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Selective Mapping of Psychopathy and Externalizing to Dissociable Circuits for Inhibitory Self-Control

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Abstract

Antisociality is commonly conceptualized as a unitary construct, but there is considerable evidence for multidimensionality. In particular, two partially dissociable symptom clusters—psychopathy and externalizing—have divergent associations to clinical and forensic outcomes and are linked to unique patterns executive dysfunction. Here, we used fMRI in a sample of incarcerated offenders to map these dimensions of antisocial behavior to brain circuits underlying two aspects of inhibitory self-control: interference suppression and response inhibition. We found that psychopathy and externalizing are characterized by unique and task-selective patterns of dysfunction. Although higher levels of psychopathy predicted increased activity within a distributed frontoparietal network for interference suppression, externalizing did not predict brain activity during attentional control. By contrast, each dimension had opposite associations to frontoparietal activity during response inhibition. These findings provide neurobiological evidence supporting the fractionation of antisocial behavior and identify dissociable mechanisms through which different facets predispose dysfunction and impairment.

Keywords

psychopathy, externalizing, self-control, impulsivity, fMRI

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Antisocial behavior is characterized by a persistent pattern of social, legal, and moral norm transgression, including high levels of criminal offending. Recent estimates suggest that the annual cost of criminal behavior may reach as high as $3.3 trillion per annum in the United States (converted into 2015 dollars; Anderson, 1999). Despite the significance of antisocial behavior as a driver of costly criminal offending, we still know relatively little about its underlying cognitive and neurobiological mechanisms. This is due, in part, to a failure to distinguish between distinct, antisocial syndromes. Although antisocial behavior is commonly conceptualized in terms of antisocial personality disorder (APD), many have argued that the diagnostic criteria for APD do not account for the rather evident heterogeneity that exists within this clinical population (Edens, Kelley, Lilienfeld, Skeem, & Douglas, 2015; Moffitt, 1993; Poythress et al., 2010; Skeem & Cooke, 2010; Skeem, Polaschek, Patrick, & Lilienfeld, 2011; Venables & Patrick, 2012). In particular, at least two partially dissociable dimensions—externalizing and psychopathy—are thought to be nested within the superordinate construct of antisocial behavior (Edens et al., 2015; Edens, Poythress, Lilienfeld, Patrick, & Test, 2008; Frick & Viding, 2009; Krueger et al., 2002; Krueger, Markon,

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Patrick, Benning, & Kramer, 2007; Moffitt, 1993; Poythress et al., 2010; Skeem et al., 2011; Venables & Patrick, 2012).

Externalizing can be conceptualized as a normally distributed latent trait that accounts for the comorbidity among multiple syndromes linked to antisocial behavior, such as attention-deficit/hyperactivity disorder (ADHD), conduct disorder (in adolescents), APD (in adults), and substance abuse (Krueger et al., 2002; Krueger et al., 2007; Krueger, Markon, Patrick, & Iacono, 2005; Patrick et al., 2013). In turn, heritability studies indicate that symptom covariance among these syndromes is driven by a common genetic liability factor. This supports the notion that externalizing reflects a symptomatically unified and etiologically coherent dimension typified by disinhibition (e.g., impulsivity) and negative affect (e.g., reactive aggression; Krueger et al., 2002; Krueger et al., 2005; Krueger et al., 2007; Patrick et al., 2013).

By contrast, psychopathy encompasses aspects of socioaffective function that distinguish it from externalizing. Cleckley's original characterization of psychopathy centered on three cardinal facets: positive adjustment (low anxiety and neuroticism; superficial charm), behavioral deviance (inadequately motivated antisocial behavior; irresponsibility), and emotional-interpersonal deficits (lack of remorse, empathy, and shame; shallow affect; Cleckley, 1988; Patrick, 2006; Skeem et al., 2011). Modern conceptualizations of psychopathy have largely retained these features; interpersonal (e.g., manipulation, pathological lying) and affective (e.g., callousness, diminished empathy) deficits are considered central for defining psychopathy, along with lifestyle and antisocial symptoms (but see Skeem & Cooke, 2010).

Externalizing and psychopathy are dissociable at multiple levels of analysis. Compared with externalizing, psychopathy is associated with more severe, stable, and violent forms of antisocial behavior in both youth and adults (Blair, 2013; Frick, 2009; Raine, 2002). Distinct patterns of comorbidity have been reported as well. Although anxious and depressive symptoms are relatively common concomitants of externalizing, the oft-noted absence of such features in psychopathy has led some to suggest that it acts as a protective factor against mood and anxiety psychopathology (Willemsen, Vanheule, & Verhaeghe, 2011). Genetic data provide further evidence for the distinctiveness of these two dimensions. Although both externalizing and psychopathy show evidence of moderate to high heritability, heritability magnitude estimates vary according to the presence or absence of the affective-interpersonal personality features (e.g., callous-unemotional traits) that are core to psychopathy (Viding, Jones, Frick, Moffitt, & Plomin, 2008). Differential heritability implies the existence of dissociable genetic architectures for each dimension (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005) and, in turn, distinct etiological origins for the characteristic symptoms of each.

The clinical and genetic data cited earlier support the notion that externalizing and psychopathy represent distinct antisocial syndromes, and imply the existence of dimension-specific cognitive and neurobiological mechanisms that predispose a common behavioral endpoint (antisocial behavior). However, the identification of dimension-selective mechanisms has proved challenging. Studies of antisocial behavior commonly rely on one measure, often the Psychopathy Checklist–Revised (PCL-R; Hare, 2006). Inferences about dimensional selectivity are gleaned by examining phenotypic associations with the measure's principal subscales (commonly referred to as "factors"). Factor 1 indexes the emotional and interpersonal symptoms that many consider core to the construct, whereas Factor 2 captures behaviors that align more with the externalizing dimension noted earlier, such as impulsivity, irresponsibility and aggression. Despite being labeled as factors, Factor 1 and Factor 2 exhibit a modest positive correlation (typically ~.5–.6; Hare & Neumann, 2008). Consistent with the notion that PCL-R Factor 2 accesses the externalizing dimension, modest correlations between Factor 2 and scores from the Externalizing Spectrum Inventory (ESI; Venables & Patrick, 2012) have been reported. Furthermore, these correlations are significantly stronger than the association between Factor 1 and ESI scores (Patrick et al., 2013; Venables & Patrick, 2012). On the whole, this pattern of covariance suggests that commonly used clinical assessments of antisocial behavior are relatively nonselective. This situation limits the specificity of inference when such measures are used as predictors of cognitive and neurobiological phenotypes, as it is unclear whether significant associations are driven by shared variance between psychopathy and externalizing or due to the unique variance associated with either dimension.

Notwithstanding the methodological confound noted earlier, relatively consistent evidence for dimension-specific mechanisms can be gleaned from studies of executive function (EF). Although executive dysfunction has long been noted in antisocial individuals (Dolan, 2012; Dolan & Park, 2002; Morgan & Lilienfeld, 2000), recent work suggests that externalizing and psychopathy are associated with distinct patterns of EF deficits, particularly in the domain of selective attention. Research to date suggests that externalizing individuals have deficits in multiple domains of EF, including, selective attention, interference suppression, and response inhibition. By contrast, many of these EF subcomponents appear to be preserved, and in some cases enhanced, in psychopathy. For example, although externalizing predicts larger "attentional blinks" in a rapid serial visual presentation task (Baskin-Sommers, Wolf, Buckholtz, Warren, &

Newman, 2012), the attentional blink is attenuated in psychopathic individuals (Wolf et al., 2012). These findings may reflect fundamental differences in the flexible allocation of selective attention between the two dimensions (see Baskin-Sommers & Newman, 2013, for review). Consistent with this hypothesis, PCL-R factor 1 (indexing affective-interpersonal dysfunction) and PCL-R factor 2 (thought to preferentially access externalizing) appear to have opposite associations to (self-reported) attentional control. Specifically, the core features of psychopathy are linked to enhanced, and impulsive-antisocial features to diminished, selective attention (Baskin-Sommers et al., 2015; Baskin-Sommers, Zeier, & Newman, 2009).

Although such findings might suggest that psychopathic individuals have superior EF overall, this is not consistently found across the entire range of EF subcomponents. Although both interference suppression and response inhibition appear to be compromised in externalizing psychopathology (Heritage & Benning, 2013; Sadeh & Verona, 2008; Sellbom & Verona, 2007; Swann, Lijffijt, Lane, Steinberg, & Moeller, 2009; Zeier, Baskin-Sommers, Hiatt Racer, & Newman, 2012), the evidence that psychopathic individuals are better at inhibiting prepotent motor responses is inconsistent at best (Feilhauer, Cima, Korebrits, & Kunert, 2012; Sadeh & Verona, 2008; Sellbom & Verona, 2007). Moreover, enhanced interference suppression in psychopathy is context-dependent, with psychopathic individuals showing reduced interference only in conditions where their attention is cued to the target location (Hiatt, Schmitt, & Newman, 2004; Zeier, Maxwell, & Newman, 2009; Zeier & Newman, 2013). On the whole, neuropsychological work suggests that psychopathic individuals inflexibly allocate limited capacity early attentional resources. This may lead to an attentional "bottleneck" that limits the ability to process information that is motivationally salient but peripheral to their goal-directed task focus (Baskin-Sommers, Curtin, & Newman, 2011; Baskin-Sommers, Curtin, Li, & Newman, 2012).

Taken together, work to date suggests that externalizing is associated with deficits in selective attention, interference suppression, and response inhibition. By contrast, these aspects of EF appear to be preserved, and in some cases enhanced, in psychopathy. However, the neural mechanisms underlying these putatively dimension-selective associations with EF remain unknown. The goal of the current study is to map the unique variance associated with externalizing and psychopathy to well-characterized brain circuitry for interference suppression and response inhibition. To that end, we used a multimethod approach that integrates clinical, trait, neuropsychological and neurobiological assessments. Specifically, we scanned a sample of 49 incarcerated offenders while they performed a modified Eriksen flanker task that separately manipulated the requirement for interference suppression (IS) and response inhibition (RI). We predicted that after adjusting for shared variance, psychopathy and externalizing would show an opposing pattern of correlation (psychopathy positive, externalizing negative) with dissociable frontoparietal networks subserving IS and RI.

Methods

Participants

Participants were recruited from two medium-security correctional institutions in Wisconsin. A total of 49 right-handed male participants were enrolled (age range = 20–45; $M = 31.52 \pm 7.1$ years). Criteria for eligibility were defined as follows: 45 years old or younger, Wechsler Adult Intelligence Scale–III IQ above 70 (Wechsler, 1997), and not concurrently taking psychotropic medications. Three participants were excluded from analyses due to excessive head movement (two subjects) or poor fMRI quality assurance metrics (one subject; see Methods). Oral and written consent were obtained for all participants, and all methods and procedures were approved by the University of New Mexico, University of Wisconsin–Madison, and Harvard University Institutional Review Boards.

Measures

Participants completed a battery of clinical and neuropsychological assessments.

PCL-R. The PCL-R (Hare et al., 1990) is a gold standard for the forensic evaluation of psychopathy. PCL-R assessment was performed by a trained rater using information from prison files and a semistructured interview that lasted approximately 60 min. Based on information gathered from the interview and file review, the 20 items of the PCL-R were rated 0, 1, or 2, reflecting the degree to which a trait was present: *significantly* (2), *moderately* (1), or *not at all* (0). The reliability and validity of the PCL-R is well established (Hare et al., 1990). In the present study the interrater reliability was .96 on 30% of the sample with dual ratings.

Addiction Severity Index (ASI). The ASI (Leonhard, Mulvey, Gastfriend, & Shwartz, 2000; Rosen, Henson, Finney, & Moos, 2000) was used to estimate severity of substance misuse. In addition to the original ASI questions, participants were asked to indicate, for each substance they endorsed using, their total years of use. We summed each answer across all drugs to calculate a "cumulative use" score (range = 0–76, $M = 14.89$), which

was then used as a covariate in subsequent analyses to control for the potentially confounding effects of chronic substance use on brain function. The validity and reliability of the ASI are well established (McLellan et al., 1985). Interrater reliability data for the ASI were not obtained for this sample.

ESI. Externalizing was measured using the ESI (Krueger et al., 2007), a 100-item self-report questionnaire developed to assess a broad range of behavioral (e.g., substance use) and personality characteristics (e.g., alienation, rebelliousness, and impulsivity) associated with the externalizing spectrum of psychopathology. The 100-item version was derived from Krueger et al.'s (2007) 415-item self-report measure and is correlated at $r = .98$ with the original measure (Krueger et al., 2007). The total range of scores on the ESI is 100 to 400. The validity and reliability of the ESI is well established (Venables & Patrick, 2012). For this sample the internal consistency (Cronbach's alpha) was .96.

Delis-Kaplan Executive Function System (D-KEFS). The D-KEFS (Delis, Kramer, Kaplan, & Holdnack, 2004) was developed to assess components of EF through well-established neuropsychological tests. Contrast measures from the Color-Word Interference Test (inhibition vs. color naming [scaled], inhibition switching vs. color naming [scaled], inhibition errors [percentile rank], inhibition switching errors [percentile rank], inhibition switching vs. inhibition [scaled]) were analyzed. The validity and reliability of the D-KEFS is well established (Delis et al., 2004). We did not assess interrater reliability for the D-KEFS in this sample.

Experimental task

Participants completed a modified version of the Eriksen flanker task that incorporated a go/no go manipulation (Blasi et al., 2006; Eriksen & Eriksen, 1974). On each trial, participants were instructed to indicate, via button press, the direction of a central target arrow (left vs. right) that was situated between a set of flanking arrows. The flanking arrows either pointed in the same direction as the target (congruent condition) or in the opposite direction (incongruent condition). In addition, on some trials (~22%), the central arrow was surrounded by Xs, signifying the need to withhold a response (no-go condition), or by squares (neutral condition; participants were instructed to respond normally). The incongruent condition introduces interference that must be resolved or suppressed to respond appropriately. By contrast, optimal performance in the no-go condition requires participants to inhibit a prepotent motor response (i.e. to press a button). These conditions were displayed in a pseudorandom order over two runs; stimulus order within a run was fixed across participants, and run order counterbalanced between participants. Each stimulus was presented for 800 ms. This duration was selected to ensure low error rates, as the focus of this study was on IS and RI rather than error monitoring. Between trials, a fixation cross was presented; duration of the intertrial interval randomly jittered across trials, according to a Laplacian distribution with $M = 3.5$ s and range = 2–5 s. Each run contained 81 trials, including 23 incongruent and 23 congruent trials, 18 no-go trials, and 17 neutral trials.

fMRI data acquisition

Participants were scanned using a 1.5 Tesla Siemens Magnetom Avanto mobile MRI machine equipped with a 12-channel head coil. While lying supine in the scanner, participants were able to view the stimulus via a back-projection system and made responses on an MRI compatible button box. The presentation of the stimulus and performance of the modified flanker task (described earlier) was synchronized to fMRI volume acquisition. Functional (T2* weighted) images were collected using a gradient-echo EPI pulse sequence (interleaved) using the following parameters: TR 2500 ms, TE 39 ms, flip angle 90°, 33 slices, voxel resolution 3.4 × 3.4 × 3.4 mm, FOV 220 mm. High resolution T1-wighted structural MRI scans were also acquired to coregister the functional images to a standardized anatomical space (multiecho MPRAGE; 1 × 1 × 1.3 mm).

fMRI preprocessing

Prior to analysis, task-related functional images were slice-time corrected using the first slice as a reference, and motion corrected via spatial realignment (2nd-degree B-spline) of all images to a mean image after alignment to the first image of each run. Images were then spatially normalized using unified segmentation and normalization, via the NewSegment routine in SPM, into a standard stereotactic space (Montreal Neurological Institute, MNI template), resampled into 2 mm isotropic voxels, and smoothed with a 6 mm full-width-half-maximum Gaussian kernel. A high-pass filter (128-s cutoff) was applied to remove low-frequency signal drift. Runs were removed if they had a total rotational plus translational displacement of 1 mm or a mean BOLD signal > 3 standard deviations from the sample average, using the ART (artifact detection) tool in Nipype. Two subjects were excluded from final analysis due to movement; another was excluded because their mean BOLD signal for each run was > 3 standard deviations above the group mean.

Behavioral analyses

We used linear mixed model analyses in SPSS 24 to examine the impact of congruency condition on performance (reaction time) and its interaction with psychopathy and externalizing. Fixed effect predictors included condition (congruent vs. incongruent), PCL-R scores, ESI scores, age, and ASI scores, along with condition × PCL-R and condition × ESI interaction terms. Reaction times were not normally distributed (skew = 1.47), and so were log-transformed prior to analysis. Subject was treated as a random effect. PCL-R and ESI scores were included in the same model to capture unique variance associated with psychopathy and externalizing. Robust regression in Stata 14 (RReg) was used to assess relationships between psychopathy, externalizing, and no-go commission error rates. For these analyses, we created an adjusted psychopathy variable by regressing PCL-R, age, and ASI scores against participants' ESI scores and saving the residuals; adjusted externalizing values were similarly constructed. These residual values capture unique variance in psychopathy after controlling for externalizing (and vice versa), age, and substance abuse history. In addition, we employed robust regression to measure associations between adjusted ESI and PCL-R scores, brain activity, and behavior. For robust regression analyses, we report unstandardized coefficients and 95% confidence intervals (CIs); in addition, we provide effect size estimates derived from the equivalent ordinary least squares regression analysis. Age and ASI scores were included as covariates in all robust regression analyses. Multivariate general linear model (GLM) analyses were used to assess relationships between adjusted ESI and PCL-R scores, brain activity, and neuropsychological variables. Age and ASI scores were included as covariates.

fMRI analyses: Task effects

Trial onsets were modeled using a canonical hemodynamic response function with a time derivative. All runs of the task were modeled together. The design matrix for our first-level GLM included trial onset regressors for each condition (incongruent, congruent, no-go, neutral), motion parameters estimated from realignment, a regressor specifying motion outlier time points, and a regressor of onsets for error trials. To reveal activity related to IS, we constructed contrasts of the beta weights for incongruent and congruent trials (incon > con); RI effects were visualized by contrasting brain activity during no-go trials with that during congruent trials (no-go > congruent). The inclusion of predictors for each trial type in the GLM permits assessment of IS, controlling for RI (and vice versa). First-level contrasts were created for each subject; the resulting contrast images were entered into a random-effects one-sample *t* test at the second level (i.e., treating participant as a random effect). To control for Type 1 error due to multiple comparisons, we used a cluster-level false discovery rate (FDR) threshold of $p < .05$ in conjunction with a cluster-forming height threshold of $t > 3$.

fMRI analyses: Individual differences

To identify relationships between psychopathy, externalizing, IS and RI, we created two multiple regression models in SPM8. In the first, PCL-R and ESI scores, along with age and substance abuse values, were modeled as predictors of IS-related activation (incongruent > congruent contrasts). In the second, the same set of variables were modeled as predictors of RI-related activity (no-go contrasts). In each model, PCL-R and ESI predictors were separately weighted with a "1" or "–1" to reveal correlations with psychopathy (controlling for externalizing) and externalizing (controlling for psychopathy). Control over Type 1 error across the whole brain was achieved via cluster-level FDR correction ($p < .05$, with a cluster-forming height threshold of $t > 3$).

Results

Clinical measures

The zero-order Pearson product–moment correlation between PCL-R total and ESI total scores was $r = .64$, $p < .001$; correlations between ESI total and PCL-R Factor 1 and Factor 2 scores were $r = .45$, $p = .002$ and $r = .65$, $p < .001$, respectively. The two PCL-R factors were correlated at $r = .53$, $p < .001$.

Behavior

We found a main effect of congruency on reaction time, $F(1, 45) = 108.06$, $p < .001$, $\eta_p^2 = .71$, such that responses were significantly faster for congruent trials (.595 s ± .105) than incongruent trials (M = 650 s ± .113). We did not find significant congruency × ESI, $F(1, 43) = 3.45$, $p = .07$; $\eta_p^2 = .07$, or congruency × PCLR interactions, $F(1, 43) = 0.63$, $p = .43$; $\eta_p^2 = .01$, indicating that neither psychopathy or externalizing-unique variance moderated the effect of congruency on response times during the task. Likewise, we did not observe significant congruency × ESI or congruency × PCLR interactions when ESI and PCLR were considered on their own (i.e., in separate models; $ps > .08$). However, main effects for psychopathy were evident: Adjusted PCL-R scores were associated with slower response times overall, $t(41) = 2.59$, $p = .01$, $\eta_p^2 = .14$, whereas adjusted ESI scores predicted faster response times irrespective of congruency condition

(t = –2.17, p = .04, η_p^2 = .1). The association between adjusted externalizing scores and no-go error rates was not significant (B = 0.008, –0.0009 to 0.012, p = .07, η_p^2 = .08), nor was the association between adjusted psychopathy scores and no-go error rates (B = –0.05, –0.12 to 0.02, p = .17, η_p^2 = .05).

fMRI: Task effects

Consistent with prior reports (Blasi et al., 2006), IS engaged a distributed frontoparietal network with prominent foci in the supplementary motor area, frontal eye fields, inferior frontal gyrus (pars opercularis; IFG_{OPR}) and inferior parietal cortex (see Table S1 available online; Fig. 1). By contrast, activity during RI (No-Go > congruent) was strongest in the inferior frontal gyrus (encompassing pars orbitalis and pars triangularis; IFG_{ORB}, IFG_{TRI}), the temporoparietal junction, anterior cingulate cortex (ACC; Brodmann Area 24/32), dorsolateral prefrontal cortex (DLPFC; Brodmann Area 9), and anterior prefrontal cortex (Brodmann Area 10; see Table S2 available online; Fig. 2).

fMRI: Individual differences

We did not observe any significant correlations with adjusted ESI scores and brain activity during IS. By contrast, significant positive relationships between adjusted PCL-R scores and IS-related BOLD signal were found in left IFG_{ORB} (BA 47; –50, 30, 20 [MNI]; k = 95, peak Z = 3.81), left DLPFC (BA 46; –48, 36, –16 [MNI]; k = 84, peak Z = 3.75), anterior medial prefrontal cortex (amPFC; BA 10/32; –2, 64, 22 [MNI]; k = 203, peak Z = 3.69), and left temporoparietal junction (TPJ; –52, –56, 30 [MNI]; k = 158, peak Z = 4.86; Figs. 3A–3B). During RI, externalizing and psychopathy showed opposite patterns of association to DLPFC activity: Higher adjusted ESI scores predicted lower left DLPFC activation during RI (–50, 12, 40 [MNI]; k = 263, peak Z = 4.22, whereas adjusted PCL-R scores were positively correlated with inhibition-related

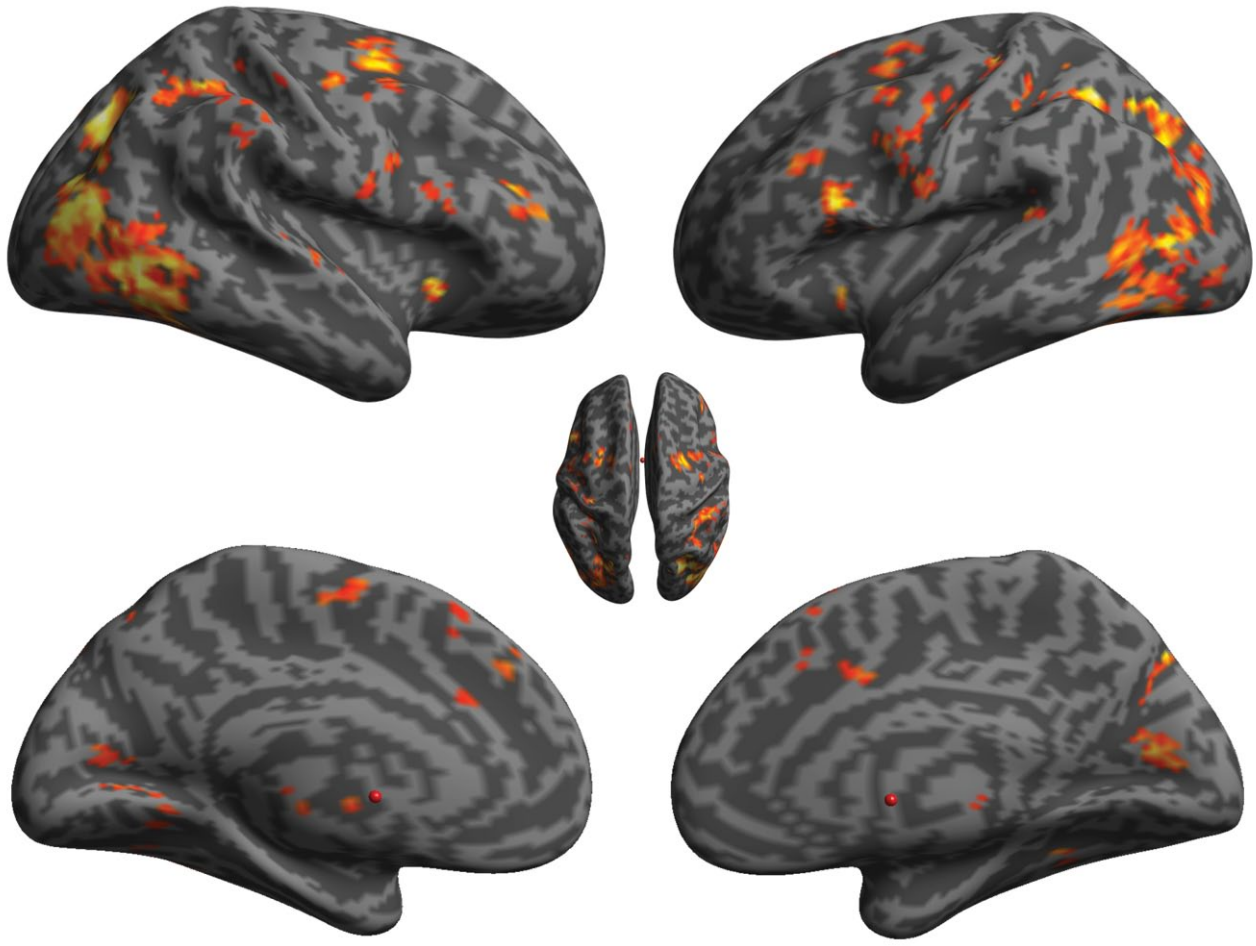

Fig. 1. Brain activation during interference suppression. Statistical parametric map (SPM) displays significant foci revealed by the incongruent > congruent contrast. SPM is thresholded at $p_{Cluster\text{-}FDR} < 0.05$, using a cluster defining height threshold of $t > 3$.

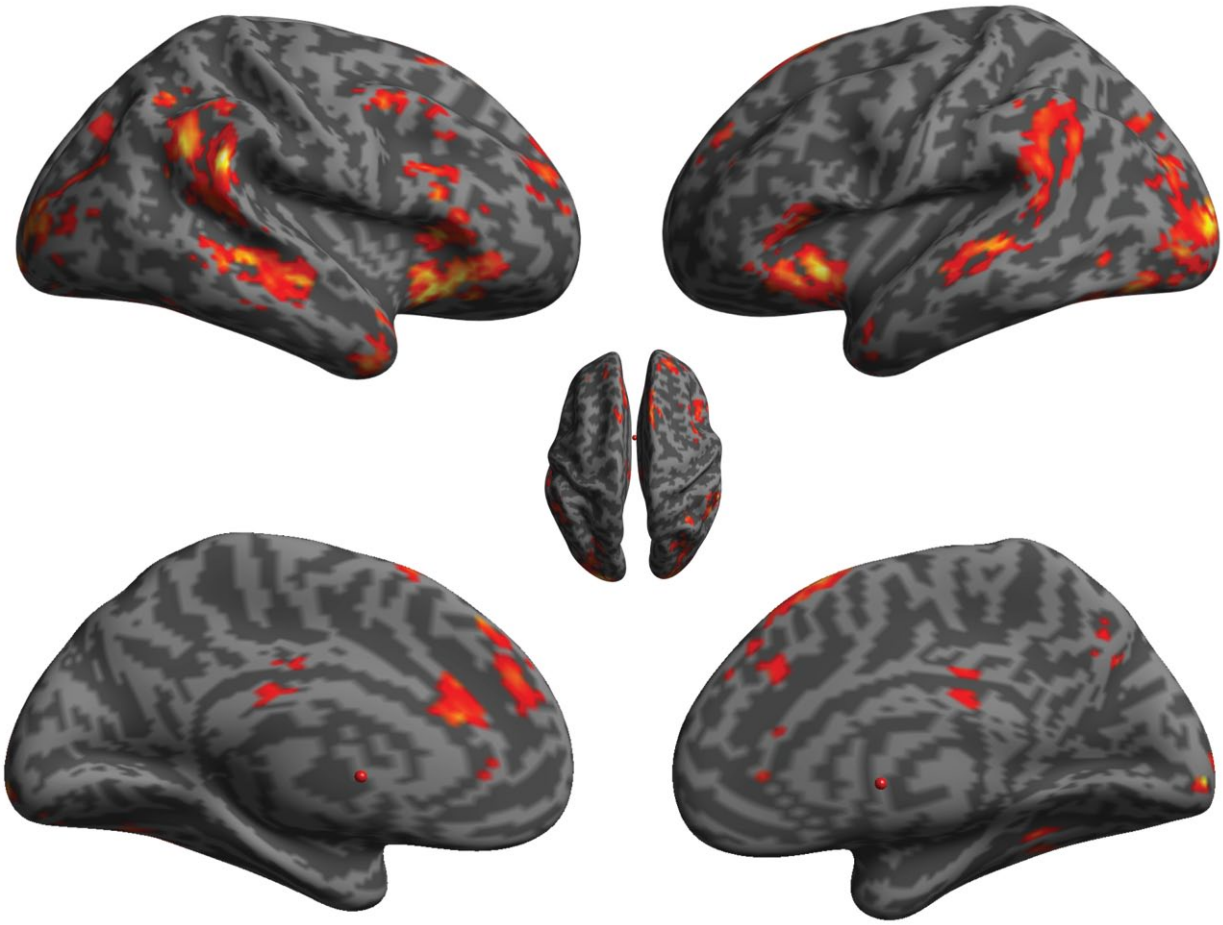

Fig. 2. Brain activation during response inhibition. SPM displays significant foci revealed by the no-go > congruent contrast. SPM is thresholded at $p_{\text{Cluster-FDR}} < 0.05$, using a cluster defining height threshold of $t > 3$.

activity within left DLPFC (–50, 28, 24 [MNI]; k = 100, peak Z = 4.31) and left TPJ (–54, –58, 30 [MNI]; k = 105, peak Z = 4.06; Figs. 3C–3D). In sum, these results show that psychopathy-specific variance is associated with heightened frontoparietal activity during both IS and RI. Externalizing-specific variance, on the other hand, was linked to decreased prefrontal BOLD signal during RI and showed no association to IS-related activity.

Brain-behavior relationships

fMRI task performance. To determine the relevance of psychopathy and externalizing-linked differences in brain activation to task performance, we extracted BOLD signal from 8 mm spheres centered on the peak coordinates of activation foci identified from the adjusted ESI and PCL-R correlation contrasts for IS and RI maps. For IS, we subtracted reaction times in the congruent condition from those in the incongruent condition to create an index of susceptibility to interference (RT_{Diff}). RT_{Diff} values were negatively associated with IS-related activation in IFG (B = –0.004, –0.006 to –0.008, p = .01, η_p^2 = .01). This result showed that individuals with higher IFG activation during IS exhibited decreased distractor susceptibility in the flanker task. Associations between RT_{Diff} and activity within DLPFC, amPFC and TPJ were not significant (p value range = .33–.72).

A similar analysis was performed for RI trials, revealing a negative relationship between commission error rate and DLPFC activation during the task (B = –2.28, –0.51 to –0.06, p = .01, η_p^2 = .16, activation focus from EXT SPM; B = –0.25, –0.44 to –0.06, p = .01, η_p^2 = .19, activation focus from PCL-R SPM). This indicates that individuals with lower DLPFC activity during RI were more prone to impulsive responding. Thus, the pattern of activation linked to unique variance in psychopathy (higher IFG activity during IS and high DLPFC activity during RI) was associated with decreased distractor susceptibility and reduced motor impulsivity. By contrast, the activation pattern that tracked unique variance in

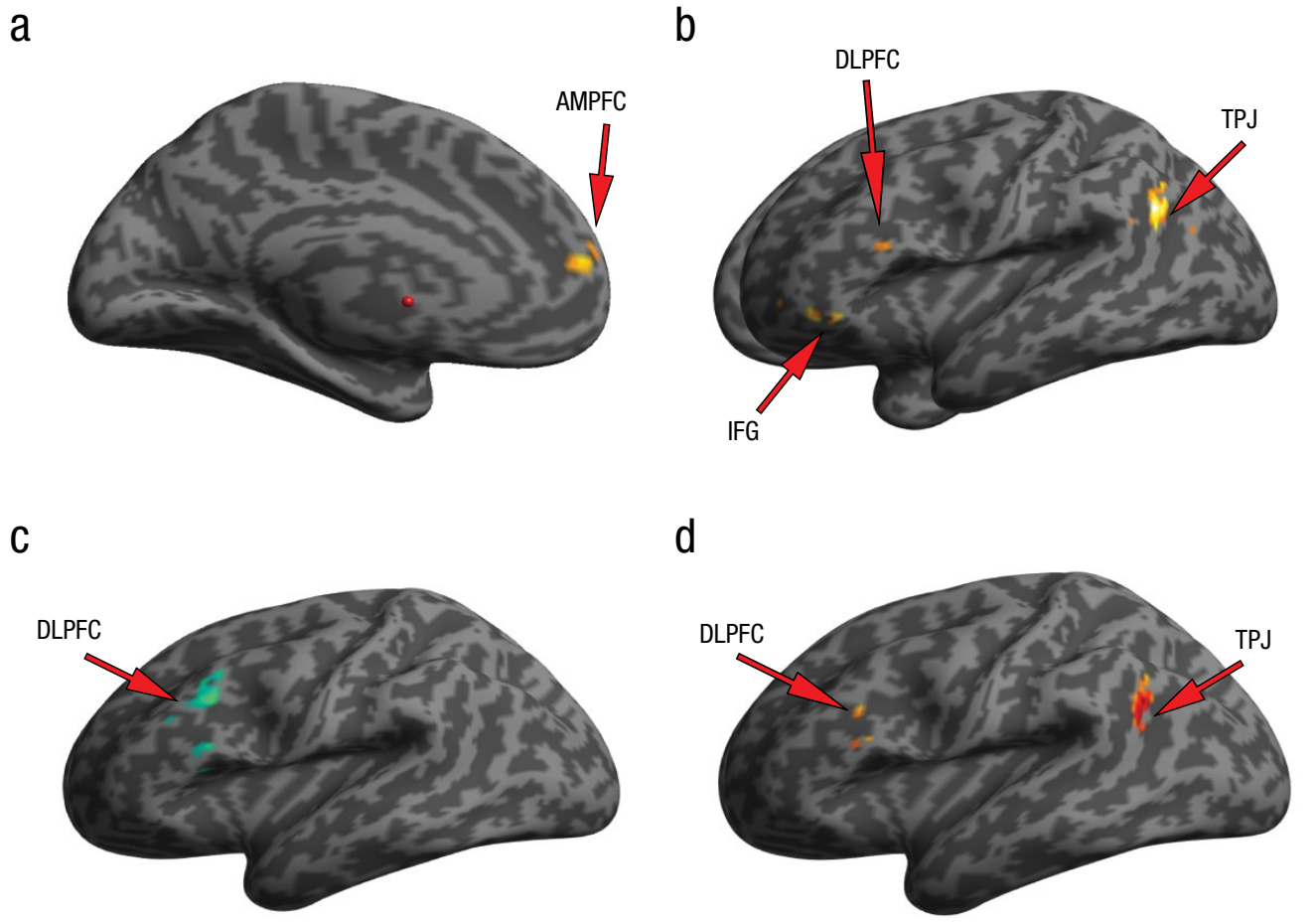

Fig. 3. Differential effects of psychopathy and externalizing on frontoparietal circuit function during inhibititory self-control. Panels A–B depict regions where adjusted PCL-R scores are significantly positively correlated with brain activity during interference suppression (incongruent > congruent contrast). Panel C shows the significant negative correlation with adjusted EXT scores and DLPFC function during response inhibition (No-Go > Congruent). Panel D displays the significant positive correlation between adjusted PCL-R scores and response inhibition-related activity within DLPFC and the TPJ. SPMs are thresholded at $p_{\text{Cluster-FDR}} < 0.05$, using a cluster defining height threshold of $t > 3$.

externalizing (lower DLPFC activity during RI) was linked to increased motor impulsivity.

Color-Word Interference Test performance. As a test of convergence, we ran a multivariate GLM analysis to assess relationships between externalizing and psychopathy and measures of inhibitory control and attentional flexibility derived from the D-KEFS battery. We found that unique variance in psychopathy negatively predicted inhibition/switching performance (B = –0.21, –0.36 to –0.05, p = .01, η_p^2 = .16, scaled inhibition switch vs. color contrast; B = –0.15, –0.31 to 0.002, p = .05, η_p^2 = .1, inhibition switch time). Next, we constructed two multivariate GLM analyses in which IS and RI-related activity were separately considered as predictors of D-KEFS inhibitory control and attentional flexibility measures. For the IS analyses, we used signal from each of the four foci identified in the whole-brain individual difference analyses (i.e., DLPFC, IFG, TPJ, and amPFC). We found that IS-related BOLD signal within IFG predicted poorer inhibition/switching performance (B = –1.12, p = .02, –2.02 to –0.22, η_p^2 = .16, scaled inhibition switch vs. color contrast; B = –1.36, –2.42 to –0.29, p = .01, η_p^2 = .16, scaled inhibition switch vs. inhibition contrast). Robust regression analyses corroborated this finding (p < .001 and p = .02, respectively). For the RI analysis, we used signal from each of the three foci identified from the whole-brain correlations with adjusted ESI and PCL-R scores (DLPFC, TPJ). This analysis did not reveal any significant associations between RI-related BOLD signal and D-KEFS measures of inhibitory or attentional control. On the whole, these findings suggest that psychopathy, and psychopathy-linked heightened frontoparietal BOLD signal during IS, is associated with diminished attentional flexibility during a Stroop-like color-word interference test.

Discussion

Here, we employed a multilevel and multimeasure approach to map externalizing and psychopathy to brain circuitry supporting two executive capacities for inhibitory self-control: IS and RI. A modified Eriksen flanker task permitted selective evaluation of IS and RI. The unique variance attributable to psychopathy was positively associated with frontoparietal activation during both IS and RI. By contrast, the unique variance attributable to externalizing was negatively associated with DLPFC activity during RI; no relationship to IS-related brain activity emerged. These results provide a neurobiological dissociation of externalizing and psychopathy: The former is linked to relatively weaker prefrontal activity during RI, whereas the latter is characterized by relatively stronger recruitment of frontoparietal networks during both RI and IS.

On the whole, these findings accord well with prior work showing reduced cortical thickness (Yang & Raine, 2009) and poor performance on RI tasks (Dolan, 2012; Dolan & Park, 2002) in participants with high levels of externalizing. Our analyses suggest that externalizing is associated with reduced DLPFC activation during RI. Although the correlation between adjusted ESI scores and commission errors was not significant, the strong negative relationship between RI-related DLPFC BOLD signal and commission errors implies that diminished DLPFC engagement in externalizing individuals is dysfunctional.

A significant open question pertains to the relevance of inhibitory control deficits for "real-world" self-control failure (e.g., substance abuse, aggression, and criminal behavior) in externalizing individuals. Prevailing models assume that antisocial behavior in externalizing individuals results from a deficit in the capacity to actively inhibit the execution of prepotent responses to threat or reward associated stimuli (Dolan, 2012; Dolan & Park, 2002; Herpertz et al., 2008; Hobson, Scott, & Rubia, 2011; Kirisci, Tarter, Mezzich, & Vanyukov, 2007; Patrick, Durbin, & Moser, 2012; Raine & Yang, 2006; Swann et al., 2009). The current results would appear to support this model, and are consistent with other brain imaging studies in antisocial offenders that have reported reductions in DLPFC gray matter volume and cortical thickness DLPFC (Dolan, 2012; Montigny et al., 2013; Sarkar et al., 2015; Wallace et al., 2012; Weiland et al., 2014; Yang & Raine, 2009; Yang, Raine, Colletti, Toga, & Narr, 2010), as well as reduced DLPFC activation during classic neuropsychological indices of inhibitory control (Moeller et al., 2014; Vollm et al., 2004; Yang & Raine, 2009; Ziermans et al., 2012). By contrast, externalizing individuals appear to have relatively exaggerated responses to threat stimuli (within the amygdala) and reward cues (within the striatum; Bjork, Chen, & Hommer, 2012; Buckholtz, Treadway, Cowan, Woodward, Benning, et al., 2010; Buckholtz, Treadway, Cowan, Woodward, Li, et al., 2010; Carré, Hyde, Neumann, Viding, & Hariri, 2013; Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Coccaro, Sripada, Yanowitch, & Phan, 2011; Hyde, Byrd, Votruba-Drzal, Hariri, & Manuck, 2014; Pujara, Motzkin, Newman, Kiehl, & Koenigs, 2014). Together, such findings are often construed as evidence that the impulsive-reactive antisocial behavior characteristic of externalizing occurs when bottom-up "affective" signals activate or generate a prepotent behavioral response that is inadequately inhibited by top-down "cognitive" resources due to poor prefrontal control. However, we (Buckholtz, 2015) have speculated that the relevance of EF deficits for antisocial behavior in externalizing individuals may be more apparent than real. Central to this argument is the role of DLPFC; in contrast to "inhibition-centric" models of antisocial behavior, we have previously argued for a stronger focus on the role of prefrontal cortex in value-based decision-making (Buckholtz, 2015; Buckholtz & Faigman, 2014). A growing body of work suggests that prefrontal cortex can promote self-control by reweighting striatal action value signals according to prospective simulations that incorporate information about goals, costs, consequences, and context, rather than by inhibiting the execution of an action program after valuation and selection have already occurred. Prefrontal dysfunction, therefore, may predispose impulsive antisocial behavior by preventing these prospective calculations from appropriately modulating "downstream" action value signals, rather than through a failure to actively inhibit a maladaptive motor program that has already been selected for execution. If this is true, associations between inhibitory control-related brain activity and antisocial behavior link may not reflect a direct causal relationship, but rather may arise epiphenomenally from the fact that DLPFC is important for both EF and value-based decision making. In other words, EF deficits may be a "third variable" marker of compromised prefrontal value modulation. Future work should test this hypothesis by measuring prefrontal function during both RI and value-based decision-making tasks, and determining whether associations between externalizing and RI-related brain activity remain after controlling for brain activity linked to value-based decision-making. Likewise, prospective designs could determine whether EF and value-based decision making each uniquely predict future antisocial behavior in externalizing individuals (and if so, which of the two has the strongest predictive power).

Our finding that psychopathic individuals have increased frontoparietal engagement during IS accords well with reports that these individuals exhibit superior selective attention relative to individuals low on psychopathy (Baskin-Sommers et al., 2009; Baskin-Sommers

et al., 2015; Sadeh & Verona, 2008; Sellbom & Verona, 2007). Moreover, enhanced prefrontal activity during IS trials predicted less susceptibility to distractors. However, some caution is warranted in interpreting the present data as evidence for superior EF in psychopathic individuals. In particular, the observed correlations between psychopathy-linked frontoparietal activity and inhibition/switching performance on the Color-Word Interference Test implies that attentional flexibility is compromised in psychopathy. On the whole, the combination of decreased distractor susceptibility and poorer attentional flexibility is consistent with the suggestion that psychopathic individuals have a deficit in early attentional selection mechanisms, leading to an attentional bottleneck phenomenon (Baskin-Sommers, Curtin, et al., 2012; Hamilton, Baskin-Sommers, & Newman, 2014). Future imaging studies with IS tasks that manipulate these early attentional selection mechanisms will be necessary to clarify and extend the present findings.

Taken together, these findings provide neurobiological evidence supporting the existence of two distinct dimensions of antisocial behavior. In addition, they shed light on dimension-specific systems-level pathomechanisms. However, several issues merit consideration. First, we did not observe any significant relationships between adjusted EXT or PCL-R scores and task performance. This may be due to our task design, which was optimized for imaging and resulted in most participants performing near ceiling. Although this was done to reduce errors (and potentially confounding error-related activity), by minimizing individual variation in performance we may have reduced the likelihood of detecting associations between our assessment measures and task behavior. Future imaging work in this area would benefit from the use of a task design that induces more variable performance, and which includes enough trials to enable an appropriately powered investigation of error-related activity (Aharoni et al., 2013). Second, the associations reported here are modest in size. This is consistent with a multifactorial model of antisociality, wherein relative deficits in multiple cognitive, affective, social and motivational processes contribute to the expression of antisocial behavior (Buckholtz & Meyer-Lindenberg, 2012). Third, we limited our investigation of EF to only two subcomponent processes—IS and RI—due to practical considerations. Within the domain of "cognition" alone, this leaves many other candidate processes—such as response selection, action cancellation, and error detection—unexamined. Future work in this area should endeavor to develop a more precise and comprehensive mapping of cognitive, affective, social, and motivational processes to common and unique variance associated with externalizing and psychopathy.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at http://cpx.sagepub.com/content/by/supplemental-data.

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