# Cortical structure in relation to empathy and psychopathy in 800 incarcerated men

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**Keywords**

Brain structure; Empathy; Psychopathy; Multivariate prediction; Structural gradients; Antisocial behavior

## Abstract

**Background.** Reduced empathy is a hallmark of individuals with high psychopathy, who are overrepresented among incarcerated men. However, a comprehensive mapping of cortical structure in relation to empathy and psychopathy is lacking.

**Methods.** In 804 incarcerated adult men, we administered the Perspective Taking (IRI-PT) and Empathic Concern (IRI-EC) subscales of the Interpersonal Reactivity Index, Hare Psychopathy Checklist-Revised (PCL-R; Interpersonal/Affective [F1] and Lifestyle/Antisocial [F2] factors), and T1-weighted MRI to quantify cortical thickness (CT) and surface area (SA). We also included the male sample from the Human Connectome Project (HCP; N = 501) to probe the replicability of structural-covariance gradients.

**Results.** PCL-R F1 was uniquely negatively related to IRI-EC, while PCL-R F2 was uniquely negatively related to IRI-PT. Cortical structure was not related to either IRI subscale, although there was effect-size differentiation by cytoarchitectonic class and/or functional network. CT was related to PCL-R F1 (mostly positively), SA was related to both PCL-R factors (only positively), and both cortical indices demonstrated out-of-sample predictive utility for PCL-R F1. The high-psychopathy group (N = 178) scored uniquely lower on IRI-EC while having increased SA (but not CT); across the cortex, effect sizes were largest in the paralimbic class and somatomotor network, and meta-analytic task-based activations corroborated affective/sensory importance. Finally, the total sample revealed anterior-posterior gradients of covariance, which were replicated in the HCP sample. In the high-psychopathy group, the gradient of CT (but not SA) was globally compressed.

**Conclusions.** Most notably, high-psychopathy men had reduced empathic concern, increased SA, and compressed macroscale organization of CT.

## Introduction

Empathy is widely recognized as comprising desirable psychological traits that allow us to understand another person (cognitive empathy), share their emotion (affective empathy), and care for them (empathic concern) (1-6). Reduced empathy, particularly affective empathy and/or empathic concern, is a hallmark of individuals with high psychopathy (7-12). Psychopathy is a constellation of interpersonal/affective traits (e.g., lack of attachment and remorse, beyond lack of empathy) and lifestyle/antisocial traits (e.g., impulsivity, parasitic lifestyle, criminal versatility), as operationalized by Factors 1 and 2, respectively, of the Hare Psychopathy Checklist-Revised (PCL-R) (13-15). The prevalence of high psychopathy based on the PCL-R (typically, $\geq$ 30 out of 40 points) approximates 1.2% in the general population; it is higher in incarcerated individuals (up to 25%) and in males/men than females/women (16-18). Because psychopathy incurs societal costs that reach hundreds of billions of USD per annum through violence, crime, and recidivism (19), we need a better neuroscientific understanding of psychopathic traits, such as reduced empathy, to improve their treatment in the long run (20-22).

Empathy, which is often measured with the Interpersonal Reactivity Index (IRI) (23-25), has garnered substantial interest in cognitive neuroscience. However, this is less evident from a brain-structural perspective (with a meta-analysis still missing; e.g., [26-28]) than from a brain-functional one (e.g., [29-31]; for meta-analyses, see, e.g., [32-34]). Across these functional meta-analyses, cognitive empathy preferentially implicates the medial prefrontal cortex, temporoparietal junction, and precuneus compared to affective empathy, which in turn preferentially implicates the insula, midcingulate cortex, and inferior frontal gyrus. Empathic concern notably involves reward-related activation in ventromedial-prefrontal and subcortical regions, such as the ventral striatum (3,35,36; for a meta-analysis, see [37]). Given this regional separability, cognitive empathy maps more closely onto the default-mode network while affective empathy onto the somatomotor and ventral-attention/salience networks (34; see also [37] for the role of the latter network in empathic concern). Such neurobiological characterizations of empathy in the general population are valuable for characterizing reduced empathy in high-psychopathy individuals and thus advancing our understanding of both empathy and psychopathy (38).

A growing body of research on criminal male psychopathy suggests atypical functional processing in relation to empathy (e.g., [39-44]) on top of widespread differences in brain function, including at the network level (e.g., [45-50]; for meta-analyses, see [51-53]). These functional differences occur alongside structural differences (e.g., [54-59]), with most studies focusing on gray-matter volume (GMV) and noting reductions therein (for a meta-analysis, see [60]; for a meta-analysis across antisocial populations, see [61]). One conceptual attempt to synthesize this multimodal literature concerns paralimbic regions (62; for subsequent reviews, see [21,63,64]). Having laminar differentiation, these regions form a ring at the base of the cortex to cover some of the affective-empathy hubs (i.e., portions of the insula and cingulate cortex) (65-67). While high results heterogeneity in the neuroimaging of psychopathy should be acknowledged, in part owing to low statistical power (68), integrating such cytoarchitectonic alongside functional-organizational (69) information may enhance the interpretability of these results.

The PCL-R factors may be differentially related not only to empathy (12) but also GMV (60). Since cortical thickness (CT) and surface area (SA) contribute to GMV through largely independent

genetic (70) and developmental (71) mechanisms, it is crucial to distinguish between them, as they may also be differentially related to the PCL-R factors. Furthermore, SA was shown to be more sensitive than CT to broadly construed antisocial behavior in community (72) and mixed (community/incarcerated [73]) samples. Yet, despite these pressing arguments for acknowledging the cortical distinction, SA has not yet been examined in relation to psychopathy – and this is also true of empathy, which exposes remarkable gaps that warrant joint examination. This examination could further benefit from a multivariate framework with predictive modeling to support discovery and generalizability (74-76).

Finally, it remains unknown whether psychopathic differences in the brain extend to gradients, that is, global patterns of covariance (including structural covariance [77-79]) – an opportunity for insights beyond more traditional frameworks by embedding cortical topography in a low-dimensional space (80-82). Based on meta-analytic data, CT gradients differ across major psychiatric conditions in a transdiagnostic fashion (83-85) that dovetails with differences along the primary axis of functional connectivity in schizophrenia (86), autism (87), or depression (88). In these conditions, the unimodal-transmodal gradient was observed to be compressed (as opposed to expanded [89]). This compression corresponds to a smaller gradient range and suggests reduced differentiation between its ends, with sensorimotor regions on one end and association regions on the other having more similar connectivity patterns (90). Importantly, an anterior-posterior compression was also observed for CT in schizophrenia (91), and it needs to be established whether high-psychopathy individuals may exhibit any differences in macroscale structural organization.

In sum, a comprehensive mapping of CT and SA in relation to empathy and psychopathy is lacking. We addressed this gap in a large sample of incarcerated adult men (N = 804) through five overarching questions (see *Statistical analysis and hypotheses* in the Supplement):

Q1: How is psychopathy related to empathy, considering the multidimensional nature of both constructs and their potential for unique relationships?

Q2: How is cortical structure related to empathy and psychopathy, considering the cortical distinction?

Q3: Does cortical structure predict empathy and psychopathy in out-of-sample individuals? This question complemented the univariate analyses for Q2 by leveraging a multivariate framework with a train-test split and cross-validation.

Q4: How does cortical structure differ between individuals with high and low psychopathy? This question complemented Q2 by leveraging a categorical framework.

Q5: How does structural covariance differ by psychopathy group? Based on the same categorical framework, this question centered on psychopathic differences in gradients whose replicability was probed using independent data from the general population.

## Methods and Materials

### Participants

Out of 912 adult men (gender self-reported) recruited by the Mind Research Network (MRN) from correctional facilities in the southwestern and midwestern United States, we included N = 804 who met these sequential criteria: (1) passed MRI quality control ($N_{excluded}$ = 105); (2) had data on empathy, psychopathy, age, and IQ ($N_{excluded}$ = 1); and (3) had an IQ of at least 70 ($N_{excluded}$ = 2) (for participant characteristics, see *Table **1***). Previous MRN work with this population suggests an overrepresentation of violent-offense convictions (92). All participants gave written informed consent, and all research protocols were approved by the Institutional Review Board of the University of New Mexico or the Ethical and Independent Review Services for data collection post June 2015.

To probe the replicability of structural-covariance gradients in the total sample, we included the male sample from the Human Connectome Project (HCP) Young Adult S1200 release (93-95) with structural-MRI and IQ data (N = 501). Participant characteristics are provided in *Supplementary Table **S1***; note that this sample did not have our measures of empathy and psychopathy available.

**Table 1. Participant characteristics**

| | Total | Low psychopathy | High psychopathy | Cohen's D, P |
|---|---|---|---|---|
| N | 804 | 289 | 178 | – |
| Age | 33.78 ± 8.23 | 34.20 ± 8.51 | 33.69 ± 8.19 | -0.06, 0.521 |
| *Range* | [18.75, 62.83] | [18.75, 60.56] | [19.47, 62.83] | – |
| IQ | 97.88 ± 13.14 | 98.56 ± 13.26 | 100.03 ± 12.68 | 0.11, 0.277 |
| *Range* | [71, 137] | [72, 134] | [72, 137] | – |
| PCL-R total | 22.85 ± 7.06 | 15.15 ± 3.83 | 32.04 ± 1.96 | 5.19, 7e-74 * |
| *Range* | [3.20, 38] | [3.20, 20] | [30, 38] | – |
| PCL-R F1 | 7.90 ± 3.61 | 4.74 ± 2.58 | 12.14 ± 1.86 | 3.17, 2e-70 * |
| *Range* | [0, 16] | [0, 12] | [8, 16] | – |
| PCL-R F2 | 12.79 ± 4.01 | 8.97 ± 3.26 | 16.85 ± 1.89 | 2.80, 5e-68 * |
| *Range* | [1.10, 20] | [1.10, 17] | [11, 20] | – |
| Race (W) | 534 | 225 | 100 | 3e-07 * |
| SU | 21.63 ± 21 | 19.44 ± 19.81 | 22.34 ± 19.44 | 0.15, 0.022 * |
| *Range* | [0, 158] | [0, 107] | [0, 111] | – |
| Adj. SU | 7.01 ± 3.64 | 6.40 ± 3.74 | 7.43 ± 3.55 | 0.28, 0.008 * |
| *Range* | [0, 18.89] | [0, 15.19] | [0, 18.89] | – |
| TIV | 1.58e+06 ± 1.5e+05 | 1.60e+06 ± 1.4e+05 | 1.58e+06 ± 1.6e+05 | -0.13, 0.293 |
| *Range* | [9.6e+05, 2.0e+06] | [1.1e+06, 2.0e+06] | [1.1e+06, 1.9e+06] | – |
| Euler no. | 11.88 ± 4.95 | 12.20 ± 4.99 | 12.31 ± 5 | 0.02, 0.753 |
| *Range* | [0, 24] | [3, 24] | [3, 23] | – |

*Note.* Given are means and standard deviations (or frequencies for race) as well as Cohen's Ds for the high-psychopathy (PCL-R $\geq$ 30) versus low-psychopathy (PCL-R $\leq$ 20) groups, with P-values derived from Wilcoxon's rank-sum test (or Pearson's $\chi^2$ test for race); * = P < 0.05, uncorrected. IQ = full-scale IQ estimate based on the Vocabulary and Matrix Reasoning subtests of the Wechsler Adult Intelligence Scale-III or Wechsler Abbreviated Scale of Intelligence-II; PCL-R F1 = Interpersonal/Affective factor; PCL-R F2 = Lifestyle/Antisocial factor (N = 778); Race (W) = White (versus non-White; N = 789); SU = total years of substance use based on the Addiction Severity Index (ASI; N = 748); Adj. SU = age-corrected and square-root-transformed (to correct for opportunity to use and skewness) total years of substance use based on the ASI (N = 748); TIV = estimated total intracranial volume [$mm^3$]; Euler no. = total number of topological defects in the cortical surface prior to fixing in the FreeSurfer pipeline (to be treated as a measure of data quality).

**MRI**

On the grounds of the correctional facilities, high-resolution T1-weighted MRI scans were acquired with the MRN mobile scanner (i.e., 1.5-T Siemens MAGNETOM Avanto with a 12-channel, multi-echo MPRAGE pulse sequence). The scanning parameters were as follows: repetition time = 2,530 ms; echo times = 1.64 ms, 3.50 ms, 5.36 ms, and 7.22 ms; inversion time = 1,100 ms; flip angle = 7°; slice thickness = 1.3 mm; matrix size = 256 × 256, yielding 128 sagittal slices with an in-plane resolution of 1.0 mm × 1.0 mm.

Each scan underwent the standard "recon-all" pipeline in FreeSurfer version 7.4.1 ([96]; https://surfer.nmr.mgh.harvard.edu/) and was parcellated in the "HCP-MMP1.0" atlas (97). For quality control and HCP data, see *MRI* in the Supplement.

**Empathy**

Cognitive empathy and empathic concern were measured with the Perspective Taking (IRI-PT) and Empathic Concern (IRI-EC) subscales of the Interpersonal Reactivity Index (IRI [23]). The IRI is a self-report questionnaire of trait empathy widely used in both community and incarcerated samples (note that across these samples, IRI-EC is often labeled a measure of affective empathy [11,12,98-101]). The remaining Fantasy and Personal Distress subscales were not included, as they are less frequently used to distinguish cognitive empathy from affective empathy and empathic concern (98,99), and are less related to psychopathy, if at all (11). IRI-PT assesses the "tendency to spontaneously adopt the psychological point of view of others" while IRI-EC the "feelings of sympathy and concern for unfortunate others" ([23], pp. 113–114). Each subscale includes seven items scored on a five-point scale ranging from "Does not describe me well" (zero points) to "Describes me very well" (four points) (for all items, see *Supplementary Table **S2***). Possible scores thus range from 0 to 28 points per subscale, with higher scores indicating higher empathy. See also *Internal consistency* in the Supplement.

**Psychopathy**

Psychopathy was measured with the Hare Psychopathy Checklist-Revised (PCL-R) (14). All PCL-R scores were based on both a semi-structured interview and institutional-file review conducted by the MRN research staff, with a bachelor's degree or higher, following rigorous training designed and supervised by K.A.K. MRN has historically completed independent double-ratings on ~10% of all PCL-R interviews, obtaining excellent rater agreement (intraclass-correlation coefficient = 0.96) (54). The PCL-R includes 20 items that correspond to two factors: Interpersonal/Affective (F1) and Lifestyle/Antisocial (F2) (for all items, see *Supplementary Table **S3***). Each item is scored zero, one, or two points, indicating no evidence, some evidence, and pervasive evidence, respectively. The total score is a sum across the 20 items, thus ranging from 0 to 40 points, with a higher score indicating higher psychopathy. PCL-R total and PCL-R F1 were available for the total sample (i.e., N = 804, where N = 582 and N = 798 had complete item-level data, respectively), while PCL-R F2 was available for N = 778 (where N = 617 had complete item-level data). For items omitted due to insufficient information, we used a prorating formula to estimate the total and factor scores with possible decimals. See also *Internal consistency* in the Supplement.

Following both the PCL-R guideline (14) and extensive work with incarcerated adult males/men (e.g., [39,40,42,43,46,50,102-104]), we defined "high psychopathy" as scoring 30 or above, and "low psychopathy" as scoring 20 or below.

## Results

### Empathy and psychopathy (Q1)

In 804 incarcerated adult men, we first tested for relationships between empathy and psychopathy (Q1). In particular, five continuous variables of interest – IRI-PT, IRI-EC, PCL-R total, PCL-R F1, and PCL-R F2 – were tested for relationships with empathy as the dependent variable, controlling for age and IQ. PCL-R F1 had a negative relationship with IRI-EC, while PCL-R total and PCL-R F2 had negative relationships with both IRI subscales (*Fig.* ***1A-B***, *Supplementary Fig.* ***S1***, and *Supplementary Table* ***S4***). In categorical analyses, the high-psychopathy group compared to the low-psychopathy group scored lower on both IRI subscales, with a larger effect size for IRI-EC, controlling for age and IQ. These relationships somewhat clarified when additionally controlling for the other IRI subscale; PCL-R total and PCL-R F1 were uniquely negatively related to IRI-EC, while PCL-R F2 was uniquely negatively related to IRI-PT. Further, the group difference on IRI-PT became insignificant, while the group difference on IRI-EC remained significant, and this did not change when additionally controlling for race and substance use (*Fig.* ***1C***, *Supplementary Fig.* ***S1***, and *Supplementary Table* ***S5***). For sample-specific correlations, see *Fig.* ***1D***.

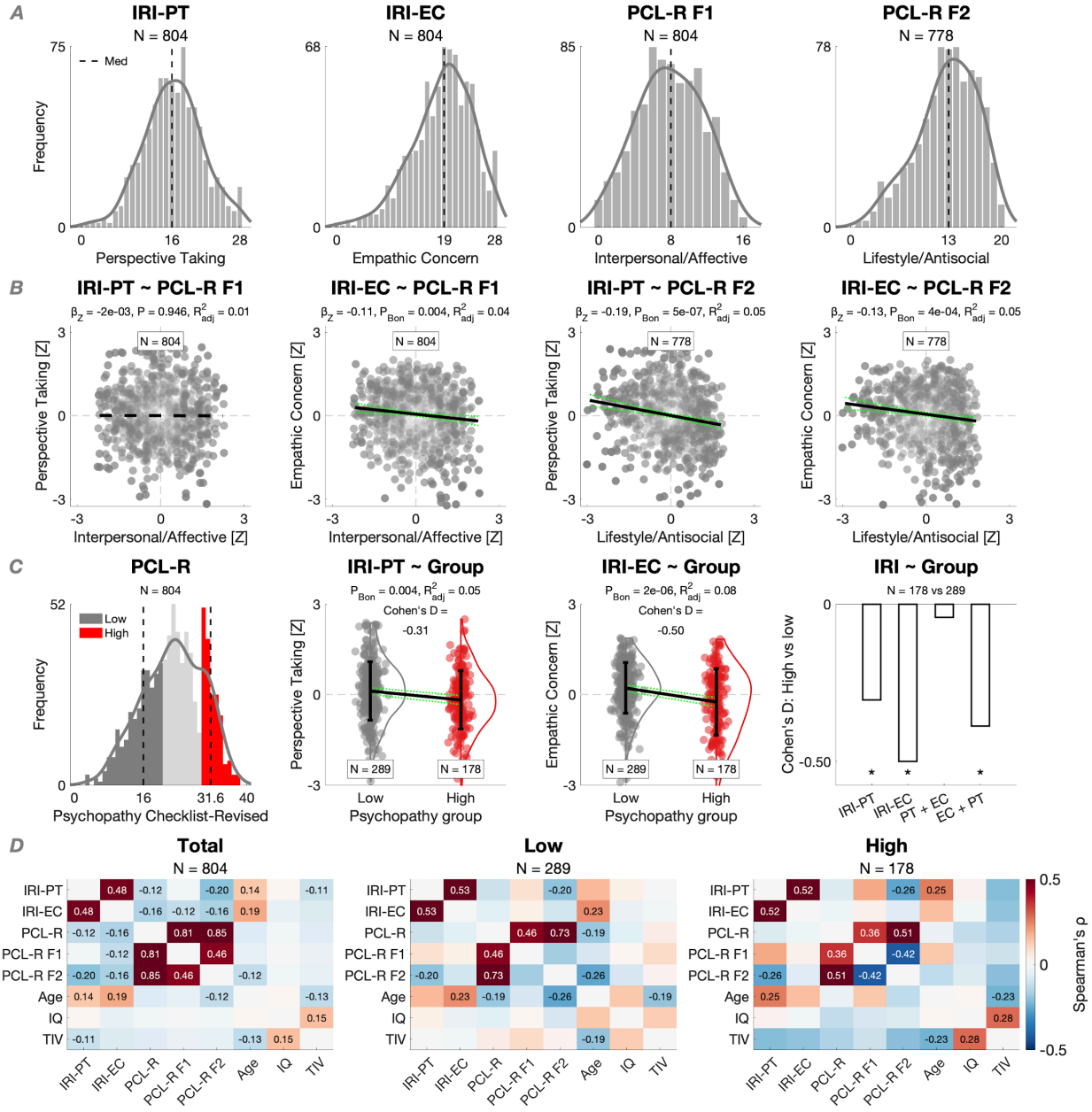


**Figure 1. Empathy and psychopathy.** (A) Distribution of four continuous variables of interest. (B) Negative relationships of PCL-R F1 and PCL-R F2 with IRI-PT and IRI-EC, controlling for age and IQ in a robust linear regression, with Bonferroni correction across the two IRI subscales. (C) From left to right: Distribution of PCL-R total, depicting the low-psychopathy (PCL-R $\leq$ 20; dark gray) and high-psychopathy (PCL-R $\geq$ 30; red) groups; lower scores on IRI-PT and IRI-EC in the high-psychopathy group, controlling for age and IQ, with Bonferroni correction across the two IRI subscales; lower score in the high-psychopathy group on IRI-EC but not IRI-PT when additionally controlling for the other IRI subscale. (D) Sample-specific Spearman's correlation matrices, with numeric effect sizes displayed at $P_{Bon} < 0.05$ following correction across the 28 tests. PCL-R F2 was available for N = 778 in the total sample.

**Cortical structure, empathy, and psychopathy (Q2-Q3)**

Next, we tested for relationships of CT and SA with empathy and psychopathy (Q2). Controlling for age and IQ, CT was not related to either IRI subscale – nor to PCL-R total or PCL-R F2. However, CT in 16 parcels had a positive relationship with PCL-R F1, while CT in six parcels had a negative relationship (*Supplementary Figs.* ***2-3*** and *Supplementary Table* ***S6***). These results were then characterized across the cortex using the well-established frameworks of four cytoarchitectonic classes (66) and seven functional networks (69) (see *Cytoarchitectonic classes and functional networks* in the Supplement). For PCL-R F1, effect sizes were largest in the heteromodal class and frontoparietal network (positive median in both cases), with differentiation by both class (e.g., heteromodal > paralimbic) and network (e.g., frontoparietal > visual). Similarly to CT, SA was not related to the IRI subscales, additionally controlling for total intracranial volume (TIV). In contrast, SA had a positive relationship with PCL-R total in 51 parcels, PCL-R F1 in 103 parcels, and PCL-R F2 in the same three parcels in the right superior-temporal/auditory cortex (*Fig.* ***2***, *Supplementary Fig.* ***S2***, and *Supplementary Tables* ***S7-S9***). Consistently for the three variables, effect sizes were largest in the paralimbic class and somatomotor network (positive median in all cases), with differentiation by class for PCL-R total and PCL-R F2 (e.g., paralimbic > heteromodal), and by network for all three variables (e.g., somatomotor > dorsal-attention).

We then ran sensitivity and multivariate analyses for the IRI subscales and PCL-R variables. The null CT and SA findings for the IRI subscales did not change when leveraging their psychometrically modified versions (see *Internal consistency* in the Supplement; *Supplementary Fig.* ***S4***), while the positive CT and/or SA findings for PCL-R total and PCL-R F1 (and less so for PCL-R F2) remained highly consistent when taking two alternative approaches to structural-data quality control (see *MRI* in the Supplement; *Supplementary Fig.* ***S5***). Furthermore, addressing Q3, relationships of SA with PCL-R total and of CT and SA with PCL-R F1 (but not PCL-R F2) were corroborated in a multivariate framework with a train-test split and cross-validation (see *Multivariate prediction* in the Supplement), explaining ~5%, ~6%, and ~8% of the out-of-sample variance, respectively (*Fig.* ***3*** and *Supplementary Fig.* ***S6***).

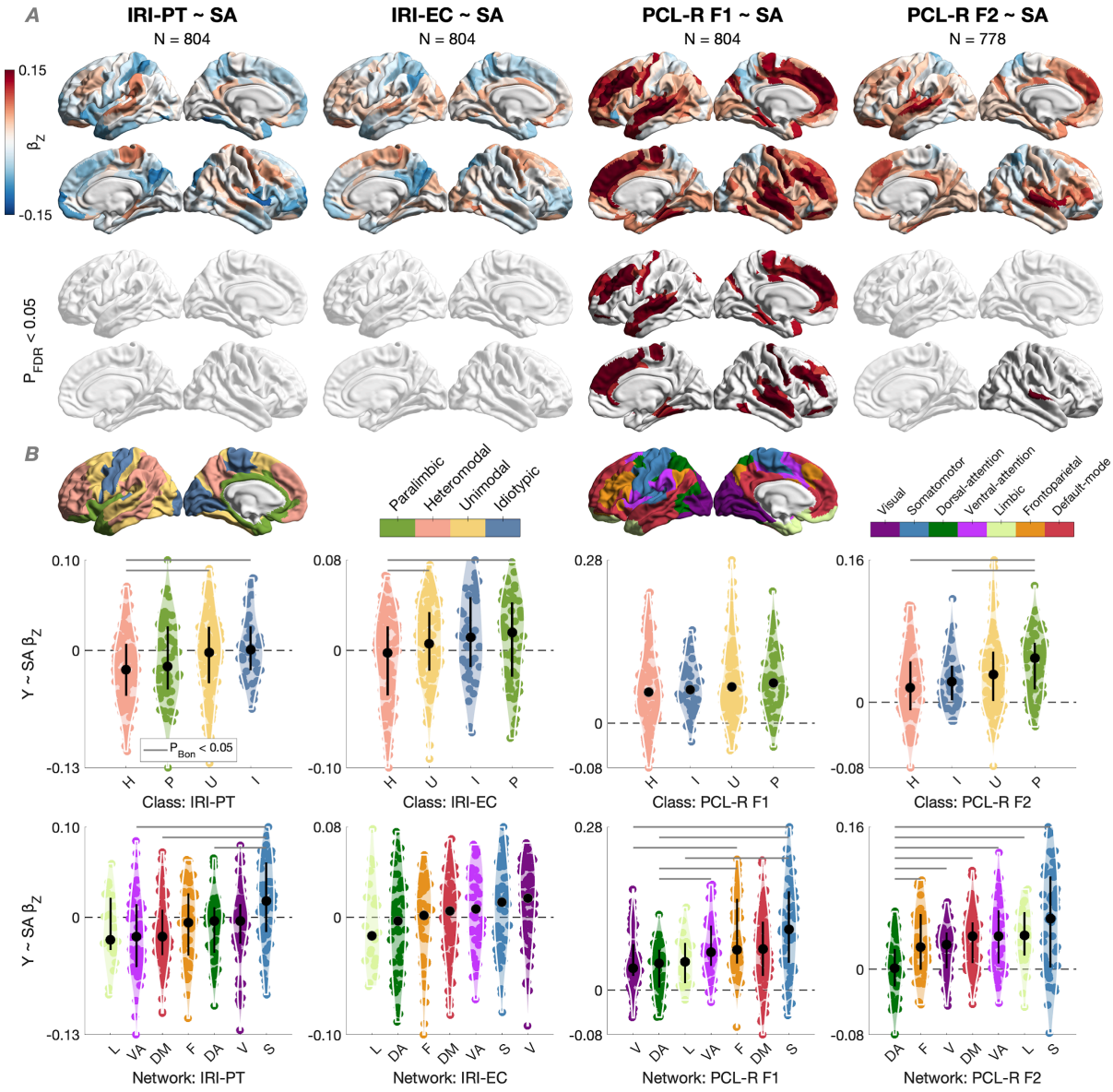


**Figure 2. SA in relation to empathy and psychopathy.** (A) Relationships of SA (positive, if any) with four continuous variables of interest, controlling for age, IQ, and TIV in a robust linear regression with FDR correction. (B) Standardized betas across the cortex by cytoarchitectonic class and functional network, median-ordered and tested for distribution differences using Wilcoxon's rank-sum test with Bonferroni correction within class (six comparisons) or network (21 comparisons).

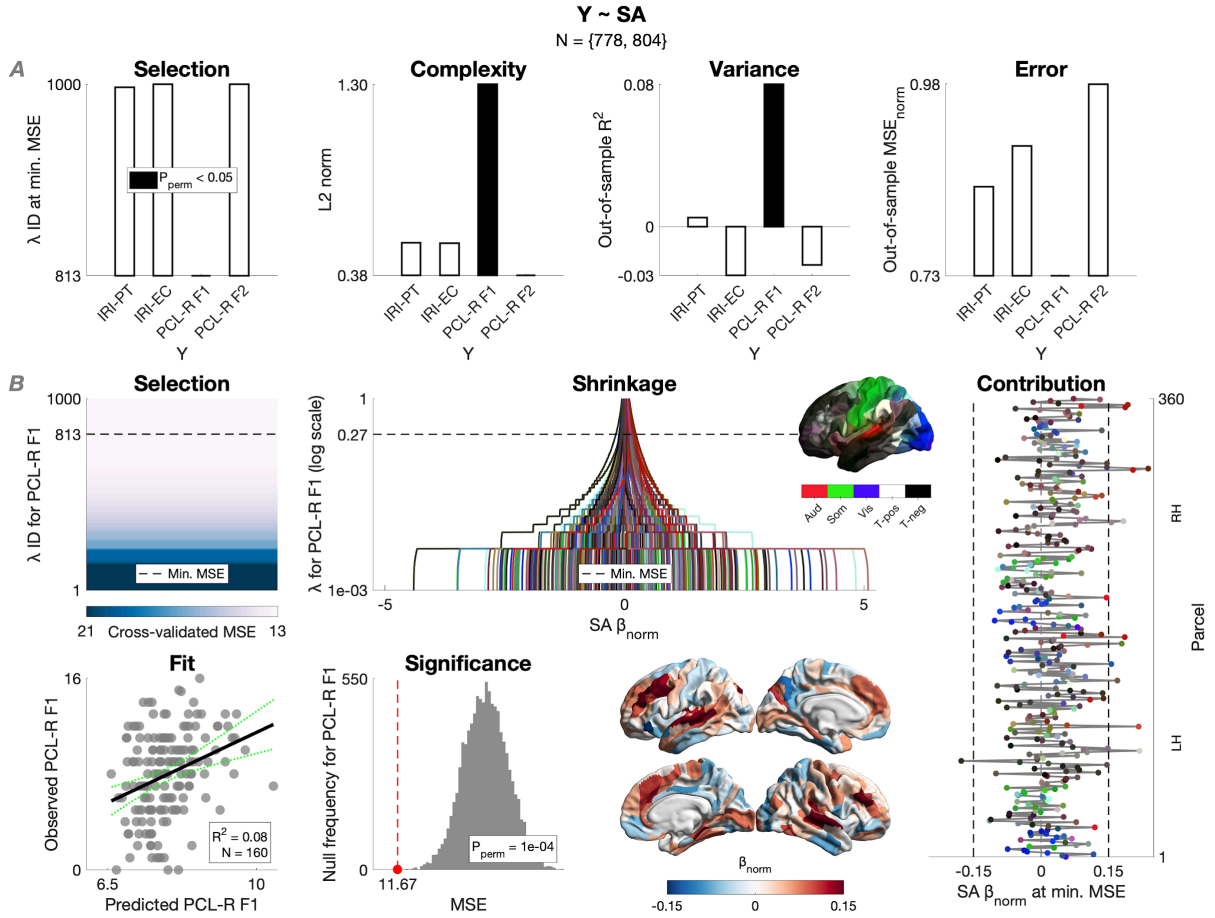


**Figure 3. Multivariate prediction of empathy and psychopathy from SA.** (A) For four continuous variables of interest, we inform on: model selection using cross-validated ridge regression (i.e., lambda corresponding to the minimum cross-validated MSE at which the model was selected); model complexity (i.e., Euclidean norm of the final beta vector); variance explained (i.e., out-of-sample coefficient of determination); and prediction error (i.e., out-of-sample MSE divided by the maximum possible score and thus normalized). SA was corrected for age, IQ, and TIV separately in the training (N = 644/623) and test (N = 160/155) sets. Among the four variables, only PCL-R F1 was able to be predicted ($R^2$ = 0.08 [95% CI: 0.02, 0.13], $P_{perm}$ = 1e-04). (B) For PCL-R F1, we inform on model selection, beta shrinkage, final beta vector, predicted-observed fit, and significance based on permutation for out-of-sample MSE ($N_{perm}$ = 10,000).

## Cortical structure by psychopathy group (Q4)

Next, we tested for global and regional differences in cortical structure by psychopathy group (Q4). Controlling for age and IQ, there was no difference in CT (*Supplementary Fig.* ***S7***). In contrast, additionally controlling for TIV, the high-psychopathy group compared to the low-psychopathy group had increased total SA; regionally, there was an increase in 65 parcels (*Fig.* ***4A*** and *Supplementary Table* ***S10***). Consistently with the dimensional analyses, effect sizes were largest in the paralimbic class and somatomotor network (positive median in both cases), with differentiation by both class (paralimbic > heteromodal) and network (e.g., somatomotor > dorsal-attention). We then characterized the cluster of FDR-corrected SA increases in the high-psychopathy group using meta-analytic task-based activations underlying cognitive and affective empathy (34) (see *Meta-analytic activations* in the Supplement). The SA increases

overlapped up to four times more with affective- than cognitive-empathy clusters, and this affective-first ranking was observed across different cluster thresholds (*Fig.* ***4B*** and *Supplementary Fig.* ***S8***). Additionally, the paralimbic class and somatomotor network – where the effect sizes were largest – both mapped better onto affective than cognitive empathy. We also explored the broader psychological relevance of these SA increases using another meta-analytic resource, Neurosynth (105). Across 24 wide-ranging terms, the overlap was highest for affective/sensory ones, such as "pain" or "auditory", and lowest for visual ones, such as "visuospatial" (*Fig.* ***4C***). Repeating the regional analysis for SA while additionally controlling for race and substance use yielded highly similar results (*Supplementary Fig.* ***S9***).

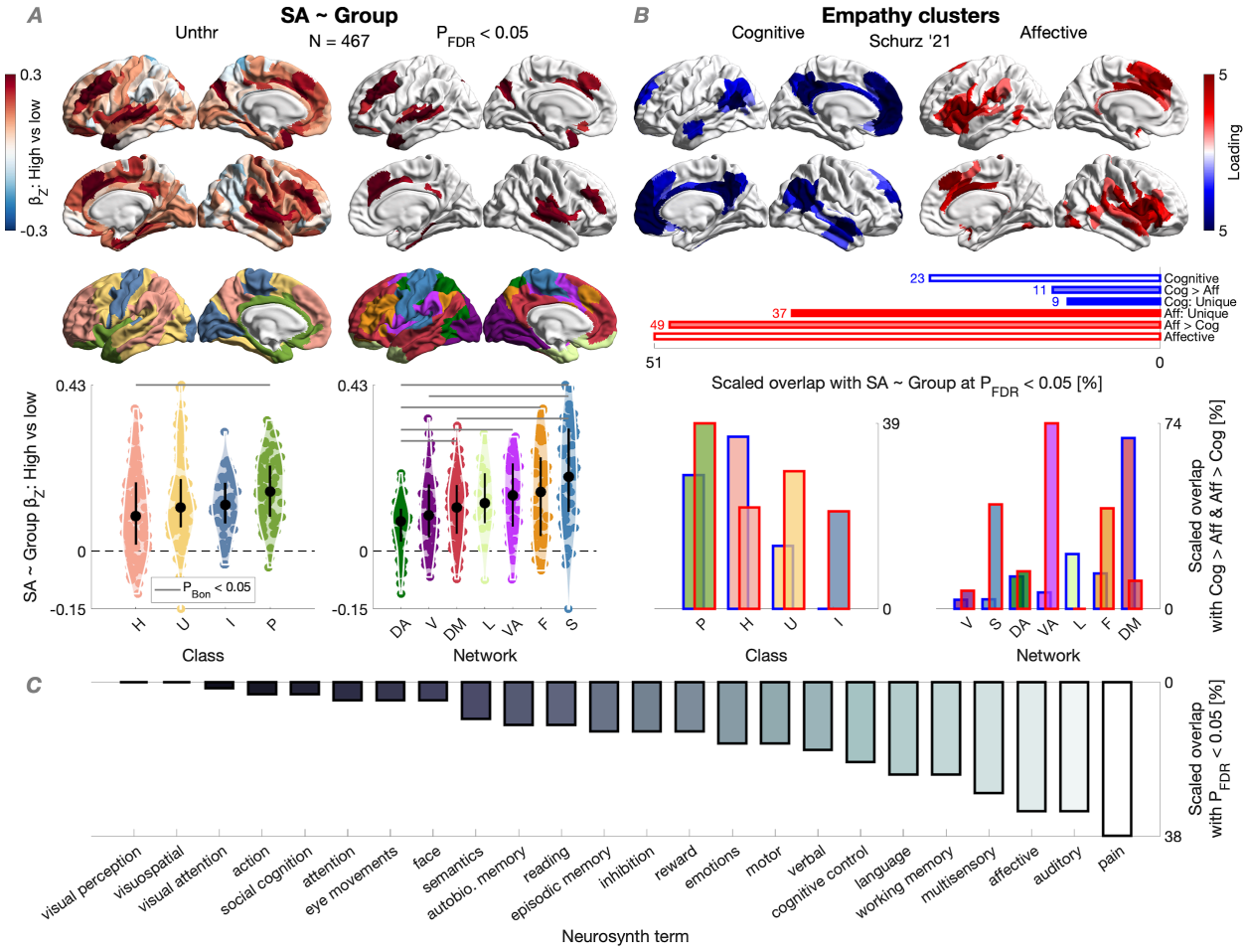


**Figure 4. SA by psychopathy group.** (A) Differences in SA by psychopathy group (high [N = 178] versus low [N = 278]), controlling for age, IQ, and TIV in a robust linear regression with FDR correction; 65 parcels showed an increase in the high-psychopathy group. Standardized betas across the cortex were median-ordered by class and network, and tested for distribution differences using Wilcoxon's rank-sum test with Bonferroni correction within class (six comparisons) or network (21 comparisons). Total SA was increased in the high-psychopathy group as well, controlling for the same covariates ($\beta_Z$ = 0.25 [95% CI: 0.14, 0.36], P = 9e-06, adj. $R^2$ = 0.67, Cohen's D = 0.39, N = 467). (B) Meta-analytic clusters of cognitive and affective empathy across more than a hundred studies (34). The FDR-corrected cluster of SA increases in the high-psychopathy group overlapped up to four times more with clusters of affective empathy compared to cognitive empathy. "Cognitive" and "Affective" = baseline, partly overlapping clusters; "Cog > Aff" and "Aff > Cog" = clusters with a

higher loading compared to their baseline counterpart; "Cog: Unique" and "Aff: Unique" = clusters of unique parcels compared to their baseline counterpart (for all clusters, see *Supplementary Fig.* ***S8***). (C) Overlap between the FDR-corrected cluster and Neurosynth (105) clusters.

**Structural covariance by psychopathy group (Q5)**

Finally, we tested structural covariance by psychopathy group (Q5). First, we examined gradients, corrected for age and IQ with CT, and additionally for TIV with SA (see *Structural gradients* in the Supplement). To probe the replicability of gradients in the total sample, we included the male sample from HCP (N = 501). Taking the non-linear approach of diffusion-map embedding to decompose the high dimensionality of cortical structure, we observed that the primary gradient of CT traversed an anterior-posterior axis, which was similar for SA. Both gradients were correlated positively with gradients in the HCP sample, thus confirming their replicability (*Fig.* ***5A*** and *Supplementary Fig.* ***S10***). We then tested for psychopathic differences in gradients aligned to those in the total sample to ensure that they were directly comparable (for raw gradients, see *Supplementary Fig.* ***S11***, with gradients in the high-psychopathy group seeming to traverse different axes). The primary gradient of CT was compressed in the high-psychopathy group compared to the low-psychopathy group, such that its distribution had a smaller range and was pulled toward the center. Such a global compression was not observed for SA (*Fig.* ***5B-D***). In sensitivity analyses for CT, compression was also observed (1) for high versus moderate psychopathy (to a lesser extent), (2) when lowering the high-psychopathy threshold, (3) when matching the high- and low-psychopathy groups for size, and (4) when using different alignment templates (*Supplementary Fig.* ***S12***). In addition, aggregating gradient loadings by class and network revealed compression-oriented differences in the limbic network for both cortical indices, and further CT differences in the paralimbic and unimodal classes as well as in the visual and dorsal-attention networks (*Fig.* ***6***). Secondly, at the edge level, there was no difference by psychopathy group either in CT or SA (*Supplementary Fig.* ***S13***).

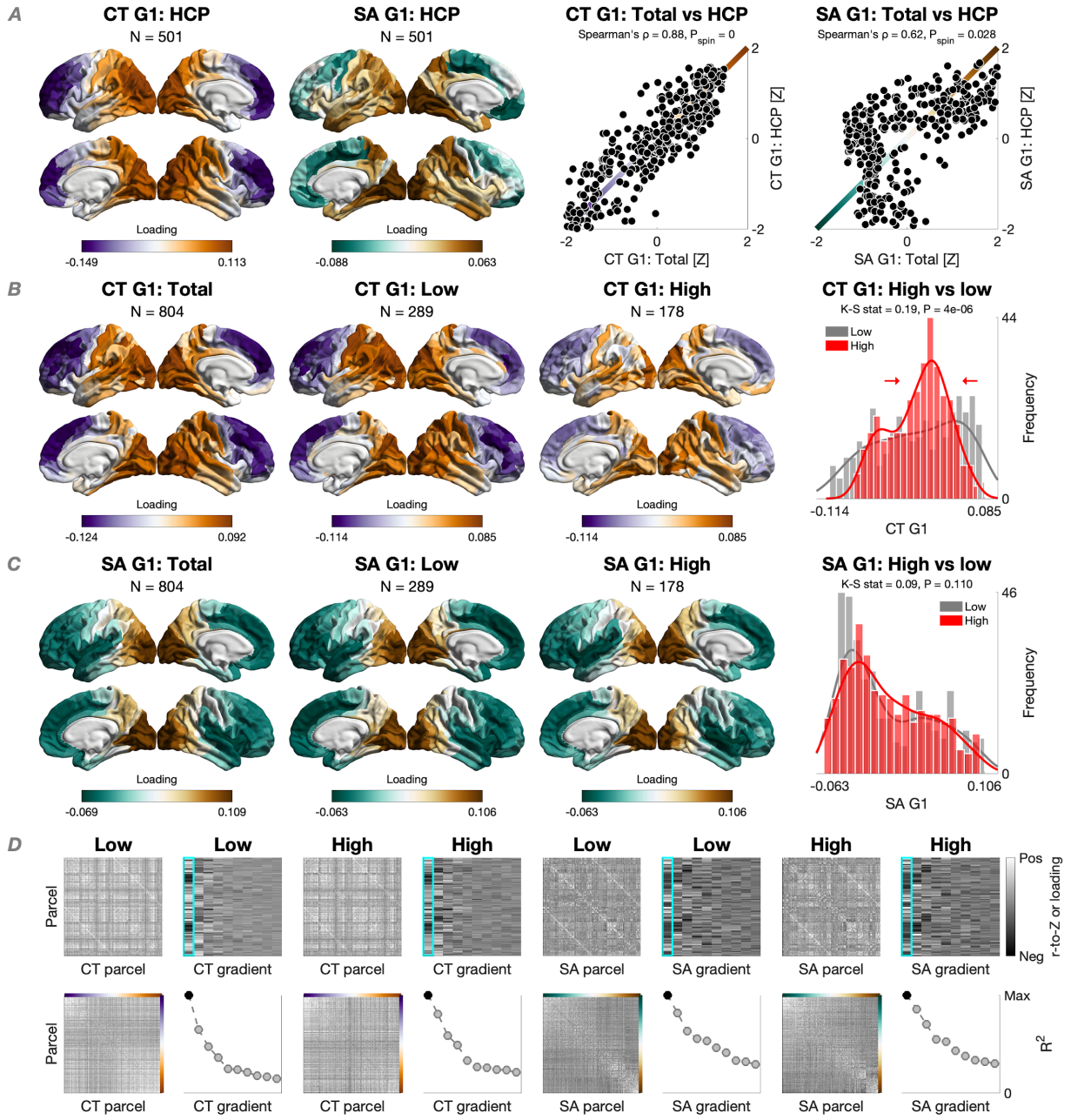


**Figure 5. Macroscale organization of CT and SA by psychopathy group: Global analysis.** (A) Primary gradients of CT and SA in the HCP sample (N = 501) and their positive correlations with gradients in the total sample (N = 804) following spin permutation ($N_{perm}$ = 1,000). In both datasets, CT was corrected for age and IQ while SA additionally for TIV. (B) Primary gradients of CT in the total sample, the low-psychopathy group, and the high-psychopathy group. The CT gradient was compressed in the high-psychopathy group compared to the low-psychopathy group using Kolmogorov-Smirnov's test. (C) Primary gradients of SA in the three samples. The SA gradient did not differ by psychopathy group using Kolmogorov-Smirnov's test. (D) Consider the two-by-two left-hand tiles: Sample-specific covariance matrix (top left), array of the first 10 gradients (top right), covariance matrix ordered by the primary gradient (bottom left), and the first 10 gradients ordered by the proportion of variance explained (i.e., scaled eigenvalues; bottom right). All matrices were set to the range [-0.5, 0.5]; all arrays were set to the minimum-maximum range.

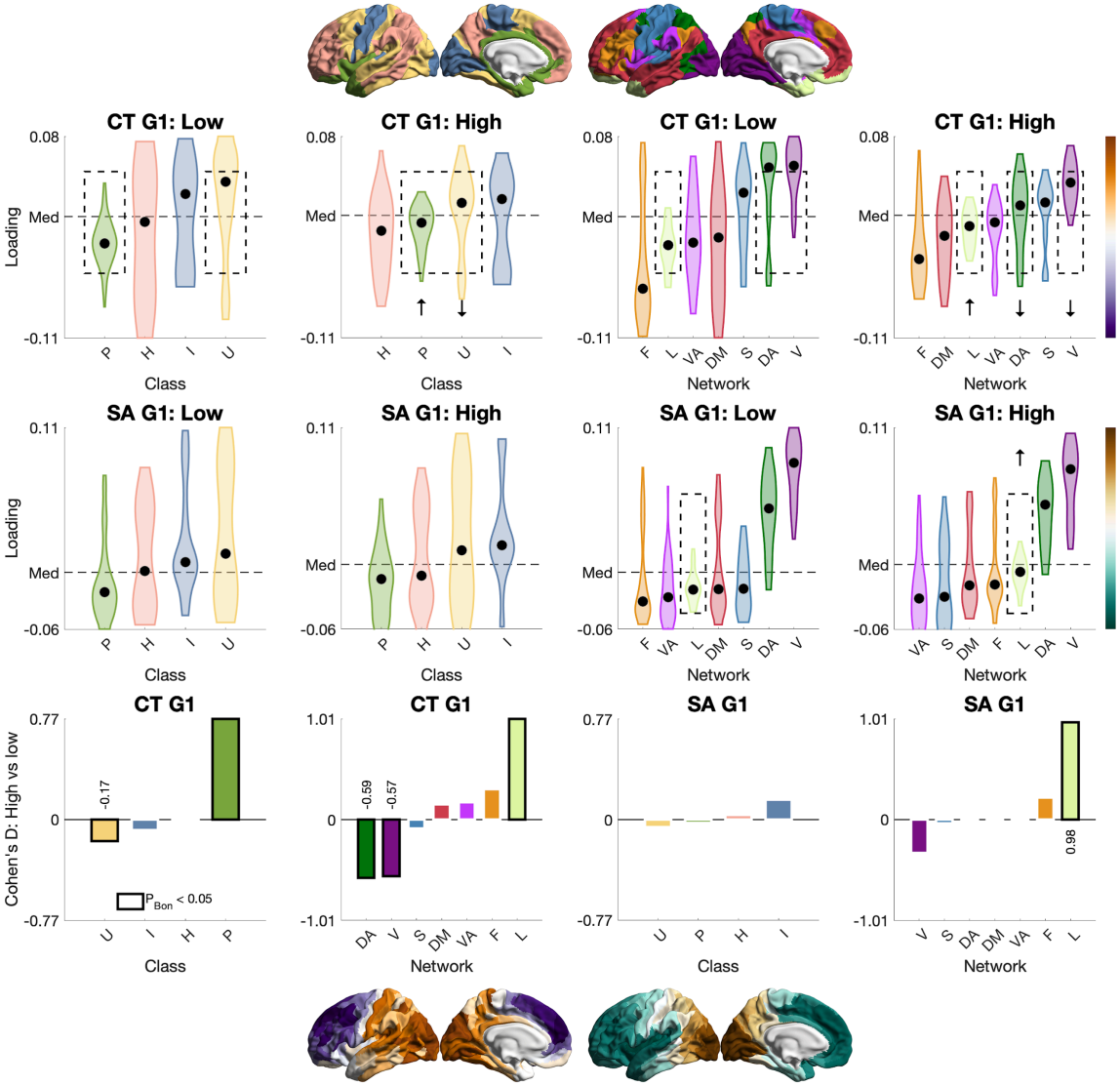


**Figure 6. Macroscale organization of CT and SA by psychopathy group: Local analysis.** Top two rows: CT and SA gradients by class and network in the low-psychopathy (N = 289) and high-psychopathy (N = 178) groups, median-ordered. For instance, both gradients in the low-psychopathy group traversed a frontoparietal-visual axis. Bottom row: Using Wilcoxon's rank-sum test with Bonferroni correction within class (four tests) or network (seven tests), the CT gradient differed in the high-psychopathy group compared to the low-psychopathy group (where the median loading was pulled toward the center) in the paralimbic and unimodal classes as well as in the visual, dorsal-attention, and limbic networks. The SA gradient recapitulated the difference in the limbic network.

## Discussion

A comprehensive mapping of CT and SA in relation to empathy and psychopathy has been lacking. We addressed this gap in 804 incarcerated adult men through five overarching questions.

As expected, psychopathy had negative relationships with empathy (Q1). PCL-R F1 had a negative relationship with IRI-EC but not IRI-PT, while PCL-R total and PCL-R F2 had negative relationships with both IRI subscales. Controlling for the other subscale revealed unique contributions of PCL-R total and PCL-R F1 to IRI-EC and of PCL-R F2 to IRI-PT. Consistently, the high-psychopathy group scored lower on both IRI subscales, but only the IRI-EC difference (which was larger) proved unique. This is in line with meta-analytic evidence that high-psychopathy individuals exhibit a larger reduction in affective empathy and/or empathic concern than cognitive empathy, whether as measured by the IRI (11) or across tasks (12) (for a similar conclusion based on self-report data, see, e.g., [106]). We further add to this literature by revealing a pattern of unique relationships, suggesting that the psychopathic reduction in cognitive empathy – which is meta-analytically replicable also based on performance data (107-109) – may, at least in part, depend on the reduction in empathic concern.

SA had positive relationships with psychopathy (Q2). This was observed for 51 out of 360 parcels for PCL-R total, 103 parcels for PCL-R F1, and three parcels for PCL-R F2. The superior-temporal/auditory cortex, playing a role in affective-speech processing (110), emerged for all three variables, with whole-cortical effect sizes being consistently largest in the paralimbic class and somatomotor network. These SA increases were in contrast to our hypothesis based on meta-analytic GMV reductions observed for psychopathy (60), knowing that GMV closely tracks SA genetically and phenotypically (111). Conversely, we did not observe any relationship between SA and the IRI subscales, which raises questions about the reliability of the latter (112) and calls for measuring empathy beyond self-report in forensic neuroimaging. However, we did observe subscale-specific differentiation by class and/or network, thus motivating future work into brain relationships with empathy in cytoarchitectonic and functional-organizational contexts. Regarding CT, we observed more circumscribed relationships with PCL-R F1 compared to SA (mostly positive) but not with any other behavioral variable tested. Together with our multivariate prediction of PCL-R F1 (but not PCL-R F2), for which SA explained more out-of-sample variance than CT did (Q3), our findings suggest that PCL-R F1 is more neurobiologically distinctive than PCL-R F2, and that this is better captured by SA than CT. Importantly, this is the first evidence of any relationship between SA and psychopathy.

Corroborating the dimensional findings, the high-psychopathy group had increased SA (Q4). These increases spanned 65 parcels, also covering the superior-temporal/auditory cortex, while whole-cortical effect sizes were likewise largest in the paralimbic class and somatomotor network. The paralimbic class was hypothesized to be especially relevant to psychopathy under the "paralimbic-dysfunction" model (62). While our findings indeed highlight paralimbic relevance, the observed SA increases do not readily align with the GMV reductions posited by the model and commonly reported (64). In our study, what further suggests the relevance of affect/sensation to psychopathy is that the SA increases overlapped up to four times more with meta-analytic clusters of affective than cognitive empathy across more than a hundred studies (34). In addition, across 24 wide-ranging meta-analyses from Neurosynth (105), the overlap was highest for affective/sensory

terms, such as “pain” or “auditory”. Beyond general sensory processing, these SA increases could underlie the hallmark psychopathic reduction in affective empathy and/or empathic concern that transcends the IRI (12). In contrast, we observed no group difference in CT, in line with predominantly null findings in the literature that typically includes N < 100 per study (62). Similarly to the GMV discrepancy, while our findings agree with a greater sensitivity of SA than CT to broadly construed antisocial behavior, they differ in direction – reductions rather than increases in SA were reported among antisocial individuals, although drawn largely (73) or exclusively (72) from community rather than incarcerated samples. It will be essential for future work to reconcile the SA increases we observed for psychopathy, dimensionally and categorically, with the SA reductions observed for antisocial behavior and the GMV reductions meta-analytically observed for psychopathy (although, again, across mixed samples and using voxel-based morphometry [60]; see also [64,68] for systematic reviews noting GMV increases for psychopathy). To enhance the developmental framing of the SA increases observed here, considered should be the cellular and physical mechanisms driving cortical expansion and folding, such as neural-progenitor proliferation, tangential neuronal migration, or mechanical stress (113-115).

Finally, the macroscale organization of CT was compressed in the high-psychopathy group (Q5). The primary gradient in the total sample traversed an anterior-posterior axis for CT (as reported across the sexes in HCP, likely mirroring the temporal sequence of neurogenesis [78]) and a similar axis for SA (as reported in the genomic literature [116]). Both axes replicated in the male HCP sample, taking the same gradient-modelling approach. When testing for psychopathic differences along these axes, we observed a globally compressed gradient of CT but not SA. At the class and/or network level, the high-psychopathy group further showed compression-oriented differences for both cortical indices, with these differences converging in the limbic network. CT gradients are known to differ across (83-85) and within (91) major psychiatric conditions, as is the primary functional gradient in terms of compression (86-88). We provide the first evidence that high-psychopathy individuals may exhibit similar macroscale properties of the cortex, suggesting reduced differentiation between anterior/transmodal regions (e.g., frontoparietal) and posterior/unimodal regions (e.g., visual) – which anchored the ends of the CT gradient in our data. It is an open question whether such reduced differentiation reflects disrupted integration and segregation from a connectomic perspective (90,117,118), and a critical next step will be to test for psychopathic differences along the unimodal-transmodal axis itself (82). This axis not only recapitulates the anterior-posterior axis of CT (78), but may be clinically compressed (86) along with the CT axis, as suggested for schizophrenia (91).

There are limitations to our study. First, a performance test of empathy could have yielded additional insights. Indeed, our use of the IRI cannot be conclusive given the questionable correspondence between self-reported and tested empathy in the general population (119), and the potential for social-desirability bias and metacognitive deficits in the incarcerated population. As above, these findings may not generalize to community (120) but also to female samples, with there being sex/gender differences in psychopathy as well as empathy (121-123) and cortical structure (124). To address our study’s broader relevance, recruiting from the general and incarcerated female populations should thus be prioritized. While this could be the largest brain-structural study on PCL-R-based psychopathy to date (for comparison, the GMV meta-analysis [60] included N = 519

in total; see also [61,62,125]), it is possible that some analyses were still underpowered due to sample size, effect size, or both (76,126,127). Finally, the roles of other cortical indices, and subcortical structures, remain to be elucidated.

In conclusion, high-psychopathy men had uniquely reduced empathic concern, increased SA, and compressed macroscale organization of CT. Future work should aim to replicate and build upon these novel findings in community and incarcerated samples to improve the treatment of psychopathic traits in the long run.

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Author contributions are as follows: Conceptualization: M.A.R., E.S., G.L., G.H., S.P., P.P., K.A.K., & L.C. Data processing: M.A.R., J.M.M, K.A.H., D.D.S, & L.C. Data analysis: M.A.R. & L.C. Data visualization: M.A.R. Writing – original draft: M.A.R. Writing – review and editing: All authors. Funding and data acquisition: J.D., S.P., & K.A.K. Supervision: P.P., K.A.K., & L.C.

Some of the generated data and code will be shared online upon publication (https://github.com/MARadecki/EmpathyPsychopathy). Regarding the source data, please contact K.A.K. Human Connectome Project data are available online pending access approval (https://www.humanconnectome.org/). The following resources are further available online: meta-analytic empathy data (https://osf.io/pav27/ [34]); Neurosynth data (https://neurosynth.org/ [105]); volumetric and surface-based template data as part of neuromaps (https://github.com/netneurolab/neuromaps [128]); code for volume-to-surface mapping as part of the Connectome Workbench (https://www.humanconnectome.org/software/connectome-workbench [129]); code for cortical parcellation as part of the ENIGMA Toolbox (https://enigma-toolbox.readthedocs.io/ [130]); code for gradient mapping as part of the BrainSpace toolbox (https://brainspace.readthedocs.io/ [131]).

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