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# A multidimensional examination of psychopathy traits and gray matter volume in adults

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## Abstract

Uncovering the neurobiological abnormalities that may contribute to the manifestation of psychopathic traits is an important step toward understanding the etiology of this disorder. Although many studies have examined gray matter volume (GMV) in relation to psychopathy, few have examined how dimensions of psychopathic traits interactively relate to GMV, an approach that holds promise for parsing heterogeneity in neurobiological risk factors for this disorder. The aim of this study was to investigate the affective-interpersonal (Factor 1) and impulsive-antisocial (Factor 2) dimensions of psychopathy in relation to cortical surface and subcortical GMV in a mixed-gender, high-risk community sample with significant justice-system involvement ($N = 156$, 50.0% men). Cortex-wide analysis indicated that (i) the Factor 1 traits correlated negatively with GMV in two cortical clusters, one in the right rostral middle frontal region and one in the occipital lobe, and (ii) the interaction of the affective-interpersonal and impulsive-antisocial traits was negatively associated with GMV bilaterally in the parietal lobe, such that individuals high on both trait dimensions evidenced reduced GMV relative to individuals high on only one psychopathy factor. An interactive effect also emerged for bilateral amygdalar and hippocampal GMV, such that Factor 1 psychopathic traits were significantly negatively associated with GMV only at high (but not low) levels of Factor 2 traits. Results extend prior research by demonstrating the neurobiological correlates of psychopathy differ based on the presentation of Factor 1 and 2 traits.

**Key words:** psychopathy; affective-interpersonal; impulsive-antisocial; gray matter volume

Psychopathy is a construct defined by a constellation of personality traits that include callousness, shallow affect, grandiosity, impulsivity and antisocial behavior (Hare, 2003). Psychopathic traits are robustly associated with negative clinical outcomes, including substance use disorders and incarceration (Beaver *et al.*, 2014), which makes identifying the etiology of these personality traits an important area of research. Etiological models of psychopathy recognize that psychopathy is a multidimensional construct comprised of at least two primary dimensions: Factor 1 (F1) represents the affective and interpersonal symptoms of psychopathy (e.g. lack of remorse, shallow affect, superficial charm, deceitfulness) and Factor 2 (F2) captures the chronic engagement in impulsive and antisocial acts (e.g. need for stimulation, irresponsibility, criminal behavior) (Hare, 2003; Hare and Neumann, 2005; Lilienfeld *et al.*, 2015). Although high scores on both factors are needed for an individual to meet criteria for a diagnosis of psychopathy (Hare *et al.*, 1990), a growing body of research has illustrated that the psychopathy factors show unique relations with etiological mechanisms that are not apparent when psychopathy is examined as a unitary construct (Benning *et al.*, 2003; Harpur *et al.*, 1989; Verona, Patrick and Joiner, 2001; Ross *et al.*, 2009). At the same time, individuals who score high on both psychopathy factors are known to be at highest risk for criminal recidivism and the most difficult to treat (Douglas *et al.*, 2018), underscoring the importance of characterizing the etiological profiles of individuals who manifest elevations on both primary dimensions of psychopathy. Given that distinct neurobiological abnormalities may predispose individuals to develop the affective-interpersonal and impulsive-antisocial features of the disorder (Umbach *et al.*, 2015), the objective of this study was to further understand the neurobiology of psychopathy by examining associations between the psychopathy factors and gray matter volume (GMV).

## Etiological pathways to psychopathy factors

The psychopathy dimensions are theorized to index separable etiological pathways to criminal behavior that are characterized by distinct risk factors (Harpur *et al.*, 1989; Hall *et al.*, 2004; Fowles and Dindo, 2006; Sadeh *et al.*, 2013a). For example, the interpersonal-affective (F1) dimension has been associated with low levels of fear, resilience to mood disorders and abnormal attentional functioning (e.g. Benning *et al.*, 2003; Harpur *et al.*, 1989; Sadeh and Verona, 2008), whereas the impulsive-antisocial

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(F2) dimension has been linked to high levels of distress and psychopathology, working memory deficits and psychosocial adversity (Harpur *et al.*, 1989; Benning *et al.*, 2003). Abnormalities in neural circuitry postulated to modulate the experience of fear, such as the amygdala and paralimbic system (e.g. Kiehl, 2006; Blair, 2013), are thought to motivate the interpersonal-affective symptoms of psychopathy. For example, functional studies have shown that F1 traits are negatively associated with amygdala activation to emotional stimuli (Gordon *et al.*, 2004; Buckholtz *et al.*, 2010). Conversely, F2 traits are typically positively associated with activation in emotional processing and reward anticipation regions, including the amygdala and nucleus accumbens (Gordon *et al.*, 2004; Buckholtz *et al.*, 2010), suggesting hyper-responsivity to motivationally relevant stimuli.

There is also evidence that differences in executive functioning drive etiological differences related to the two psychopathy factors. Research suggests that the interpersonal-affective traits (F1) drive psychopathic individuals to perform abnormally on attentionally demanding tasks (e.g. Sadeh and Verona, 2008; Koenigs *et al.*, 2011; Baskin-Sommers *et al.*, 2013; Larson *et al.*, 2013). In contrast, deficits in working memory and response inhibition are differentially associated with the impulsive-antisocial dimension (F2) (Sadeh and Verona, 2008). Given this pattern, there are likely both functional and structural neurobiological differences that explain these differences in cognition between the two psychopathy dimensions (Moreira *et al.*, 2019).

## Psychopathy factors and gray matter volume

Consistent with the theory that the psychopathy dimensions index distinct risk processes for antisocial behavior, research examining F1 and F2 traits and GMV have shown some differential correlates. Previous research suggests that affective-interpersonal F1 traits have specifically been negatively associated with GMV in the dorsolateral prefrontal cortex (Korponay *et al.*, 2017), the orbitofrontal cortex (De Brito *et al.*, 2021) and the anterior and posterior cingulate cortices (Caldwell *et al.*, 2019), consistent with the paralimbic hypothesis (Kiehl, 2006). Impulsive-antisocial F2 psychopathic traits also evidence some distinct GMV associations. Findings have shown positive associations with GMV of the basal ganglia (e.g. the striatum) (Cope *et al.*, 2012; Korponay and Koenigs, 2021) and negative associations between F2 traits and the middle occipital gyrus (De Brito *et al.*, 2021) and the supplementary motor area (Leutgeb *et al.*, 2015), all regions important for reward processing and impulse control. There have also been F2 trait associations with GMV in the prefrontal cortex, although the findings have been mixed, with some studies reporting positive GMV associations in the prefrontal cortex (Korponay *et al.*, 2017; Korponay and Koenigs, 2021) and others reporting negative associations (De Brito *et al.*, 2021), including with the OFC. It is possible that these observed discrepancies could be due to the types of populations being studied, as recent research has pointed out that the neural mechanisms underlying impulsive traits may differ in forensic *vs* community samples (Korponay and Koenigs, 2021). In spite of these possible specific GMV associations with the psychopathic trait dimensions, it should also be noted that distinct associations are not always observed, as evidenced by studies showing both psychopathy factors are negatively associated with GMV in the orbitofrontal cortex (Ermer *et al.*, 2012) and the amygdala (Yang *et al.*, 2009b; Ermer *et al.*, 2012; Pardini *et al.*, 2014; Vieira *et al.*, 2015; Ling and Raine, 2018). Thus, whether the psychopathy dimensions are associated with unique or overlapping neurobiological vulnerabilities remains unclear and requires further investigation.

## Neurobiology of interactive effects of psychopathic traits

In addition to the unique variance associated with each psychopathy factor, it may be important to characterize the neurobiological correlates of their interactive effects. Many studies have examined neurobiological differences between individuals who meet criteria for a diagnosis of psychopathy (i.e. individuals who score high on both F1 and F2) *vs* those who do not (Griffiths and Jalava, 2017; Hofhansel *et al.*, 2020) and report reductions in GMV in paralimbic structures (OFC, insula) (Nummenmaa *et al.*, 2021), and the supplementary motor area (Leutgeb *et al.*, 2015) in psychopathic individuals, as well as mixed findings with striatal volumes (Boccardi *et al.*, 2013; Lam *et al.*, 2017). While informative about extreme manifestations of psychopathic traits, such group-based designs are inconsistent with research showing psychopathy is best characterized as a continuous, rather than taxonic construct (e.g. Edens *et al.*, 2006). The continuous analysis of psychopathic traits, and their interactive effects, provides insight into the link between the severity of psychopathic traits and GMV characteristics. However, almost no research has examined how factor-level interactions relate to abnormalities in GMV (Hofhansel *et al.*, 2020), which is important for clarifying how one psychopathy factor may attenuate or accentuate the effects of the other (Walsh and Kosson, 2008; Sprague *et al.*, 2012; Verona *et al.*, 2012).

## Current study

To further understand how the interplay of the psychopathy factors relates to GMV, the aim of this study was to investigate the main and interactive effects of affective-interpersonal (F1) and impulsive-antisocial (F2) psychopathic traits on GMV in a mixed-gender, high-risk community sample. In light of the broader literature on the external correlates of the psychopathy dimensions, we expected the interpersonal-affective dimension (F1) to be negatively associated with GMV in dorsolateral prefrontal cortex, orbitofrontal cortex, cingulate cortices and subcortical limbic regions, specifically the amygdala and hippocampus (Caldwell *et al.*, 2019; De Brito *et al.*, 2021). We expected the impulsive-antisocial dimension (F2) to be negatively related to GMV in supplementary motor area and positively associated with subcortical reward processing regions, specifically the subregions of the striatum (Glenn *et al.*, 2010; Cope *et al.*, 2012; Leutgeb *et al.*, 2015; Korponay and Koenigs, 2021). We also expected that the interactive effects of the psychopathy factors would parallel the neurobiological abnormalities observed for individuals high on psychopathy.

## Methods

### Participants

Participants were recruited from the community using flyers and online advertisements. To be eligible for the study, participants were required to be between 18 and 50 years old and fluent in English. Participants were excluded if they reported a serious medical condition, history of head trauma resulting in loss of consciousness for over 30 min or lasting effects, current psychosis, any MRI contraindications or an estimated IQ of less than 80.

Written and oral informed consent was obtained from all subjects, as approved by the University of Delaware Institutional Review Board.

We excluded two participants with unreliable self-report data, one participant missing handedness, and 10 participants that did not pass MRI data quality assurance standards. This resulted in a final sample size of 156 adults (50.0% men; $M/SD_{age} = 30.7/8.4$ years old). The sample was racially and socioeconomically diverse. Participants identified as Caucasian (55.8%), Black or African American (34.0%), Asian American (9.6%) or 'Other' (1.9%) and 13.5% identified as Hispanic or Latino. The median household income was just over $36 000 for the last year, and most participants came from communities with high rates of violent and non-violent crime (https://www.neighborhoodscout.com/de/wilmington/crime on 7 January 2021). About half of the sample reported being arrested at least once in their lifetime.

## Measures

### *Psychopathic traits*

The Self-Report Psychopathy Scale-Fourth Edition (SRP-4) (Paulhus *et al.*, 2015) is a 64-item measure developed based on the well-established Hare Psychopathy Checklist-Revised (Hare, 2003) and is designed to assess psychopathic traits in community and forensic samples. Each item is scored on a five-point Likert Scale (1 = 'Strongly Disagree', 5 = 'Strongly Agree'). Responses on the Affective and Interpersonal facets were summed to create a combined Affective-Interpersonal factor (F1) and responses on the Lifestyle and Antisocial facets were summed to create a combined Impulsive-Antisocial factor (F2). In this sample, Cronbach's alpha reliabilities were 0.89 and 0.88 for F1 and F2, respectively.

### *Substance use*

The Risky, Impulsive and Self-Destructive Behavior Questionnaire (Sadeh and Baskin-Sommers, 2017) was used to assess the frequency of lifetime alcohol and drug use (cannabis, cocaine, opioids, prescription pills, psychedelic drugs). Participants reported how many times they engaged in substance use in the past month and throughout their lifetime. Responses were categorized into five bins that constrained the range of possible responses at the high end of the distribution: 0, 1–10, 11–50, 51–100, >100 times. Positive skewness was further reduced using a Blom transformation.

### *MRI acquisition*

Data were collected using a Siemens 3T Magnetom Prisma scanner with a 64-channel head coil. T1-weighted multi-echo MPRAGE anatomical scan (resolution = 1 $mm^3$, TR = 2530 ms, TEs = 1.69, 3.55, 5.41, 7.27 ms) was collected, which has the advantage of less distortion and higher contrast than standard MPRAGE sequences, resulting in more reliable cortical models (van der Kouwe *et al.*, 2008). A T2-weighted variable flip-angle turbo spin-echo scan (resolution = 1 $mm^3$, TR = 3200 ms, TE = 564 ms) was collected, to be used in FreeSurfer to better differentiate the gray-matter-dura boundary.

### *Gray matter volume*

The cortical surface volume of each vertex was estimated using FreeSurfer's (v6) standard morphometric pipeline (Fischl, 2012). Data were spatially smoothed using a Gaussian kernel of 15 mm full-width at half maximum, following previous similar analyses (Bernal-Rusiel *et al.*, 2010; Hyatt *et al.*, 2012). The Freesurfer (v6) standard volume-based stream was used to segment the subcortical volumes (amygdala, hippocampus, caudate, putamen and nucleus accumbens) and label cortical and subcortical tissue classes, the steps of which are fully described elsewhere (Fischl *et al.*, 2002, 2004).

## Data analytic plan

General linear models were used to examine associations between psychopathic traits and GMV. For all models, SRP-4 F1 and SRP-4 F2 scores were included in each model simultaneously to examine the unique associations of each factor with GMV, and the interaction term was entered in a separate step. Age, biological sex (sex), scaled verbal IQ scores (Wechsler, 2008), estimated total intracranial volume (eTIV) and handedness were included in all analyses as covariates of no interest, based on previous analyses, and given associations between these variables, psychopathy and brain morphology in previous research (Szabo *et al.*, 2001; Baskin-Sommers *et al.*, 2016; Sajous-Turner *et al.*, 2020; Pan *et al.*, 2021). We also conducted follow-up analyses for significant results examining whether the findings were attributable to substance use using total lifetime drug and alcohol use (substance use).

### *Cortical surface volumes*

FreeSurfer's QDEC application was used to test general linear models examining the associations between the main and interactive effects of SRP-4 F1 and SRP-4 F2 traits and cortical GMV. All variables were standardized prior to analysis. To correct for multiple comparisons, we applied a standard correction procedure using the FreeSurfer analysis software. Volumetric results were corrected for the number of tests conducted using pre-cached Gaussian Monte Carlo simulation (10 000 iterations, cluster-wise threshold: $P < 0.05$, sign: absolute) that was created based on the size of the ROI examined (Hagler *et al.*, 2006).

### *Subcortical volumes*

We employed multivariate hierarchical linear regression analysis using SPSS version 26 to evaluate associations between psychopathic traits and subcortical volumes. Specifically, we entered amygdala, hippocampus, caudate, putamen and nucleus accumbens for each hemisphere as dependent variables (total of 10 dependent variables), the psychopathy factors and their interaction as predictors, and age, sex, VIQ, eTIV and handedness as covariates in this analysis. All variables were standardized prior to analysis. We present the covariates, main effects and interactive effects in separate steps of the regression to allow for interpretation of the variance in the dependent variables accounted for by variables of no interest (covariates) separately from the explanatory variables (psychopathy dimensions).

# Results

## Descriptive statistics

On average, scores on the affective-interpersonal (SRP-4 F1: $M/SD = 74.7/17.4$) and impulsive-antisocial (SRP-4 F2: $M/SD = 71.8/18.0$) trait dimensions were higher than those typically observed in community samples (SRP-4 F1: $M/SD = 61.6/11.8$; SRP-4 F2: $M/SD = 63.1/15.6$) and lower than those normed from forensic samples (SRP-4 F1: $M/SD = 89.0/16.5$; SRP-4 F2: $M/SD = 100.6/16.2$) (Paulhus *et al.*, 2017). As expected, the psychopathy factors were moderately intercorrelated ($r = 0.56$, $P < 0.001$).

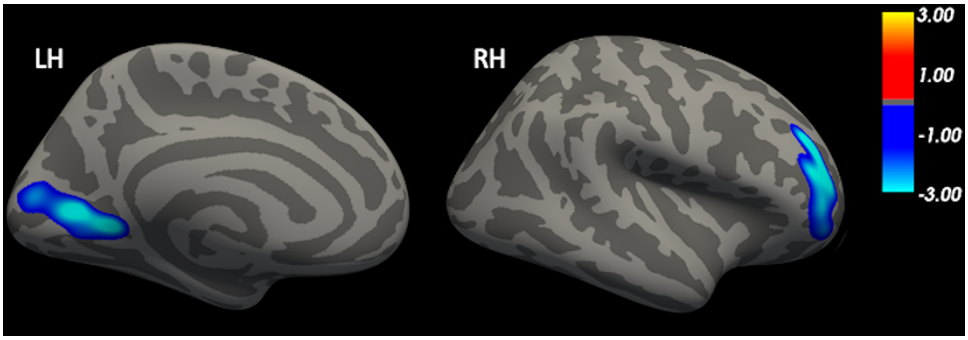


**Fig. 1.** F1 psychopathic traits negatively related to cortical volume in regions of both hemispheres, after controlling for age, sex, VIQ, eTIV, handedness and F2. Left Hemisphere: Peak *F*-Value = –3.54. Peak MNI (x,y,z) = (–21.6, –73.7,3.1). No. of Vertices = 3150. Cluster Size ($mm^2$) = 1747. Right Hemisphere: Peak *F*-Value = –4.61. Peak MNI (x,y,z) = (29.4,35.4,22.2). No. of Vertices = 2974. Cluster Size ($mm^2$) = 1884.

## Psychopathic trait relations with cortical surface volumes

Cortex-wide analysis of GMV identified two clusters associated with the psychopathy factors that survived correction for multiple comparisons. In the right hemisphere, SRP-4 F1 was negatively associated with GMV in a cluster centered on the rostral middle frontal gyrus, and spanning the fronto-marginal gyrus, and the orbital gyrus. In the left hemisphere, SRP-4 F1 was negatively associated with GMV in a cluster centered on the pericalcarine region and spanning the cuneus and lingual gyrus (Figure 1). No clusters survived correction for multiple comparisons for the SRP-4 F2 analysis.

The interaction of the psychopathy factors (SRP-4 F1 × SRP-4 F2) was negatively associated with GMV in two cortical clusters and positively associated with GMV in one cortical cluster (Table 1 and Figure 2). The first cluster in the left hemisphere was negatively associated with the interaction, peaked in the superior parietal lobule and included the angular gyrus, precuneus, paracentral lobule and superior frontal gyrus (Cluster A). The second cluster in the left hemisphere was positively associated with the interaction, peaked in the lingual gyrus and spanned the cuneus (Cluster B). The third cluster in the right hemisphere was negatively associated with the interaction, peaked in the supramarginal gyrus and spanned the angular gyrus, anterior transverse temporal gyrus, superior parietal lobule, lateral aspect of the superior temporal gyrus and temporal plane of the superior temporal gyrus (Cluster C). For clusters A and C, as scores on F2 increased, the association between SRP-4 F1 and cortical volume became more strongly negative. Specifically, for these clusters, the correlations between SRP-4 F1 and cortical GMV were positive at low values of F2 (–0.5 SD, *rs* = 0.16–0.28) and negative at high values of F2 (+0.5 SD, *rs* = –0.31 to –0.46). For Cluster B, the association was the opposite; as scores on F2 decreased, the association between SRP-4 F1 and cortical volume became more strongly positive. Specifically for Cluster B, the correlation between SRP-4 F1 and cortical GMV was negative at low values of F2 (–0.5 SD, $r = -0.47$) and positive at high values of F2 (+0.5 SD, $r = 0.35$).

Next, we examined whether the observed findings could be accounted for primarily by history of lifetime substance use, given the positive association between the impulsive-antisocial traits of psychopathy and substance use. All reported findings remained significant when this variable was included as a covariate in the models.

## Psychopathic trait relations with subcortical volumes

Next, we examined associations between the psychopathy factors and subcortical GMV. The results are displayed in Table 2. The multivariate effect was significant for SRP-4 F1 [$F(10, 139) = 1.98$, $P = 0.04$], and this effect remained significant with substance use included in the model [$F(10, 138) = 1.96$, $P = 0.04$]. SRP-4 F1 was significantly inversely related to GMV in bilateral hippocampus (left: $\beta = -0.14$, $SD = 0.07$, $P = 0.04$; right: $\beta = -0.14$, $SD = 0.07$, $P = 0.04$) and marginally related to left caudate GMV ($\beta = -0.14$, $SD = 0.08$, $P = 0.05$). The addition of substance use as a covariate in the model strengthened the relationship between SRP-F1 and caudate GMV, such that it was significantly inversely related to volume in left caudate ($\beta = -0.16$, $SD = 0.07$, $P = 0.03$). No multivariate main effect was observed for SRP-4 F2 in relation to subcortical GMV.

A significant SRP-4 F1 × SRP-4 F2 interaction did emerge in the multivariate analysis [$F(10, 138) = 2.30$, $P = 0.02$], and this effect remained significant with substance use in the model [$F(10, 137) = 2.28$, $P = 0.02$]. The interaction effect was significantly associated with GMV in bilateral amygdala (left: $\beta = -0.98$, $SD = 0.46$, $P = 0.03$; right: $\beta = -1.62$, $SD = 0.40$, $P < 0.001$) and bilateral hippocampus (left: $\beta = -0.87$, $SD = 0.41$, $P = 0.03$; right: $\beta = -1.20$, $SD = 0.41$, $P = 0.004$). To decompose the interaction effect, we conducted follow-up analyses and found that the multivariate test for SRP-4 F1 was significant at high levels of SRP-4 F2 (+0.5 SD) [$F(4, 36) = 6.54$, $P = 0.001$], but not low levels of SRP-4 F2 (–0.5 SD) [$F(4, 32) = 0.76$, $P = 0.56$]. At high values of SRP-4 F2, SRP-4 F1 was negatively associated with bilateral amygdala and hippocampal volume, whereas it was positively related to bilateral amygdala and hippocampal volume at low levels of SRP-4 F2 (Figure 3).

# Discussion

Given that dimensions of psychopathic traits are theorized to index separable etiological pathways to criminal behavior, and there is evidence that these traits are at least partly neurobiologically instantiated (Hofhansel *et al.*, 2020), we used a multidimensional framework to examine unique associations between the psychopathy factors and GMV. Consistent with research indicating individuals who score high on both the affective-interpersonal (F1) and impulsive-antisocial dimensions (F2) of psychopathy are neurobiologically distinct from those high (Figure 2) on only one

**Table 1.** Gray matter volume clusters showing a significant relationship with F1 × F2 psychopathic traits

| Cluster No. | Hemisphere | Annotation | Peak *F*-value | Peak MNI (*x*,*y*,*z*) | No. of vertices | Cluster size ($mm^2$) |
|---|---|---|---|---|---|---|
| A | L | SPL<br>AG<br>Precuneus<br>PCL<br>SFG | −4.07 | −30.8, −39.6, 44.6 | 10 141 | 4271 |
| B | L | LG<br>Cuneus | 4.51 | −6.1, −84.4, −5.3 | 2834 | 2157 |
| C | R | SMG<br>AG<br>ATTG<br>SPL<br>STG-TP<br>STG-L | −3.12 | 55.0, −39.0, 41.9 | 7726 | 3283 |

N = 156. All clusters survived Monte Carlo Simulation correction for multiple comparisons ($P < 0.05$). Covariates included age, sex, verbal IQ, handedness and eTIV. RH = right hemisphere. LH = left hemisphere. AG = angular gyrus. ATTG = Anterior transverse temporal gyrus. LG = lingual gyrus. PCL = paracentral lobule. SFG = superior frontal gyrus. SMG = supramarginal gyrus. SPL = superior parietal lobule. STG-L = lateral aspect of the superior temporal gyrus. STG-TP = temporal plane of the superior temporal gyrus.

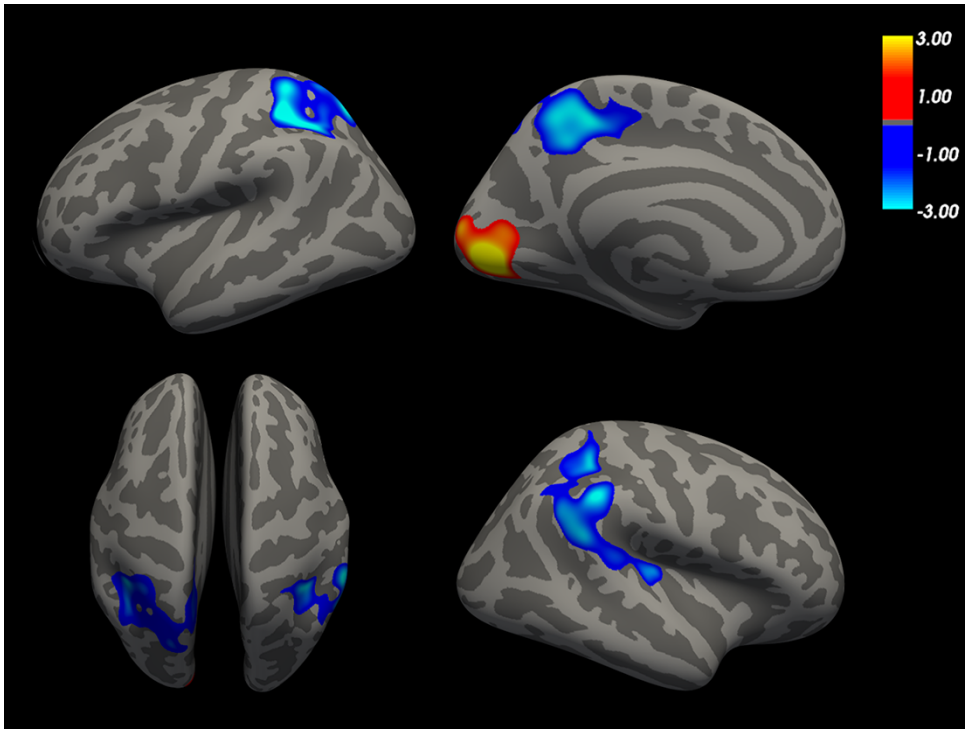


**Fig. 2.** F1 × F2 predicts lower cortical surface volume in the left and right hemispheres, after controlling for age, sex, VIQ, handedness and eTIV.

psychopathy factor (Seara-Cardoso *et al.*, 2012), we found that GMV in several brain regions was associated with the interaction of the factor-level traits. Specifically, the interaction of the affective-interpersonal and impulsive-antisocial trait dimensions was associated with reduced GMV in clusters of the right temporal lobe, bilateral parietal lobes, amygdala and hippocampus. Notably, each of these findings indicated that decrements in GMV increased as individuals high on impulsive-antisocial traits showed increasing levels of the affective-interpersonal traits. The interaction was also associated with greater volume in a cluster of the left occipital lobe. These findings underscore the potentially unique etiology of individuals who manifest elevations on both of the primary psychopathy dimensions. Interestingly, we also found that the unique variance associated with the affective-interpersonal trait dimension was negatively associated with GMV in one cluster centered on the rostral middle frontal gyrus and one cluster of the pericalcarine region, suggesting lower cortical surface volume in these regions among individuals who score high selectively on the callousness, superficial charm and deceitfulness that are core features of the disorder. Overall, our results provide new evidence that the neurobiological profiles of individuals high on both the affective-interpersonal and impulsive-antisocial trait dimensions are different from those who score high selectively on one trait dimension, findings that are important for potentially parsing etiological heterogeneity in psychopathy.

Although most research that has examined neurobiological variation associated with the affective-interpersonal and

**Table 2.** SRP-4 psychopathy traits relate to subcortical gray matter volumes in bilateral amygdala and hippocampus

| | Multivariate Test | | | Between-Subjects Effects (ß-Statistic) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Amygdala | | Hippocampus | | Caudate | | NAcc | | Putamen | |
| | *F Statistic* | *P-Value* | $np^2$ | *L* | *R* | *L* | *R* | *L* | *R* | *L* | *R* | *L* | *R* |
| *Step 1* | | | | | | | | | | | | | |
| Age | 3.02 | 0.002 | 0.18 | −0.22* | −0.14* | −0.04 | −0.06 | −0.22* | −0.22* | −0.12 | −0.16* | −0.27* | −0.27* |
| Sex | 2.76 | 0.004 | 0.16 | −0.04 | 0.04 | −0.08 | −0.10 | −0.14 | −0.11 | −0.21* | −0.15 | 0.16 | 0.13 |
| Verbal IQ | 2.99 | 0.039 | 0.12 | −0.18* | −0.07 | 0.05 | 0.00 | 0.01 | −0.01 | −0.06 | 0.01 | −0.02 | 0.03 |
| eTIV | 21.06 | <0.001 | 0.60 | 0.68* | 0.71* | 0.77* | 0.78* | 0.69* | 0.70* | 0.65* | 0.64* | 0.53* | 0.53* |
| Handedness | 0.85 | 0.581 | 0.06 | – | – | – | – | – | – | – | – | – | – |
| *Step 2* | | | | | | | | | | | | | |
| SRP4- F1 Affective- Interpersonal Traits | 1.98 | 0.04 | 0.12 | 0.05 | −0.07 | −0.14* | −0.14* | −0.15 | −0.13 | 0.06 | −0.06 | −0.09 | −0.08 |
| SRP-4 F2 Impulsive Antisocial Traits | 0.87 | 0.57 | 0.06 | – | – | – | – | – | – | – | – | – | – |
| *Step 3* | | | | | | | | | | | | | |
| SRP-4 F1 × SRP-4 F2 | 2.30 | 0.02 | 0.14 | −0.98* | −1.62* | −0.87* | −1.20* | −0.53 | −0.71 | −0.41 | −0.22 | −0.35 | −0.63 |

*Note:* $N = 156$. SRP-4 = Self-Report Psychopathy Scale–4th Ed. NAcc = Nucleus Accumbens. L = Left. R = Right. Between-subjects effects are only presented for significant multivariate effects. All predictor and outcome variables were standardized. *$P < 0.05$.

impulsive-antisocial traits has considered these dimensions in an additive fashion, there is a growing body of research that suggests these traits may be better conceptualized as interactive (Walsh and Kosson, 2008; Sprague *et al.*, 2012; Verona *et al.*, 2012). We found interactive effects of the affective-interpersonal and impulsive-antisocial dimensions on GMV, such that reductions in GMV were more pronounced in individuals high on both primary psychopathy traits relative to those who were high on only one trait dimension in three cortical clusters. One cluster centered on the superior parietal lobule and included the angular gyrus, precuneus, paracentral lobule and superior frontal gyrus. The superior parietal lobule has been previously associated with violent behavior and psychopathic traits (Yang *et al.*, 2015; Lamsma *et al.*, 2017) and is known to play a role in many sensory and cognitive processes (Wang *et al.*, 2015). The angular gyrus plays a role in memory and reasoning and is thought to be part of a 'moral neural circuit' that has been shown to function abnormally in psychopathic individuals (Glenn *et al.*, 2009). The paracentral lobule is part of a sensorimotor network (Spasojević *et al.*, 2013) that has previously been found to function abnormally with respect to psychopathic traits (Espinoza *et al.*, 2018). The superior frontal gyrus has been previously found to support attention (Li *et al.*, 2013), perception and working memory (du Boisgueheneuc *et al.*, 2006), so it follows that lower GMV (Cope *et al.*, 2012) and abnormal functioning of this region (Sadeh *et al.*, 2013b) have both been previously associated with psychopathic traits (particularly the affective-interpersonal dimension), consistent with theories that attentional deficits in psychopathy contribute to core features of the disorder (Smith and Lilienfeld, 2015). The precuneus is an important node of the default mode network (Fransson and Marrelec, 2008) and likely contributes to disturbed self-referential processing and moral decision making in individuals with psychopathic traits (Juárez *et al.*, 2013; Freeman *et al.*, 2014; Espinoza *et al.*, 2018).

The second cluster in the left occipital lobe was positively associated with the interaction, peaked in the lingual gyrus and spanned the cuneus. While abnormalities within occipital regions involved in basic visual processing are not traditionally predicted by neurobiological models of psychopathy, recently meta-analyses have begun to show structural alterations in these areas (De Brito *et al.*, 2021) that may relate to functional findings. Some recent fMRI studies have reported reduced functional connectivity between the occipital cortex and other cortical (Juárez *et al.*, 2013) and subcortical structures (Contreras-Rodríguez *et al.*, 2015) in individuals with psychopathic traits, which other authors have speculated could compromise normal processes that upregulate activity in these regions during emotional processing tasks (Anderson *et al.*, 2017; Espinoza *et al.*, 2018), but these hypotheses require further testing. Future research is needed to examine the relationship between greater GMV and functional connectivity within the context of the whole-brain networks (e.g. executive control, salience), as it is likely that relatively greater or lower GMV can contribute to abnormalities in brain functioning.

The third cluster in the right hemisphere was negatively associated with the interaction, peaked in the supramarginal gyrus and spanned the superior parietal lobule, angular gyrus, anterior transverse temporal gyrus, lateral aspect of the superior temporal gyrus and temporal plane of the superior temporal gyrus. Reductions in GMV of the supramarginal gyrus have been previously linked to psychopathic traits, and this region is thought to be a part of the 'theory of mind' network (Hofhansel *et al.*, 2020). Reduced volume in the temporal cortex associated with psychopathic traits is also consistent with past research (Muller

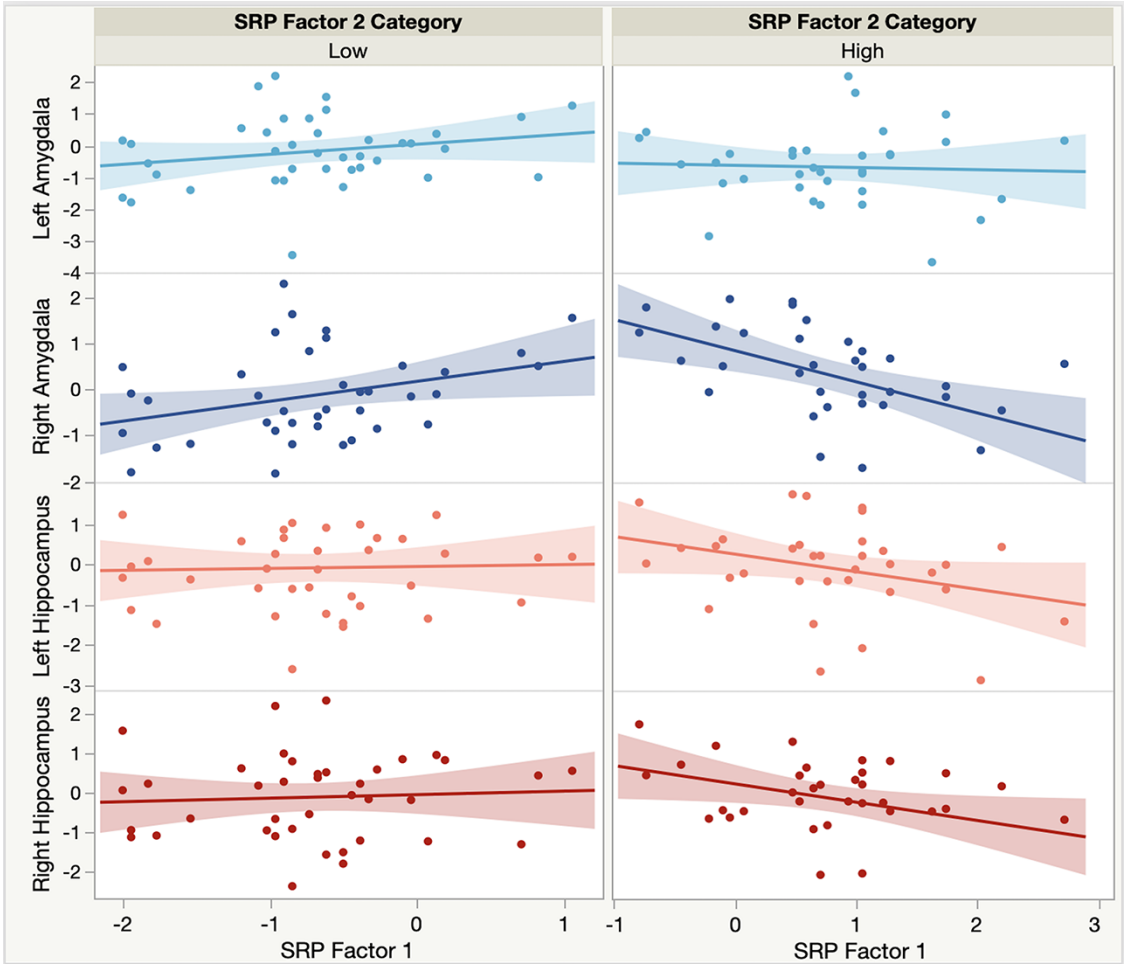


**Fig. 3.** Associations between F1 psychopathic traits and subcortical volumes, split by high and low levels of F2.

*et al.*, 2008; Ermer *et al.*, 2012; Ly *et al.*, 2012; De Brito *et al.*, 2021) and converges with functional data showing reduced activation in the superior temporal lobe of psychopathic individuals during moral judgement tasks (Harenski *et al.*, 2010).

Given the preponderance of research implicating alterations in several subcortical structures in the etiology of psychopathy, we examined the unique and interactive effects of F1 and F2 traits on amygdalar, hippocampal and striatal (caudate, putamen, nucleus accumbens) volumes. We found that F1 psychopathic traits were negatively associated with bilateral hippocampal volumes, which is in line with previous research (Laakso *et al.*, 2001; Boccardi *et al.*, 2010). Models that characterize psychopathy as a disorder of the paralimbic system (Kiehl, 2006) posit that damage to paralimbic regions such as the hippocampus give rise to the abnormal fear conditioning observed in psychopathic individuals (Glenn and Raine, 2008), particularly with respect to the affective-interpersonal dimension. It is not known when in development these abnormalities arise so longitudinal studies establishing causality are still needed.

Interestingly, we did not find main effects of F1 or F2 on amygdala volumes in either hemisphere. This was somewhat surprising given prior research showing negative relationships between the affective-interpersonal traits and amygdala volume (Glenn and Raine, 2008; Pardini *et al.*, 2014). A recent meta-analysis also did not find amygdala volume differences to be related to psychopathy dimensions (De Brito *et al.*, 2021), possibly suggesting that psychopathy may be more reliably associated with functional abnormalities of the amygdala rather than structural ones. Similarly, we did not find any associations between F1 or F2 traits and striatal volumes, which is in contrast to previous research showing associations particularly with respect to the impulsive-antisocial dimension (Korponay *et al.*, 2017; De Brito *et al.*, 2021). In the integrated emotions system model of psychopathy (Blair, 2013), abnormal functioning of reward regions such as the striatum, particularly the caudate is thought to be central to perpetuating the reward-processing abnormalities that characterize the impulsive-antisocial component of the disorder. Our lack of findings may be at least partially explained by evidence showing that examining the unique variance of affective-personal traits (controlling for impulsive-antisocial traits) is different than examining bivariate associations without controlling for the overlap between the psychopathy dimensions (Hicks and Patrick, 2006; Lynam *et al.*, 2006; Baskin-Sommers *et al.*, 2009), with the former approach (taken here) providing insight into the distinct neurobiological correlates of these traits, and thus, how neurobiology may contribute to the manifestation of these psychopathic phenotypes. Future investigations will be needed to tease apart structural variation in the brain as it relates to these separate factors, although the current findings point to at least one distinct neurobiological correlate associated with scoring high selectively on the affective-interpersonal trait dimension.

Tests of the interaction of the affective-interpersonal and impulsive-antisocial traits revealed that the interactive effect

of these traits was significantly associated with amygdala and hippocampal volumes in both hemispheres. Specifically, findings showed that the relationship between F1 psychopathic traits and bilateral amygdala and hippocampal volumes depended on scores on F2, such that as F2 scores increased, the relationship between F1 scores and amygdala volumes became more strongly negative. This extends previous literature showing that there is an inverse relationship between amygdala volume and psychopathy (Weber *et al.*, 2008; Yang *et al.*, 2009a; Ermer *et al.*, 2012; Boccardi *et al.*, 2013). Additionally, these results suggest that lower amygdala and hippocampal volume size could be an indicator of dysregulation of both emotional (F1) and behavioral (F2) systems, instead of primarily affective-interpersonal traits, which has been previously suggested (Yang *et al.*, 2009b).

Although much research has demonstrated that psychopathic traits are associated with neuroanatomical abnormalities, relatively less research has examined the continuous relationship between trait severity of the specific psychopathic factor dimensions and GMV. To address this gap, the current study investigated the unique variance associated with the affective-interpersonal and impulsive-antisocial trait dimensions on brain volumes. We identified a negative relationship between Factor 1 psychopathic traits and GMV in left occipital and right prefrontal (orbitofrontal) regions. The GMV reductions we found in prefrontal and orbitofrontal regions in relation to Factor 1 psychopathic traits are well aligned with previous research (Hofhansel *et al.*, 2020; De Brito *et al.*, 2021) and with neurobiological models of psychopathy, including the integrated emotions systems model (Blair, 2005) as well as the paralimbic hypothesis (Kiehl, 2006). Specifically, abnormalities in the functioning of prefrontal and orbitofrontal regions are thought to give rise to the deficits in decision making (Koenigs, 2012), reward processing (Murray *et al.*, 2018) and moral judgment (Pujol *et al.*, 2012) that characterize the disorder. GMV associations between occipital regions and psychopathic traits are relatively new and understudied (De Brito *et al.*, 2021), but may relate to functional studies showing that individuals with psychopathic traits display abnormal responding in occipital regions during affective processing tasks (Muller *et al.*, 2003).

Overall, the current study identified several regions thought to be part of a limbic circuit that is central to neural dysfunction in psychopathy (e.g. orbitofrontal cortex, supramarginal gyrus, amygdala, hippocampus) (Anderson and Kiehl, 2014). However, we did not find any GMV reductions in a few commonly implicated paralimbic regions, including the anterior or posterior cingulate cortices or temporal pole (Ermer *et al.*, 2012). We also did not observe any main associations between F2 traits (controlling for F1) and GMV, which differs from some prior research that has linked these traits to alterations in frontostriatal regions, the supplementary motor area and paralimbic regions (e.g. OFC, insula) (Leutgeb *et al.*, 2015; Korponay and Koenigs, 2021). However, the majority of the studies presented in the psychopathy literature contain predominantly male samples and often contrast psychopathic with non-psychopathic individuals (Koenigs *et al.*, 2011; Hofhansel *et al.*, 2020). These could be a few of the factors that explain the differences we observed. Additionally, very few published studies to our knowledge have used the SRP to examine GMV (Pardini *et al.*, 2014) and even fewer have examined the SRP factors using a whole-cortex approach (Johanson *et al.*, 2020). More research is needed to clarify how psychopathic trait relations with GMV differ in clinical *vs* community samples, or as a function of biological sex, sample characteristics that may have contributed to differences between the current results and prior research. A recent systematic review has suggested that the neural mechanisms underlying impulsive psychopathic traits differ in community and forensic psychopathy samples (Korponay and Koenigs, 2021). Our examination of the impulsive-antisocial traits as a continuous variable in a sample with psychopathy scores that fell between the ranges typically observed in community and clinical samples may have obscured these differences and, for example, contributed to our null findings for the main effect of F2 traits. Future replications of these analyses, as well as research directly comparing the neural correlates of impulsivity in psychopathic and non-psychopathic groups, are needed to clarify the neural regions involved in pathological impulsivity *vs* those that may only be relevant to psychopathic traits studied at the community level.

Findings from the current study must be interpreted in the context of its limitations. First, the use of a community mixed-gender sample, while filling an important gap, does not allow for the results to be generalizable to individuals diagnosed with psychopathy. Additionally, psychopathic traits were assessed with a self-report measure rather than the Psychopathy Checklist-Revised, which is the gold-standard method. Furthermore, we only examined several subcortical regions of interest, so a future direction of this research should be to examine how psychopathic traits relate to variation across all subcortical structures. The study also had several notable strengths, first, the investigation of psychopathic traits dimensionally in a relatively large, mixed-gender sample of adults recruited from the general community. The sample was both socioeconomically and ethnically diverse and reported relatively high rates of lifetime involvement in the criminal justice system, making it well-suited to study the proposed aims.

In conclusion, the current study adds to a growing body of literature that focuses on examining the factor-level interactions of the psychopathy dimensions on neurobiological characteristics. Results provide new insight into the neurobiological profiles of individuals who manifest both the affective-interpersonal and impulsive-antisocial dimensions of psychopathy and how they differ from individuals high on only one trait dimension. Further work examining psychopathic traits both interactively and specifically will be necessary to understand the direct link between the severity of psychopathic behavior and possible biological markers.

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## Conflict of interest

None declared.

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