (BA 6/8/9/32)	2355	Positive	2.64	−3	35	33	0.46*	−0.04
M OFC/Frontal Pole[b] (BA 10/11)	700	Negative	−2.72	−2	64	−20	−0.05	0.04

Note. L = left. R = right. M = medial. BA = Brodmann's Area. MTG, STG = Middle and superior temporal gyrus. MeFG, SFG = Medial and superior frontal gyrus. IPL, SPL = Inferior and superior parietal lobule. SG = Supramarginal gyrus. OFC = Orbitofrontal cortex. rACC = Rostral anterior cingulate cortex. Location = Coordinates are for the center-of-mass in MNI152 2009a symmetrical space. RT = Reaction time interference. Errors = Error interference. Correlations for RT and Errors are Pearson product-moment and Spearman rank-order correlations, respectively.
[a]Correction for all gray-matter voxels.
[b]Correction only for ventral prefrontal cortex gray matter voxels; *$P < 0.05$.

DISCUSSION

The present study investigated whether dimensions of psychopathic personality traits moderate behavioral responses and neural activation associated with selective attention in the context of emotional distractors. As predicted, the fearless-dominance and impulsive-antisociality dimensions evidenced distinct patterns of behavioral deficits and neural responses, consistent with the hypothesis that they represent separable constructs associated with differential deficits. Moreover, present findings suggest that the effects of the psychopathy dimensions are interactive, rather than simply additive, as individuals high in both fearless-dominance and impulsive-antisociality exhibited unique neural correlates. In combination, the results document heterogeneity in selective-attention deficits and sensitivity to emotional distraction associated with the psychopathy dimensions, and they illustrate the importance of specifying neurobiological processes associated with their independent and interactive effects.

Fearless-dominance

In both behavioral and neural measures, fearless-dominance was associated with an enhanced sensitivity to positive stimuli. Analysis of error rates revealed a selective deficit in suppressing the effects of positive distractor words on behavior among individuals high in fearless-dominance. In addition, fearless-dominance moderated activation to positive *vs* neutral words in left SG/IPL, a region linked to the maintenance of task sets (e.g. Dosenbach *et al.*, 2006), and a cluster in right SFG associated with the commission of errors and the receipt of negative or unexpected feedback during task performance (Akitsuki *et al.*, 2003; Fiehler *et al.*, 2004; Taylor *et al.*, 2006; Nieuwenhuis *et al.*, 2007; Wittfoth *et al.*, 2008). The finding that activation in the right SFG cluster, a region important for processing mismatches between anticipated and actual task performance, mediated the relationship between fearless-dominance and errors to positive *vs* neutral stimuli suggests that this region was recruited to increase awareness of ineffective responding and compensate for impaired attentional control induced by positive words among individuals high in fearless-dominance. The heightened attention to positive stimuli observed among individuals high on fearless-dominance may help explain findings that the affective-interpersonal features of psychopathy are associated with resilience against some forms of psychopathology, including symptoms of depression and anxiety (e.g. Benning *et al.*, 2005). Contrary to predictions, the negative relationship between fearless-dominance and emotional reactivity to negative stimuli did not emerge.

Impulsive-antisociality

In contrast to fearless-dominance, impulsive-antisociality was associated with impaired attentional control (slowed reaction time) in the context of both positive and negative stimuli (relative to neutral), as predicted. This finding is consistent with the hypothesis that individuals high in impulsive-antisociality are particularly sensitive to emotional context and that this sensitivity contributes to their behavioral dysregulation and poor cognitive control (Buckholtz *et al.*, 2010; Sprague and Verona, 2010).

The patterns of neural activation for emotional *vs* neutral stimuli associated with impulsive-antisociality were consistent with the heightened sensitivity to emotion and associated behavioral dysregulation apparent in the behavioral findings. Impulsive-antisociality correlated positively with activation in regions that instantiate processing of the emotional and motivational value of stimuli, specifically portions of left amygdala, left posterior/agranular OFC and right agranular insula/OFC (Bechara *et al.*, 2000; Anderson and Phelps, 2001; Craig, 2002; O'Doherty and Dolan, 2006;

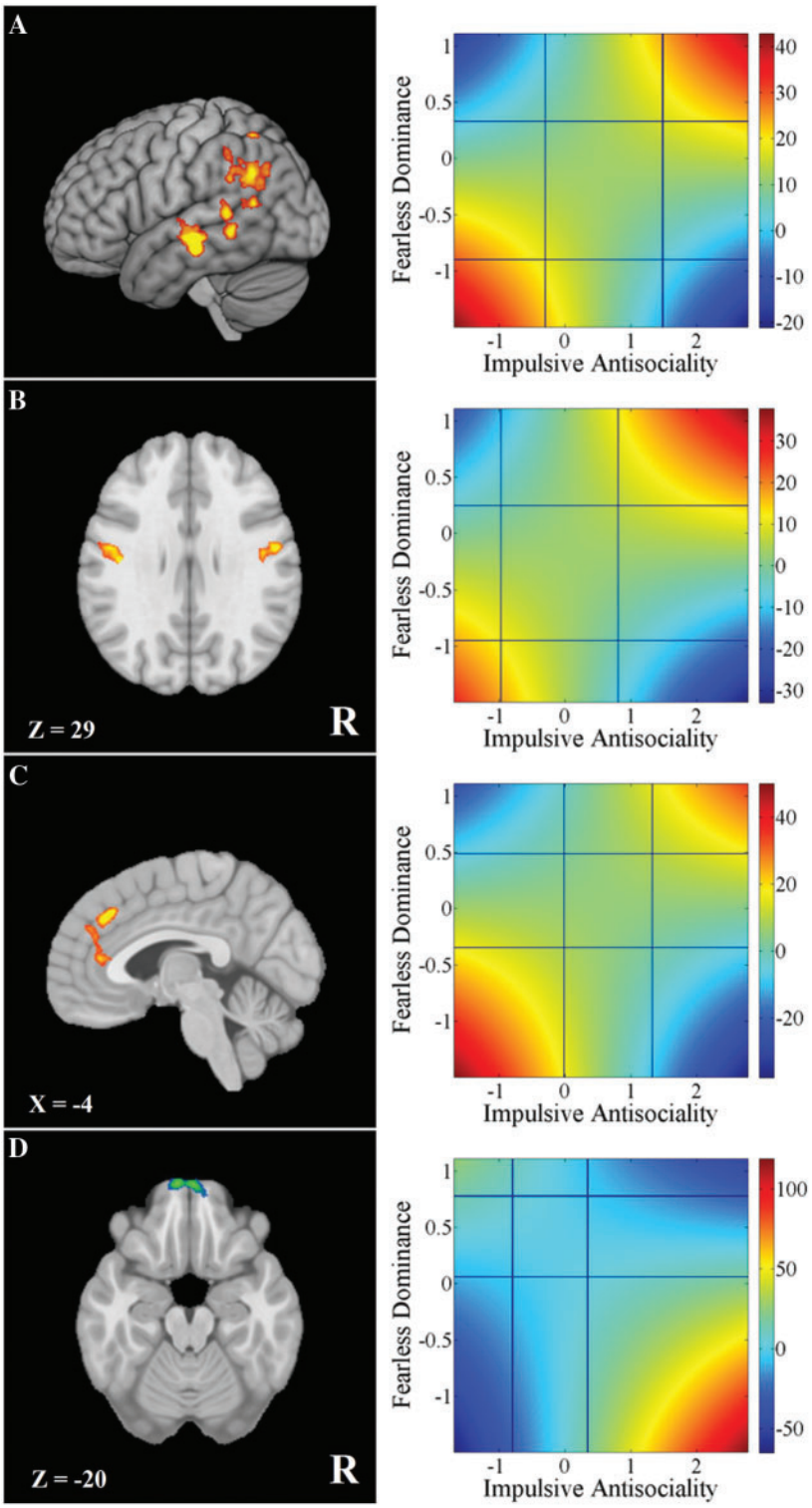

Fig. 2 Interaction of fearless-dominance and impulsive-antisociality for negative *vs* neutral words. For each row, the image on the left shows the location of the cluster showing a significant interaction of the two dimensions of psychopathy, and the panel on the right is a graph of brain activation (red = increased activation, blue = decreased activation) in that cluster as a function of scores on Fearless-Dominance (FearDom; *y*-axis) and Impulsive–Antisociality (ImpAnti; *x*-axis; the ranges of both axes are determined by the sample min and max). The horizontal and vertical grid lines in the graphs represent regions of significance. Thus, the relationship for FearDom is significant for all values of ImpAnti to the left of the leftmost vertical line and to the right of the rightmost vertical line. Similarly, the relationship for ImpAnti is significant for all values of FearDom above the higher horizontal line and below the lower horizontal line. For the images on the left, red/yellow = more activation when both dimensions are high than when only one dimension is high, and blue/green = more activation when one dimension is high than when both dimensions are high. R = Right. (**A**) Activation in the three left temporal/temporal-parietal clusters. The right panel depicts activation in the medial temporal gyrus/superior temporal gyrus cluster and is representative of the graphs for the other two clusters. (**B**) Activation in bilateral pre-central/post-central gyri. The right panel depicts activation in the left cluster and is representative of the right cluster. (**C**) Activation in rostral anterior cingulate/medial frontal gyrus/superior frontal gyrus. (**D**) Activation in medial orbitofrontal cortex/frontal pole.

Craig, 2009). Research suggests that amygdala is critical for evaluating the emotional salience of stimuli (e.g. Anderson and Phelps, 2001; Phan *et al.*, 2004), and the present finding of increased amygdala activation for impulsive-antisociality is consistent with both previous research (e.g. Gordon *et al.*, 2004) and theoretical models (e.g. Blair, 2010) positing that amygdala reactivity is associated with inappropriately strong responses to emotionally valenced stimuli that lead to reactive aggression. Research implicates agranular OFC and anterior insula in instantiating interoceptive feeling states that code for both outcome expectations and the level of uncertainty associated with those expectations (Singer *et al.*, 2009). Thus, increased activation in these areas suggests that individuals high on impulsive-antisociality are attaching greater predictive significance to the emotional words and/or have greater uncertainty about the significance of the stimuli.

In conjunction with emotional hypersensitivity, individuals high in impulsive-antisociality demonstrated increased activation in regions related to emotion regulation, cognitive control and attentional engagement during the processing of emotional distractors. First, impulsive-antisociality correlated positively with activation in two clusters in temporal cortex and one in right SFG that research suggests are important for appraising the emotional significance of stimuli (Cunningham *et al.*, 2004; Bickel *et al.*, 2009) and regulating emotion during decision-making (Harenski and Hamann, 2006; Iaria *et al.*, 2008). Although some research examining psychopathy as a unitary construct has found decreased activation in similar regions (e.g. STG, IFG; Kiehl *et al.*, 2001), the present findings are consistent with research showing excessive, rather than blunted, emotional reactivity in impulsive and antisocial individuals (Benning *et al.*, 2003; Patrick *et al.*, 2006; Cima and Raine, 2009). Second, impulsive-antisociality correlated positively with activation in areas of temporal and parietal cortex associated with the initiation and maintenance of task sets (Dosenbach *et al.*, 2006; Iaria *et al.*, 2008), and a cluster in medial frontal pole associated with directing attention toward externally generated information, as opposed to internal experiences (e.g. Burgess *et al.*, 2007). Increased recruitment of these regions by individuals high in impulsive-antisociality may represent an effort to maintain task goals by directing attention outward to ignore the emotional experience induced by the words. Impulsive-antisociality was also associated with increased activation in regions related to response preparation and motor planning, including one cluster that includes a portion of dACC (Winterer *et al.*, 2002; Ramnani and Miall, 2003; Beckmann *et al.*, 2009). These findings are consistent with the hypothesis that individuals high in impulsive-antisociality exerted extra attentional resources to decrease the effects of the emotional distractors.

In combination, the behavioral and fMRI results indicate that individuals high in impulsive-antisociality display heightened sensitivity to emotional stimuli that impaired their ability to maintain attentional control. Not only was this evident in slowing of behavioral responses to emotional *vs* neutral words, it was apparent in increased activation in regions associated with evaluating emotional and motivational significance, maintaining task goals and preparing behavioral responses. Analysis of brain–behavior relationships revealed that several of these regions were associated with response interference on the task, with two clusters in the left temporal/parietal cortices mediating the association between impulsive-antisociality and impaired behavioral performance (slowed reaction time). Thus, the emotion regulation and goal maintenance processes related to activation in these regions appear to be particularly relevant to the selective-attention deficits associated with impulsive-antisociality.

Interactive effects of fearless-dominance and impulsive-antisociality

The neural activation moderated by the interaction of the two psychopathy dimensions indicated that selective attention in individuals high on both dimensions was affected by the presence of negative words. In three clusters in left temporal cortex areas implicated in appraisal and reappraisal of emotional stimuli (Cunningham *et al.*, 2004; Harenski and Hamann, 2006; Goldin *et al.*, 2008), individuals high on both psychopathy dimensions displayed more activation than individuals high on only one dimension. This suggests that individuals high on both psychopathy dimensions recruited more resources to evaluate, or possibly reappraise, the emotional relevance of negative words. One of these clusters extended into inferior and superior parietal lobules and included a region associated with the initiation and maintenance of task sets (Dosenbach *et al.*, 2006).They also recruited regions associated with motor planning and response preparation, including bilateral pre-/post-central gyri (Krams *et al.*, 1998; Toni *et al.*, 2002), and displayed heightened activation in a region of rostral anterior cingulate cortex involved in cognitive-control processes, including prepotent response regulation, response inhibition and conflict detection (Van Veen *et al.*, 2001; Chikazoe *et al.*, 2007), that differed slightly from those found for the main effect of impulsive-antisociality. These findings suggest that individuals high on both psychopathy dimensions found the negative words to be particularly distracting and recruited additional attentional resources to compensate for the effects of distractors on performance. However, this interpretation requires further investigation to interpret the functional significance of the increased recruitment of attentional resources to negative stimuli among individuals high on both psychopathy dimensions, as it may reflect efforts to suppress the effects of emotional salience on performance or a different compensatory process (e.g. difficulty interpreting the emotional significance of the words).

Finally, the interaction of the psychopathy dimensions showed a unique relationship to activation in a cluster in anterior, medial OFC. As depicted in Figure 2D, elevated

activation emerged in this region for individuals high in impulsive-antisociality, which diminished with increasing levels of fearless-dominance. This suggests that individuals high on both psychopathy dimensions, along with those high only in fearless-dominance, assigned less motivational significance to the negative words than did individuals high only in impulsive-antisociality. Decreased activation in this area in individuals high on both psychopathy dimensions is consistent with findings that psychopaths display reduced processing in OFC during fear conditioning (Birbaumer *et al.*, 2005).

In summary, results revealed that individuals high on both dimensions recruited regions in temporal cortex to appraise the meaning of negative words and maintain attentional control in the presence of these stimuli. In addition, these individuals showed decreased activation in an area of OFC related to the maintenance of motivational value. These findings are consistent with the hypothesis that high overall psychopathy is associated with impairment in regions involved in the evaluation and maintenance of motivational significance for negative stimuli (e.g. Mitchell *et al.*, 2002; Birbaumer *et al.*, 2005). They also replicate studies that find increased activation in temporal regions in response to emotional stimuli among individuals high on total psychopathy (Kiehl *et al.*, 2001; Soderstrom *et al.*, 2002; Muller *et al.*, 2003).

This study benefited from a relatively large sample for an fMRI study, particularly one examining neural processes associated with psychopathy. It extends the literature on neurobiological processes associated with psychopathy by examining selective attention and emotional processes associated with two psychopathy dimensions. One limitation is the estimation of psychopathy from an index of general personality. Although this provides a needed link between the deficits observed in forensic studies of psychopathy and those emerging in less pathological samples, this approach may not adequately capture certain aspects of the disorder (e.g. extreme antisocial behavior) and processing deficits associated with them. Second, there is evidence that the cognitive deficits associated with fearless-dominance are primarily related to early selective attention (Newman *et al.*, 2010; Baskin-Sommers *et al.*, 2011), which was not separately manipulated in the present task. Thus, neural processes associated with what appears to be one important cognitive deficit associated with fearless-dominance were not thoroughly examined. Third, oversampling individuals with extreme scores on the psychopathy dimensions would allow for direct comparisons of the deficits that emerge using dimensional *vs* extreme group analyses, a test that is not possible with the present study design. Fourth, although present results suggest the affective word stimuli used were sufficiently distracting to affect behavior and neural activity, the generalizability of these findings to more emotionally evocative stimuli (e.g. aversive pictures, electric shocks) is a question for future research. Finally, future work would benefit from including external criterion measures and examining how differences in neural activity relate to clinically meaningful outcomes associated with psychopathy (e.g. criminal behavior, aggression, comorbid psychopathology).

Despite these limitations, this study provides new evidence that the psychopathy dimensions differentially moderate neural processes associated with selective attention and emotional processing. Overall, findings further specify the neurobiological processes associated with features of psychopathy and suggest that fearless-dominance, impulsive-antisociality and their interaction are characterized by distinct sets of cognitive and affective risk factors.

REFERENCES

Akitsuki, Y., Sugiura, M., Watanabe, J., et al. (2003). Context-dependent cortical activation in response to financial reward and penalty: an event-related fMRI study. *NeuroImage*, *19*, 1674–85.

Anderson, A.K., Phelps, E.A. (2001). Lesions of the human amygdala impair enhanced perception of emotionally salient events. *Nature*, *411*, 305–9.

Baskin-Sommers, A.R., Curtin, J.J., Newman, J.P. (2011). Specifying the attentional selection that moderates the fearlessness of psychopathic offenders. *Psychological Science*, *22*, 226–34.

Bechara, A., Damasio, H., Damasio, A.R. (2000). Emotion, decision making and the orbitofrontal cortex. *Cerebral Cortex*, *10*, 295–307.

Beckmann, M., Johansen-Berg, H., Rushworth, M. (2009). Connectivity-based parcellation of human cingulate cortex and its relation to functional specialization. *Journal of Neuroscience*, *29*, 1175–90.

Benning, S.D., Patrick, C.J., Blonigen, D.M., Hicks, B.M., Iacono, W.G. (2005). Estimating facets of psychopathy from normal personality traits: a step toward community-epidemiological investigations. *Assessment*, *12*, 3–18.

Benning, S.D., Patrick, C.J., Hicks, B.M., Blonigen, D.M., Krueger, R.F. (2003). Factor structure of the Psychopathic Personality Inventory: validity and implications for clinical assessment. *Psychological Assessment*, *15*, 340–50.

Benning, S.D., Patrick, C.J., Iacono, W.G. (2005). Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. *Psychophysiology*, *42*, 753–62.

Bickel, W.K., Pitcock, J.A., Yi, R., Angtuaco, E.J. (2009). Congruence of BOLD response across intertemporal choice conditions: fictive and real money gains and losses. *Journal of Neuroscience*, *29*, 8839–46.

Birbaumer, N., Veit, R., Lotze, M., et al. (2005). Deficient fear conditioning in psychopathy: a functional magnetic resonance imaging study. *Archives of General Psychiatry*, *62*, 799–805.

Blair, R.J.R. (2003). Neurobiological basis of psychopathy. *British Journal of Psychiatry*, *182*, 5–7.

Blair, R.J.R. (2010). Neuroimaging of psychopathy and antisocial behavior: a targeted review. *Current Psychiatry Reports*, *12*, 76–82.

Bradley, M.M., Lang, P.J. (1998). *Affective norms for English words (ANEW)*. Gainesville, FL: University of Florida, NIMH Center for the Study of Emotion and Attention.

Buckholtz, J.W., Treadway, M.T., Cowan, R., et al. (2010). Mesolimbic dopamine reward system hypersensitivity in individuals with psychopathic traits. *Nature Neuroscience*, *13*, 419–21.

Burgess, P.W., Dumontheil, I., Gilbert, S.J. (2007). The gateway hypothesis of rostral prefrontal cortex (area 10) function. *Trends in Cognitive Sciences*, *11*, 290–8.

Chikazoe, J., Konishi, S., Asari, T., Jimura, K., Miyashita, Y. (2007). Activation of right inferior frontal gyrus during response inhibition across response modalities. *Journal of Cognitive Neuroscience*, *19*, 69–80.

Cima, M., Raine, A. (2009). Distinct characteristics of psychopathy relate to different subtypes of aggression. *Personality and Individual Differences*, *47*, 835–40.

Costa, P.T., McCrae, R.R. (1992). *Revised NEO personality inventory (NEOPI-R and Five Factor Inventory (NEO-FFI) professional manual*. Odessa, FL: Psychological Assessment Resources.

Craig, A.D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Reviews Neuroscience*, *3*, 655–66.

Craig, A.D. (2009). How do you feel – now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, *10*, 59–70.

Cunningham, W.A., Raye, C.L., Johnson, M.K. (2004). Implicit and explicit evaluation: fMRI correlates of valence, emotional intensity, and control in the processing of attitudes. *Journal of Cognitive Neuroscience*, *16*, 1717–29.

Deeley, Q., Daly, E., Surguladze, S., et al. (2006). Facial emotion processing in criminal psychopathy: preliminary functional magnetic resonance imaging study. *The British Journal of Psychiatry*, *189*, 533–9.

Dosenbach, N.U.F., Visscher, K.M., Palmer, E.D., et al. (2006). A core system for the implementation of task sets. *Neuron*, *50*, 799–812.

Edens, J.F., Marcus, D.K., Lilienfeld, S.O., Poythress, N.G. (2006). Psychopathic, not psychopath: taxometric evidence for the dimensional structure of psychopathy. *Journal of Abnormal Psychology*, *115*, 131–44.

Fiehler, K., Ullsperger, M., von Cramon, D.Y. (2004). Neural correlates of error detection and error correction: is there a common neuroanatomical substrate? *European Journal of Neuroscience*, *19*, 3081–7.

Finger, E.C., Marsh, A.A., Mitchell, D.G., et al. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives of General Psychiatry*, *65*, 586–94.

First, M.B., Spitzer, R.L., Gibbon, M., Williams, J.B.W. (2002). *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-patient Edition*. New York: Biometrics Research, New York State Psychiatric Institute.

Fonov, V.S., Evans, A.C., McKinstry, R.C., Almli, C.R., Collins, D.L. (2009). Unbiased nonlinear average age-appropriate brain templates from birth to adulthood. *NeuroImage*, *47*, S102.

Fowles, D.C., Dindo, L. (2009). Temperament and psychopathy: w dual-pathway model. *Current Directions in Psychological Science*, *18*, 179–83.

Goldin, P.R., McRae, K., Ramel, W., Gross, J.J. (2008). The neural bases of emotion regulation: reappraisal and suppression of negative emotion. *Biological Psychiatry*, *63*, 577–86.

Gordon, H.L., Baird, A.A., End, A. (2004). Functional differences among those high and low on a trait measure of psychopathy. *Biological Psychiatry*, *7*, 516–21.

Hare, R.D., Clark, D., Grann, M., Thornton, D. (2000). Psychopathy and the predictive validity of the PCL-R: an international perspective. *Behavioral Sciences & the Law*, *18*, 623–45.

Harenski, C.L., Hamann, S. (2006). Neural correlates of regulating negative emotions related to moral violations. *NeuroImage*, *30*, 313–24.

Harpur, T.J., Hakstian, A.R., Hare, R.D. (1988). Factor structure of the Psychopathy Checklist. *Journal of Consulting and Clinical Psychology*, *56*, 741–7.

Harpur, T., Hare, R.D., Hakstian, A. (1989). Two-factor conceptualization of psychopathy: construct validity and assessment implications. *Psychological Assessment*, *1*, 6–17.

Iaria, G., Committeri, G., Pastorelli, C., Pizzamiglio, L., Watkins, K.E., Carota, A. (2008). Neural activity of the anterior insula in emotional processing depends on the individuals' emotional susceptibility. *Human Brain Mapping*, *29*, 363–73.

Jenkinson, M., Bannister, P., Brady, M., Smith, S. (2002). Improved optimization for the robust and accurate linear registration and motion correction of brain images. *NeuroImage*, *17*, 825–41.

Kiehl, K.A., Smith, A.M., Hare, R.D., et al. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Biological Psychiatry*, *50*, 677–84.

Krams, M., Rushworth, M.F.S., Deiber, M.P., Frackowiak, R.S.J., Passingham, R.E. (1998). The preparation, execution and suppression of copied movements in the human brain. *Experimental Brain Research*, *120*, 386–98.

MacKinnon, D.P. (2008). *Introduction to statistical mediation analysis*. NY: Psychology Press.

Marsh, A.A., Finger, E.C., Mitchell, D.G.V., et al. (2008). Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. *American Journal of Psychiatry*, *165*, 712–20.

Miller, G.A., Chapman, J.P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology*, *110*, 40–8.

Miller, J.D., Lynam, D.R., Widiger, T.A., Leukefeld, C. (2001). Personality disorders as extreme variants of common personality dimensions: can the Five-Factor Model adequately represent psychopathy? *Journal of Personality*, *69*, 253–76.

Muller, J.L., Sommer, M., Dohnel, K., Weber, T., Schmidt-Wilcke, T., Hajak, G. (2008). Disturbed prefrontal and temporal brain function during emotion and cognition interaction in criminal psychopathy. *Behavioral Sciences & the Law*, *26*, 131–50.

Muller, J.L., Sommer, M., Wagner, V., et al. (2003). Abnormalities in emotion processing within cortical and subcortical regions in criminal psychopaths: evidence from a functional magnetic resonance imaging study using pictures with emotional content. *Biological Psychiatry*, *54*, 152–62.

Murrie, D.C., Marcus, D.K., Douglas, K.S., Lee, Z., Salekin, R.T., Vincent, G. (2007). Youth with psychopathy features are not a discrete class: a taxometric analysis. *Journal of Child Psychology and Psychiatry*, *48*, 714–23.

Mitchell, D.G., Colledge, E., Leonard, A., Blair, R.J. (2002). Risky decisions and response reversal: Is there evidence of orbitofrontal cortex dysfunction in psychopathic individuals? *Neuropsychologia*, *40*, 2013–22.

Newman, J.P., Curtin, J.J., Bertsch, J.D., Baskin-Sommers, A. (2010). Attention moderates the fearlessness of psychopathic offenders. *Biological Psychiatry*, *67*, 66–70.

Nieuwenhuis, S., Schweizer, T.S., Mars, R.B., Botvinick, M.M., Hajcak, G. (2007). Error-likelihood prediction in the medial frontal cortex: a critical evaluation. *Cerebral Cortex*, *17*, 1570–81.

O'Doherty, J.P., Dolan, R.J. (2006). The role of human orbitofrontal cortex in reward prediction and behavioral choice: insights from neuroimaging. In: Zald, D.H., Rauch, S.L., editors. *The Orbitofrontal Cortex*. Oxford, UK: Oxford University Press, pp. 265–84.

Patrick, C.J., Bernat, E.M. (2009). Neurobiology of psychopathy: a two-process theory. In: Berntson, G.G., Cacioppo, J.T., editors. *Handbook of Neuroscience for the Behavioral Sciences*. New York: John Wiley & Sons, pp. 1110–31.

Patrick, C.J., Bradley, M.M., Lang, P.J. (1993). Emotion in the criminal psychopath: startle reflex modulation. *Journal of Abnormal Psychology*, *102*, 82–92.

Patrick, C.J., Cuthbert, B.N., Lang, P.J. (1994). Emotion in the criminal psychopath: fear image processing. *Journal of Abnormal Psychology*, *103*, 523–34.

Patrick, C.J., Poythress, N.G., Edens, J.F., Lilienfeld, S.O., Benning, S.D. (2006). Construct validity of the psychopathic personality inventory two-factor model with offenders. *Psychological Assessment*, *18*, 204–8.

Patrick, C.J., Zempolich, K.A. (1998). Emotion and aggression in the psychopathic personality. *Aggression and Violent Behavior*, *3*, 303–38.

Raine, A., Meloy, R., Bihrle, S., Stoddard, J., LaCasse, L., Buchsbaum, M. (1998). Reduced prefrontal and increased subcortical brain functioning assessed using positron emission tomography in predatory and affective murders. *Behavioral Sciences & Law*, *16*, 319–32.

Ramnani, N., Miall, R.C. (2003). Instructed delay activity in the human prefrontal cortex is modulated by monetary reward expectation. *Cerebral Cortex*, *13*, 318–27.

Rilling, J.K., Glenn, A.L., Jairam, M.R., et al. (2007). Neural correlates of social cooperation and non-cooperation as a function of psychopathy. *Biological Psychiatry*, *61*, 1260–71.

Ross, S.R., Benning, S.D., Patrick, C.J., Thompson, A., Thurston, A. (2009). Factors of the Psychopathic Personality Inventory: criterion-related validity and relationship to the BIS/BAS and five-factor models of personality. *Assessment*, *16*, 71–87.

Sadeh, N., Verona, E. (2008). Psychopathic traits associated with abnormal selective attention and impaired cognitive control. *Neuropsychology*, *22*, 669–80.

Sellbom, M., Verona, E. (2007). Neuropsychological correlates of psychopathic traits in a non-incarcerated sample. *Journal of Research in Personality*, *41*, 276–94.

Singer, T., Critchley, H.D., Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends in Cognitive Sciences*, *13*, 334–40.

Sobel, M.E. (1982). Asymptotic confidence intervals for indirect effects in structural equation models. *Sociological Methodology*, *13*, 290–312.

Soderstrom, H., Hultin, L., Tullberg, M., Wikkelso, C., Ekholm, S., Forsman, A. (2002). Reduced frontotemporal perfusion in psychopathic personality. *Psychiatry Research Imaging*, *114*, 81–94.

Sprague, J., Verona, E. (2010). Emotional conditions disrupt behavioral control among individuals with dysregulated personality traits. *Journal of Abnormal Psychology*, *119*, 409–19.

Taylor, S.F., Martis, B., Fitzgerald, K.D., et al. (2006). Medial frontal cortex activity and loss-related responses to errors. *The Journal of Neuroscience*, *26*, 4063–70.

Toni, I., Shah, N.J., Fink, G.R., Theonissen, D., Passingham, P.E., Zilles, K. (2002). Multiple movement representations in the human brain: an event-related fMRI study. *Journal of Cognitive Neuroscience*, *14*, 769–84.

Van Veen, V., Cohen, J.D., Botvinick, M.M., Stenger, V.A., Carter, C.S. (2001). Anterior cingulate cortex, conflict monitoring, and levels of processing. *NeuroImage*, *14*, 1302–8.

Ward, D.B. (2000). Simultaneous inference for FMRI data. http://afni.nimh.nih.gov./pub/dist/doc/manual/AlphaSim.pdf.

Widiger, T.A., Lynam, D.R. (1998). Psychopathy and the five-factor model of personality. In: Millon, T., editor. *Psychopathy: Antisocial, Criminal, and Violent Behavior*. New York: Guilford, pp. 171–87.

Williams, J.M., Mathews, A., MacLeod, C. (1996). The emotion-word Stroop task and psychopathology. *Psychological Bulletin*, *120*, 3–24.

Winterer, G., Adams, C.M., Jones, D.W., Knutson, B. (2002). Volition to action – an event-related fMRI study. *NeuroImage*, *17*, 851–8.

Wittfoth, M., Kustermann, E., Fahle, M., Herrmann, M. (2008). The influence of response conflict on error processing: evidence from event-related fMRI. *Brain Research*, *1194*, 118–29.

Woolrich, M. (2008). Robust group analysis using outlier inference. *NeuroImage*, *41*, 286–301.

Woolrich, M., Ripley, B., Brady, M., Smith, S. (2001). Temporal autocorrelation in univariate linear modeling of FMRI data. *NeuroImage*, *14*, 1370–86.