



Mapping the structural brain network of psychopathy: Convergent evidence from humans and chimpanzees

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ABSTRACT

Psychopathy, a condition marked by profound emotional and interpersonal deficits, has long been hypothesized to arise, in part, from structural abnormalities in the brain. Yet neuroimaging studies have reported widely disparate findings, preventing definitive conclusions. Here, we sought to resolve these discrepancies by testing whether heterogeneous peak locations identified across studies converge on a common brain network. We conducted a systematic literature search of PubMed, Web of Science, EMBASE, and Scopus, identifying 18 studies (20 independent samples) examining whole-brain grey matter volume in psychopathy. Using neuroanatomical data from 1000 healthy participants, we found that grey matter volume at these peak locations varied together, forming a shared network that primarily encompassed paralimbic regions, including the cingulate and insular cortices. We next examined whether psychopathic and personality traits predicted inter-individual variation in grey matter volume within this network in humans ($n = 107$) and chimpanzees ($n = 148$). Significant effects were observed in both species, explaining 15–16% and 42–48% of the variance, respectively. Taken together, these findings indicate that psychopathy is best characterized by structural deficits in a distributed neural system rather than isolated regions, providing a unifying account of decades of inconsistent findings and advancing our understanding of its clinical, biological, and evolutionary bases.

1. Introduction

Psychopathy is a personality disorder characterized by a constellation of symptoms such as shallow affect, pathological lying, low or lack of remorse and empathy, and antisocial behaviour (De Brito et al., 2021a; Hare, 2003a). The gold-standard measure of psychopathy, the Psychopathy Checklist-Revised (PCL-R) (Hare, 2003a; Hare and Neumann, 2008), maps onto two overarching factors: Factor 1 (i.e., Interpersonal and Affective facets) and Factor 2 (i.e., Lifestyle and Antisocial

facets) (Harpur et al., 1989), which can be further broken down into a four-facet structure (Neumann et al., 2015). Using this conceptualization, approximately 1% of the general population is affected, compared to up to 25% in carceral settings (De Brito et al., 2021a). Yet, a growing body of research argues that psychopathy is better conceptualized as an aberrant constellation (Miller and Lynam, 2015b) and/or covariation (Lilienfeld et al., 2019) of basic elements of personality, rather than as a distinct psychopathology. Indeed, some evidence highlights evolutionary benefits of such psychological traits that may have been

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preserved over the course of evolution (Brazil and Forth, 2020; Glenn et al., 2011). For example, in the general population, psychopathic traits have been shown to shape leadership and political affiliation (Lilienfeld et al., 2014). Yet, despite its critical importance for elucidating its pathogenesis, the neurobiological basis of psychopathy remains poorly understood.

Early neuroimaging research suggested that individuals with elevated psychopathic traits showed significant grey matter volume (GMV) reductions in the prefrontal cortex (PFC) (Raine et al., 2000). Converging evidence from lesion studies further showed that damage to the ventromedial part of the PFC (Barrash et al., 2000; Blair and Cipolotti, 2000; Eslinger and Damasio, 1985; Grafman et al., 1996) can produce a clinical profile resembling psychopathy, coined “*pseudopsychopathy*” (Blumer and Benson, 1975) or “*acquired sociopathy*” (Eslinger and Damasio, 1985). This includes severe impairments in decision-making (Koenigs et al., 2007; Young et al., 2010), disturbed social behaviour (i.e., insensitivity, social inappropriateness, inappropriate affect, aggression, (Barrash et al., 2011), reduced prosocial motivation (Lockwood et al., 2024), and diminished autonomic responses to social stimuli (Damasio et al., 1990a). In psychopathy, most structural neuroimaging studies have been conducted on prisoners and patients from correctional and forensic settings using the PCL-R; this body of work has supported the central role of the PFC (Aoki et al., 2014; De Brito et al., 2021b; Deming and Koenigs, 2020; Johanson et al., 2019; Yang and Raine, 2009), and also highlighted the importance paralimbic areas (i.e., anterior to posterior cingulate cortices, insula, parahippocampal gyri, and anterior superior temporal gyrus) (Kiehl, 2006). For example, in a large sample of male prisoners (N = 254), all PCL-R facets were found to be uniformly and consistently associated with paralimbic structures, indicating that these neural correlates are consistently shared across facets of psychopathy rather than being attributable to a specific facet (Baskin-Sommers et al., 2016). However, considerably less is known about the neurobiology of psychopathy when operationalized through self-report measures, which are mostly used in community samples. Indeed, although substantial neuroimaging research has examined clinician-rated assessments (e.g., PCL-R) on prisoners and patients, it remains unclear whether these associations with paralimbic regions generalize to self-reported psychopathic traits in community samples. In one study conducted in a community sample, psychopathic traits measured using the Triarchic Model (TriPM, Patrick et al., 2009b) and Psychopathic Personality Inventory (PPI-R, Lilienfeld et al., 2005) showed overlapping GMV associations in clusters spanning the putamen to the posterior insula and anterior cingulate cortex, while also demonstrating distinct associations in regions such as the precentral and lingual gyri (Vieira et al., 2015). Associations with paralimbic areas (e.g., anterior cingulate, insula) have been reported using the Self-Report Psychopathy Scale (Chester et al., 2023) and the PPI (Beckwith et al., 2018), whereas associations with striatal regions (Myznikov et al., 2023) have been reported using the Dirty Dozen (Jonason and Webster, 2010b). From a broader personality perspective, GMV of paralimbic structures has been associated with inter-individual variations in agreeableness and conscientiousness (DeYoung et al., 2010; Kapogiannis et al., 2013), consistent with the Five-Factor Model accounts of psychopathy, which highlight these two domains as central features from a basic personality perspective (Miller and Lynam, 2015a, 2015b).

While evidence indicates that GMV of paralimbic regions contributes to antagonistic behaviours in humans, similar patterns have been observed in non-human primates. For instance, chimpanzees exhibit smaller GMV than bonobos in multiple regions implicated in socio-affective processing and impulse control, including the PFC, insula, and anterior superior temporal regions (Rilling et al., 2012), which may partly underlie their pronounced differences in social behaviours and propensity for aggressive behaviour (Brooker et al., 2024). In line with these findings, Latzman and colleagues (Latzman et al., 2015b) showed that agreeableness was negatively associated with grey matter volume in

PFC (i.e., dorsal and medial PFC) and the anterior cingulate cortex, whereas extraversion was positively associated with grey matter volume in the anterior cingulate cortex. Paralimbic regions are undeniably central to social behaviours across species. Yet these findings indicate that their phenotypic expression may differ between humans and chimpanzees.

Recently, concerns have been raised about the reliability and reproducibility of neurobiological findings in psychopathy (Deming et al., 2024, 2022), calling into question the extent to which the disorder has identifiable neurobiological correlates (Jalava et al., 2023). More precisely, Deming and colleagues (Deming et al., 2024) recently showed that 83.3% of experiments examining the medial PFC yielded null effects in relation to psychopathy. Given the substantial clinical and methodological differences between studies, most meta-analytic findings are expected to be driven by approximately 25% of contributing studies (Eickhoff et al., 2016). However, the predominance of null findings may make systematic reviews and meta-analyses particularly sensitive to study inclusion, potentially contributing to the limited overlap in findings reported across recent reviews (Aoki et al., 2014; De Brito et al., 2021b; Deming and Koenigs, 2020; Johanson et al., 2019; Yang and Raine, 2009). Nevertheless, these findings underscore a major challenge that must be resolved before meaningful progress can be made in the field. A wide range of possible explanations for this lack of reproducibility has been proposed, including sample size (Marek et al., 2022; Yarkoni, 2009), scan length (Ooi et al., 2025), study design (Kang et al., 2024); analytical choices (Botvinik-Nezer et al., 2020; Demidenko et al., 2024), measurement error (Gell et al., 2024), as well as heterogeneity in clinical samples, differences in psychopathy measures, and the operationalization of psychopathy (Koenigs et al., 2011; Latzman et al., 2020). For example, most structural neuroimaging studies of psychopathy have been conducted in forensic and correctional samples, where psychopathy is typically assessed using clinician-rated instruments such as the PCL-R. Because distinct operationalizations (clinician-rated instruments and self-report measures) are closely linked to the populations in which they are applied, it remains unclear whether neurobiological findings identified in forensic samples generalize to personality-based conceptualizations of psychopathic traits in community populations.

A growing body of work advocates for viewing the brain as a complex system organized into functional modules of highly interconnected brain regions, which align with our long-lasting knowledge about the distributed organization of the brain (Bassett and Sporns, 2017; Noble et al., 2024; Segal et al., 2025; Westlin et al., 2023). Indeed, psychiatric neuroimaging research has traditionally examined brain regions as isolated units to identify structures implicated in disorders such as psychopathy. Yet this approach overlooks the interconnected nature of brain organization (Segal et al., 2025). From such a localizationist perspective, it is difficult to explain how damages to distinct areas in the brain (i.e., amygdala or OFC) may contribute to a similar clinical profile (Blumer and Benson, 1975; Eslinger and Damasio, 1985; Mitchell et al., 2006). This shift in paradigm allows for new hypotheses of psychiatric conditions, such as *neurobiological equifinality* (Dugré and De Brito, 2025), where regional deficits in heterogeneous locations may give rise to the same phenotype. In line with this network-based approach, Darby and colleagues (Darby et al., 2018) demonstrated that heterogeneous brain lesions known to occur prior to the onset of antisocial behaviours were functionally connected to a common neural network. Recently, Dugré and De Brito (Dugré and De Brito, 2025) extended lesion network mapping (Fox, 2018) to a coordinate-based meta-analysis of 40 fMRI samples on psychopathy, demonstrating high replicability across studies and a highly concordant network with that identified across brain lesions by Darby and colleagues (Darby et al., 2018). Identifying robust and replicable structural imaging findings in psychopathy research has yet to be achieved. To our knowledge, the application of this method to structural imaging remains largely limited and could provide a novel way to identify a common and replicable structural covariance network underpinning psychopathy. Indeed, brain

regions with similar morphological properties tend to show coordinated patterns of variation across the population (Mechelli et al., 2005). This structural covariance has, in turn, been shown to reflect underlying functional connectivity between brain regions (Alexander-Bloch et al., 2013). However, structural covariance reveals unique relationships between regions that are not fully captured by functional connectivity, suggesting a more complex biological basis that may reflect shared aetiology and/or neural plasticity (Alexander-Bloch et al., 2013; Andrews et al., 1997; Draganski et al., 2004; Romero-Garcia et al., 2018). While some studies have previously used functional connectivity to map GMV atrophy (Segal et al., 2023), retaining morphometric information may offer greater biological precision and explanation of psychiatric disorders.

In the current study, we first aimed to conduct an updated meta-analysis of voxel-based morphometry studies on psychopathy, including 20 independent samples, to address the limited statistical power of previous meta-analyses (De Brito et al., 2021b, $k = 7$ samples) & (Deming and Koenigs, 2020, $k = 9$ samples). Secondly, we aimed to extend our previous network mapping approach (Dugré and De Brito, 2025) to structural neuroimaging by specifically testing whether heterogeneous locations of GMV deficits reported across studies are structurally connected to a common morphometric network. More precisely, traditional meta-analytic methods primarily aim to identify regions of consistent volumetric deficits across studies. In contrast, our current approach, based on structural covariance, rather aims to identify inter-regional covariation network among regions showing volumetric deficits, thereby providing a more comprehensive biological account of the neurobiology of psychopathy. To this end, we used normative voxel-based volumetric data from a large sample of healthy individuals ($n = 1000$) to calculate the correlations between the average volume within the reported regions and volumes at each voxel across the rest of the brain. Similarly to Dugré et al. (2025), we first hypothesized that meta-analytic findings generated from a traditional approach would be driven by a few studies ($< 25\%$). In turn, we hypothesized that our method would allow us to identify a common structural covariance network across studies on psychopathy, with replicability of some regions greater than 60% across studies, especially in paralimbic regions. To test the specificity of this network, we examined whether it showed preferential involvement in patients with focal brain lesions who subsequently developed antisocial behaviours, relative to control lesions. Using external data from humans and chimpanzees, we tested whether personality-based conceptualizations of psychopathic traits predicted variations within this network. Here, we emphasize that these analyses are not intended to validate the full psychopathy construct, but rather to characterize the nomological network associated with this psychopathy-related brain network. As Lilienfeld (Lilienfeld, 2022, p.486) noted, “Most or all of these models may capture a kernel of the truth, but none has emerged as a comprehensive model of psychopathy.”

2. Methods

2.1. Activation likelihood estimation meta-analysis

A literature search was performed by reviewing studies included from two meta-analyses (De Brito et al., 2021b; Deming and Koenigs, 2020) and two systematic reviews (Johanson et al., 2019; Klaus et al., 2024). In addition, a systematic search strategy was conducted, using four search engines (PubMed, Web of Science, EMBASE, and Scopus) from February 2020 to October 31st 2024 to identify relevant studies, extending previous search. The following search terms were used: *psychopathy* OR *psychopathic* AND *neuroimaging* OR *MRI*. JRD screened all titles, abstracts, and full-text studies. Uncertain cases were resolved through discussion between JRD and SADB. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA, Liberati et al., 2009; Table S1) guidelines were followed. Please refer to

Supplementary Method for inclusion criteria.

Our meta-analysis of voxel-based morphometry samples, which extend previous work (De Brito et al., 2021b; Deming and Koenigs, 2020), was conducted using JALE (<https://github.com/juaml/JALE>), a python package for conducting activation likelihood estimation (ALE) meta-analyses (Eickhoff et al., 2012, 2016), Supplementary Method, <http://www.brainmap.org/ale/>). Significance of spatial convergence was established by using a threshold of $p < 0.001$ at voxel-level and $FWE-p < 0.05$ at a cluster-level with 5000 permutations, as recommended (Eickhoff et al., 2012, 2009).

2.2. Normative network mapping

A network mapping approach was conducted to identify whether heterogeneous peak coordinates reported across voxel-based morphometry samples of GMV alterations in psychopathy may be linked to a common network of structural co-variance in the healthy brain. Structural covariance analyses assess the extent to which different brain regions vary together across individuals based on anatomical properties (e.g., volume), potentially reflecting shared aetiology (e.g., genetic factors) and/or neural plasticity (Alexander-Bloch et al., 2013; Andrews et al., 1997; Draganski et al., 2004; Romero-Garcia et al., 2018). In other words, in this cross-sectional framework, if individuals with larger values in one region (e.g., mPFC) also tend to show relatively larger values in another region (e.g., amygdala), the two regions are said to structurally covary. Importantly, this approach does not directly measure physical anatomical connections, nor does it estimate latent factors in the same sense as factor analysis. Rather, it evaluates statistical relationships among observed anatomical measures across individuals.

To do so, a 4-mm sphere was first created around each coordinate reported by neuroimaging studies on psychopathy, resulting in a sample-level binary mask. Then, using morphometric data from 1000 healthy subjects (ages 18–35 years old, 50% females) from the Brain Genomics Superstruct Project (Please refer to Supplementary Methods for MRI data acquisition and processing) (Holmes et al., 2015; Yeo et al., 2011), we calculated the structural covariance profile of each sample-level mask. This allowed us to identify which voxels covaried with regions showing GMV associations with psychopathy in a given study. Specifically, we extracted the mean volume of each sphere of the sample-level mask in each of the 1000 healthy individuals and correlated them to the volume of other voxels in the brain. A partial correlation controlling for total intracranial volume was then conducted to generate a sample-level structural covariance map for each study.

To assess whether heterogeneous peaks converged on a common network, we conducted two complimentary analyses: 1) the statistical significance of the network strength across samples; 2) the degree of overlap across samples. For the first approach, sample-level maps were converted to Fisher's z scores and combined in a voxel-wise one-sample permutation test to identify regions showing greater consistency across studies than expected by chance (Winkler et al., 2014). Statistical significance was assessed using cluster-based Threshold-Free Cluster Enhancement (TFCE) with family-wise error correction (FWE) based on 5000 permutations. The second approach aimed to quantify the degree of overlap across studies. To do so, sample-level t -maps were thresholded at $T > 5$, binarized, and summed to quantify the number of studies showing significant effects at each voxel. This threshold was selected based on previous studies using a similar approach (Dugré and De Brito, 2025; Dugré and Potvin, 2024; Stubbs et al., 2023) and because it provides a fixed threshold that is not subject to sample-specific variation, as can occur with false-discovery rate-based thresholds.

In an exploratory analysis, we further examined the specificity of the identified network by testing whether it was preferentially associated with brain lesions preceding the onset of antisocial behaviours, providing a potential test of its causal relevance. Specifically, we compared the spatial similarity of lesion-derived networks from 17 cases

identified retrospectively involving individuals with focal brain injuries that preceded the onset of antisocial behaviors with that of 17 matched control lesions (Supplementary Fig. 1). A two-sample permutation test was used to test whether whether lesion-derived networks from cases showed greater spatial similarity to our structural covariance network than those from matched control lesions (5000 permutations). We additionally explored whether these associations varied across specific antisocial personality disorder symptoms (see Supplementary Methods).

2.3. Contribution of large-scale connectivity networks

Given the known modular organization of the brain, which is also captured by structural covariance (Alexander-Bloch et al., 2013; Romero-García et al., 2018), we examined whether the structural brain network underpinning psychopathy was driven by regions belonging to a particular large-scale canonical network, from the Schaefer-400 7 Networks (Schaefer et al., 2018). Network contributions were estimated as the difference between each network's observed mean and the mean network value across 5000 null maps generated using spatial autocorrelation-preserving permutation tests (spin tests) (Markello and Misic, 2021). Specifically, each null map was generated by projecting parcels onto a sphere, applying random rotations, and reassigning values to preserve the spatial autocorrelation structure of the data. Finally, the false discovery rate was controlled at $p < 0.05$.

2.4. External validation

We then examined whether the identified structural covariance network truly reflected the construct of psychopathy by testing its association with the severity of psychopathic traits, providing an assessment of its construct validity (Cronbach and Meehl, 1955). Because the structural covariance network was identified predominantly from studies using the PCL-R, it remains largely unknown whether its expression generalized to self-report measures of psychopathy in community samples. Despite conceptual and measurement differences, dimensional conceptualizations of psychopathy consistently demonstrate moderate-to-strong associations with PCL-R scores (Lynam et al., 2011; Miller and Lynam, 2012; Sleep et al., 2019) and show substantial conceptual overlap across psychopathy frameworks, as evidenced by high similarity between items from the Self-Report Psychopathy Scale and those from dimensional conceptualizations such as the Triarchic Model of Psychopathy (cosine similarity = 0.84) (Burghart et al., 2026). Moreover, recent evidence suggests that chimpanzees exhibit a similar personality-based organization of psychopathic traits (Latzman et al., 2016) providing a unique opportunity to investigate the evolutionary basis of their neural correlates (e.g., paralimbic regions). We therefore hypothesized that these measures of psychopathic traits would reliably correlate with inter-individual variations in our identified network.

2.5. Cross-species evidence from humans and chimpanzees

Neuroanatomical data from both humans and chimpanzees were used to test whether psychopathic traits were indeed associated with inter-individual variability in the identified psychopathy network. First, human T1-weighted MRI scans from 227 healthy individuals (82 females, age range 20–77 years old) were included publicly available Mind-Brain-Body dataset from the Max Planck Institute for Human and Cognitive and Brain Sciences (Babayan et al., 2019) (Please refer to Supplementary Methods for MRI data acquisition).

To test whether our structural covariance network was associated with severity of psychopathic traits across species, we computed a global brain score reflecting each individual's alignment with the identified network. The structural covariance network was parcellated into 17 data-driven parcels derived from orthogonal projective non-negative matrix factorization (Sotiras et al., 2015), which have been shown to be homologous between humans and chimpanzees (Vickery et al.,

2024). Mean t -values were extracted from each parcel and combined with each participant's regional GMV values using a dot product. This yielded a global psychopathy brain score reflecting the degree to which each participant's brain morphology aligned with the identified structural covariance network. This approach accommodates individual differences in brain-wide GMV patterns, consistent with neurobiological equifinality, whereby different neurobiological configurations may give rise to similar behavioural outcomes.

For external validation across species, psychopathic traits were assessed using the Triarchic Psychopathy Model (TriPM), which captures boldness, meanness, and disinhibition (Patrick et al., 2009b). In chimpanzees, the homologous Chimpanzee Triarchic (CHMP-Tri) scale was used as a validated measure of these dimensions (Latzman et al., 2016). Humans familiar with the chimpanzees (i.e., staff, technicians) completed a 41-item questionnaire (Freeman et al., 2013), with each item rated on a 7-point Likert scale (1 = least to 7 = most), and averaged across raters to derive a single average rank score per item. Factor analysis with varimax rotation revealed five personality dimensions including impulsivity, agreeableness, neuroticism, extraversion, and intelligence (Latzman et al., 2015a).

In humans, composite scores approximating the TriPM scales were computed from the NEO-Five Factor Inventory facets (Costa and McCrae, 1992), given the absence of a direct TriPM measure. Scores were calculated by summing the products of meta-analytically derived effect sizes linking NEO-FFI facets to the TriPM dimensions (Sleep et al., 2019) and each participant's raw facet scores. NEO-FFI was available for 108 human participants.

Multiple linear regression and dominance analyses were subsequently performed to assess the contribution of TriPM subscales to individual differences in psychopathic brain scores, after accounting for total intracranial volume. Specifically, for the chimpanzee data, linear mixed-effects models were fitted with site included as a random intercept. Standardized dominance R^2 values were reported to measure the relative contribution of each TriPM dimension to the total explained variance. A final dataset of 148 chimpanzees and 107 humans was used for further analysis after removing one chimpanzee and one human outlier ($Z > 3$). To ensure that the association between psychopathic traits and brain scores in humans a) was not driven by the choice of psychopathy measure; b) and specific to psychopathic traits, analyses were repeated using the Short Dark Triad which assess psychopathy, but also narcissism, and machiavellianism (Jones and Paulhus, 2014).

Secondary analyses were conducted to evaluate the contribution of basic personality traits in both humans (Costa and McCrae, 1992) and chimpanzees (Freeman et al., 2013; Latzman et al., 2015a). Specifically, linear models and dominance analyses were used to assess the association between broad personality factors and our structural covariance network. To evaluate the relative contribution of individual personality facets, we applied the Boruta algorithm, a random forest-based feature selection approach optimized for continuous outcomes (O'Connell et al., 2025). Across 1000 runs, the algorithm compared the importance of each personality facet with that of randomly permuted variables, providing a robust estimate of the relative contribution of each facet to the structural covariance network in both species. For humans, we generated an empirical facet profile based on both zero-order associations (Spearman correlations) and multivariate predictive weights (random forest feature importance). We then assessed the similarity of this brain-derived facet profile to established empirical and expert consensus profiles of psychopathy, antisocial personality disorder, and aggressive and antisocial behaviours (Decuyper et al., 2009; Lynam and Miller, 2015; Lynam and Widiger, 2001; Miller and Lynam, 2008; Miller et al., 2001; O'Boyle et al., 2015; Ruiz et al., 2008; Samuel and Widiger, 2008; Vize et al., 2018). To establish discriminant validity, prototypical DSM-IV-TR personality disorder profiles (Miller and Lynam, 2008; Samuel and Widiger, 2008) were included as baselines. We hypothesized that our structural covariance network would selectively align with Cluster B pathology (e.g., antisocial, borderline) while diverging

from Cluster A (e.g., schizoid) and Cluster C (e.g., dependent) profiles.

3. Results

3.1. Included studies

A total of 18 published sMRI studies were retrieved for the meta-analysis (see Fig. 1 for the flowchart, see Supplementary Tables 1–2 for PRISMA Checklist), from which 20 independent samples were included for the main analysis on psychopathy (see Supplementary Tables 3–4 for study-level comparisons with previous meta-analyses). From these 18 studies, 12 reported recruiting participants through correctional/probation settings, 1 through forensic settings, and 5 through community. The main analysis included pooled data from case-control analyses from 12 samples (296 cases versus 325 controls) and whole-brain relationship with severity of psychopathic traits (total score) among 10 samples (675 participants). Two samples included both analytic approaches from which coordinates were pooled for the main analysis resulting in 20 independent samples (Table 1). Mean age across the 20 samples was 31.8 years ($SD=5.34$, range 21.1–39.8). Fourteen clinically ascertained samples assessed psychopathy using the PCL-R or one of its variants (i.e., PCL-SV), whereas six community samples used self-report measures of psychopathic traits. Following our recent work (Dugré and De Brito, 2025), preliminary subanalyses were conducted on psychopathy subdimension in which six samples were included for both

F1 (6 samples) and F2 (6 samples).

3.2. Activation likelihood estimation meta-analysis

A coordinate-based meta-analysis was conducted with the ALE algorithm using peak coordinates of the 20 samples (280 foci). After correction for multiple comparison ($cFWE<0.05$), a small effect was found in the inferior temporal gyrus ($x = -60$, $y = -20$, $z = -26$, 89 voxels) (see Fig. 2A). More thorough investigation, in which we modeled each peak coordinates with a 4 mm sphere, revealed that the spatial overlap across foci of the 20 samples was very poor (i.e., maximum overlap = 15%; 3 studies) (Fig. 2C). Results remained unchanged after removing the six community samples that assessed psychopathy via self-report measures. Subanalyses revealed no significant effect when investigating case-control (12 samples, 205 foci) and correlational studies (10 samples, 75 foci), separately. Preliminary analyses on psychopathy subdimensions revealed neither significant effect for F1 (6 samples, 35 foci) and F2 (6 samples, 60 foci).

3.3. Normative network mapping

By modelling structural covariance networks of heterogeneous locations, we found significant convergence in many regions including prefrontal, temporal and subcortical structures, after correction for multiple comparisons (TFCE FWE $p < 0.05$). (Fig. 2B). This structural

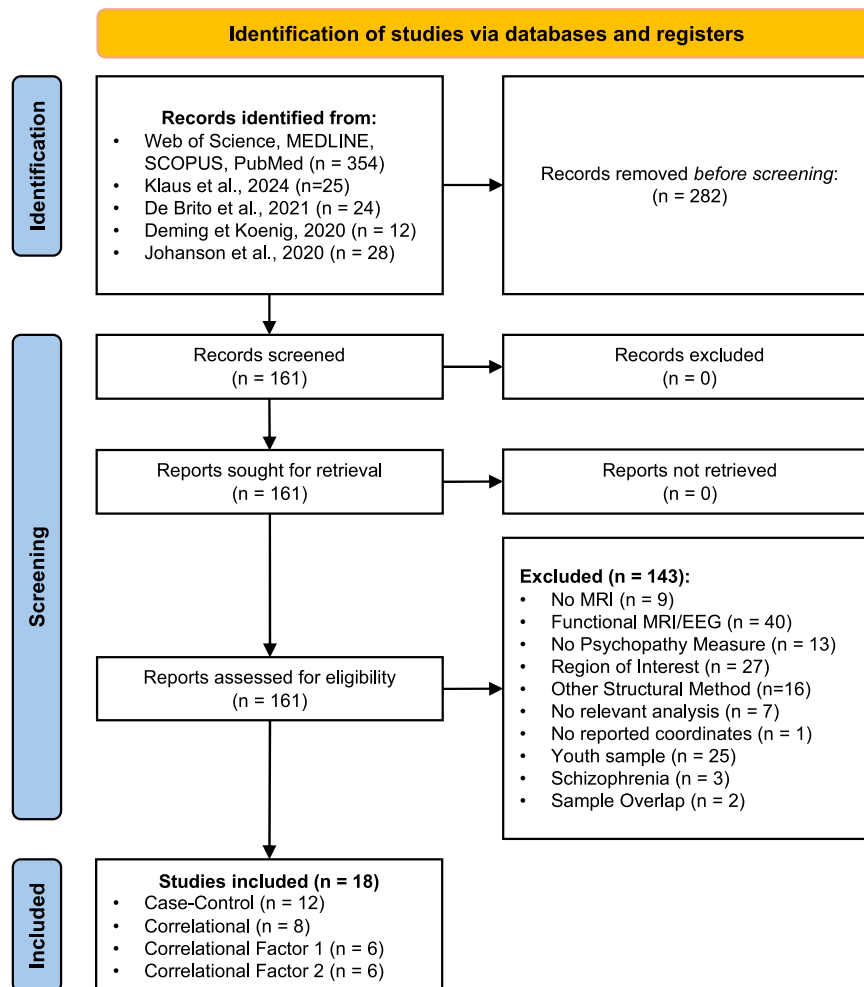


Fig. 1. Flowchart of the updated literature search for voxel-based morphometry studies of grey-matter volume on psychopathy after February 2020. Records removed before screening (n = 282) included: duplicates (n = 195), editorial, reply letter, book chapter & case reports (n = 27), literature reviews and meta-analyses (n = 46), and unrelated/irrelevant studies (n = 14).

Table 1

Characteristics of the included studies (k = 20 samples).

First Author, Year	Sample Characteristics				Psychopathy Assessment		Statistical Threshold (Voxel, Cluster Level) ^A
	N Cases	N Controls	Mean Age (SD)	Settings	Measures	Total Score	
(Beckwith et al., 2018)(A)	65	-	26.9 (1.1)	Community	PPI	378.5	p < 0.005, Cluster Corr
(Beckwith et al., 2018)(B)	90	-	26.7 (1.1)	Community	PPI	355.9	p < 0.005, Cluster Corr
(Bertsch et al., 2013)	12	14	27.3 (5.4)	Correctional	PCL-R	23.8	p < 0.001, 10 voxels*
(Chester et al., 2023) (A)	53	-	27.0 (5.1)	Community	SRP-4	50.0	p < 0.001, 10 voxels*
(Chester et al., 2023) (B)	45	-	27.0 (5.7)	Community	SRP-4	54.0	p < 0.001, 10 voxels*
(Contreras-Rodríguez et al., 2015)	22	22	39.8 (9.2)	Correctional	PCL-R	27.8	p < 0.005, 125 voxels
(Cope et al., 2012)	66	-	36.9 (7.9)	Correctional	PCL-R	18.4	p < 0.005, 5 voxels
(de Oliveira-Souza et al., 2008)	15	15	32.0 (14.0)	Psychiatric Outpatients	PCL-SV	17.8	p < 0.005, 5 voxels
(Ermer et al., 2012)	254	-	33.9 (9.5)	Correctional	PCL-R	21.3	p < .05, Cluster Corr
(Gregory et al., 2012)	17	22	38.9 (9.4)	Correctional	PCL-R	28.1	p < 0.001, 10 voxels*
(Hofhansel et al., 2020)	27	-	35.6 (9.5)	Correctional	PCL-R	16.2	p < 0.001, Cluster Corr
(Korponay et al., 2017)	41	35	31.5 (7.7)	Correctional	PCL-R	32.1	p < 0.001, Cluster Corr
(Leutgeb et al., 2015)	40	37	38.1 (12.0)	Correctional	PCL-R	20.6	p < 0.001, 10 voxels*
(Müller et al., 2008)	17	17	33.0 (5.8)	Forensic	PCL-R	33.3	p < 0.001, Not Reported
(Mysznikov et al., 2023)	57	72	24.9 (4.3)	Community	DD-DT	39.8	p < 0.001 Cluster Corr
(Noppari et al., 2022)	19	19	31.0 (7.0)	Correctional	PCL-R	26.0	p < 0.001 Cluster Corr
(Pera-Guardiola et al., 2016)	19	20	39.2 (8.9)	Correctional	PCL-R	28.1	p < 0.001, 10 voxels*
(Schiffer et al., 2011)†	11	27	34.55 (6.85)	Correctional	PCL-SV	13.6	p < 0.001, 10 voxels*
(Tiihonen et al., 2008)	26	25	32.1 (8.5)	Correctional	PCL-R	29.9	p < 0.005, Cluster Corr
(Vieira et al., 2015)	35	-	21.1 (1.8)	Community	TriPM	71.3	p < 0.005, ≈ 144 voxels

Note. (A, B) refers to independent samples A, B within the same study. ^A = to minimize heterogeneity in statistical thresholding used between studies, threshold was set at p < 0.001 with 10 voxel extent (given a 2mm3 voxel), when data were available. * = raw statistical images were shared and re-thresholded given their respective voxel size. †Data was re-analyzed by conducting a median split on PCL-SV and comparing the above-median group against controls. PCL-SV = Psychopathy Checklist – Short Version (Hart et al., 1995); PCL-R = Psychopathy Checklist – Revised (Hare, 2003b); PPI = Psychopathic Personality Inventory (Lilienfeld and Andrews, 1996); DD-DT = Dirty Dozen / Dark Triad (Jonason and Webster, 2010a); TriPM = Triarchic Psychopathy Model (Patrick et al., 2009a); SRP-4 = Self-Report Psychopathy, Fourth Edition (Roy and Neumann, 2022).

network consisted of many key connectivity hubs showing equal or greater replicability than 60%, such as bilateral mid-Insula (70% in both hemispheres), bilateral anterior insula extending to lateral OFC/ventrolateral PFC (60% both hemispheres), posterior midcingulate cortex (60%) (Fig. 3, see Supplementary Table 5 for peak coordinates of regions showing $\geq 60\%$ replicability). Spatial similarity analyses revealed highly similar neural correlates of psychopathy across clinically ascertained samples assessed with clinician-rated measures (e.g., PCL-R, PCL-SV) and community samples assessed with self-report measures (spatial similarity $r = 0.72$, $p < 0.001$). Yet, the most consistently replicated regions differed between the two sample types/assessment methods. Across the clinically ascertained samples, the bilateral insula (11/14 samples) and medial orbitofrontal cortex (9/14 samples) were the most frequently identified regions. In contrast, community samples assessed with self-report measures most consistently identified the midcingulate cortex (4/6 samples) and left amygdala (4/6 samples).

Additional specificity analyses with 17 retrospective lesion cases showed greater involvement in patients with focal brain lesions who subsequently developed antisocial behaviours, particularly among those presenting with affective deficits and irresponsibility (see Supplementary Results, Supplementary Fig 2.).

3.4. Contribution of large-scale connectivity networks

We investigated whether the identified structural brain network may be primarily explained by specific large-scale connectivity network (Schaefer et al., 2018). Network contributions were estimated as the difference between each network's observed mean and the mean network value across 5000 null maps generated using spatial autocorrelation-preserving permutation tests (Markello and Misic, 2021). Spin tests revealed that the Ventral Attention ($\text{diff}_{\text{obs-null}} = 1.73$, $P_{\text{spin}} < 0.001$) and Default-Mode ($\text{diff}_{\text{obs-null}} = 0.84$, $P_{\text{spin}} = 0.003$) networks showed the strongest contributions to the structural brain organization associated with psychopathy (Fig. 3.). Furthermore, we found similar contribution of large-scale networks irrespective of study design (i.e., case-control or correlational), or psychopathy factor (ie., Factor 1 or

Factor 2) (Supplementary Fig. 3).

3.5. Cross-species evidence from humans and chimpanzees

To examine how psychopathic traits relate to individual differences in the identified structural network, we first calculated a participant-specific brain score as a weighted sum of GMV values, with the structural network t -values serving as weights. then removed the residual effect of total intracranial volume (Fig. 4A).

In humans, the Five-Factor composite scores of the TriPM dimensions explained a total of 16.4% of the unique variance of the psychopathic brain scores ($r = 0.41$, $p < 0.001$, Fig. 4B). Specifically, this effect was predominantly driven by Disinhibition (standardized dominance $R^2 = 62.8\%$, $b = .04$, $p = .001$) and Boldness (standardized dominance $R^2 = 14.4\%$, $b = .016$, $p = 0.022$) (Fig. 4B). This was consistent with the greater relative contribution of Conscientiousness, and more precisely deliberation, self-discipline, straightforwardness, and compliance, to the unique variance in psychopathic brain scores when examining basic FFM personality dimensions (Supplementary Results, Supplementary Fig 4., Supplementary Table 8). Facet profile analyses showed greater correspondence with antisocial personality disorder and psychopathy ($r > 0.69$), and weaker correspondence with dependent and schizoid personality disorders ($r < 0.4$) (Supplementary Table 9). Exploratory analyses from the Short Dark Triad also revealed a significant relationship with psychopathic brain scores, albeit to a lesser extent, with the majority of variance accounted for by the psychopathy subscale (standardized dominance $R^2 = 60.7\%$, $b = .216$, $p = .018$) (Supplementary Results, Supplementary Fig 5).

In chimpanzees, the CHMPTri subscales explained together a total of 41.6% of the unique variance of the GMV risk scores ($r = 0.65$, $p < 0.001$, Fig. 4B), after accounting for facility. Specifically, Meanness (standardized dominance $R^2 = 57.6\%$, $b = -.30$, $p < 0.001$) and Disinhibition (standardized dominance $R^2 = 36.8\%$, $b = .29$, $p = .002$) were both significantly associated with the unique variance of the GMV risk scores (Fig. 4B). This was consistent with the greater relative contribution of Extraversion and Dominance, and more precisely active, dominant, relaxed, mischievous, timid, to the unique variance in

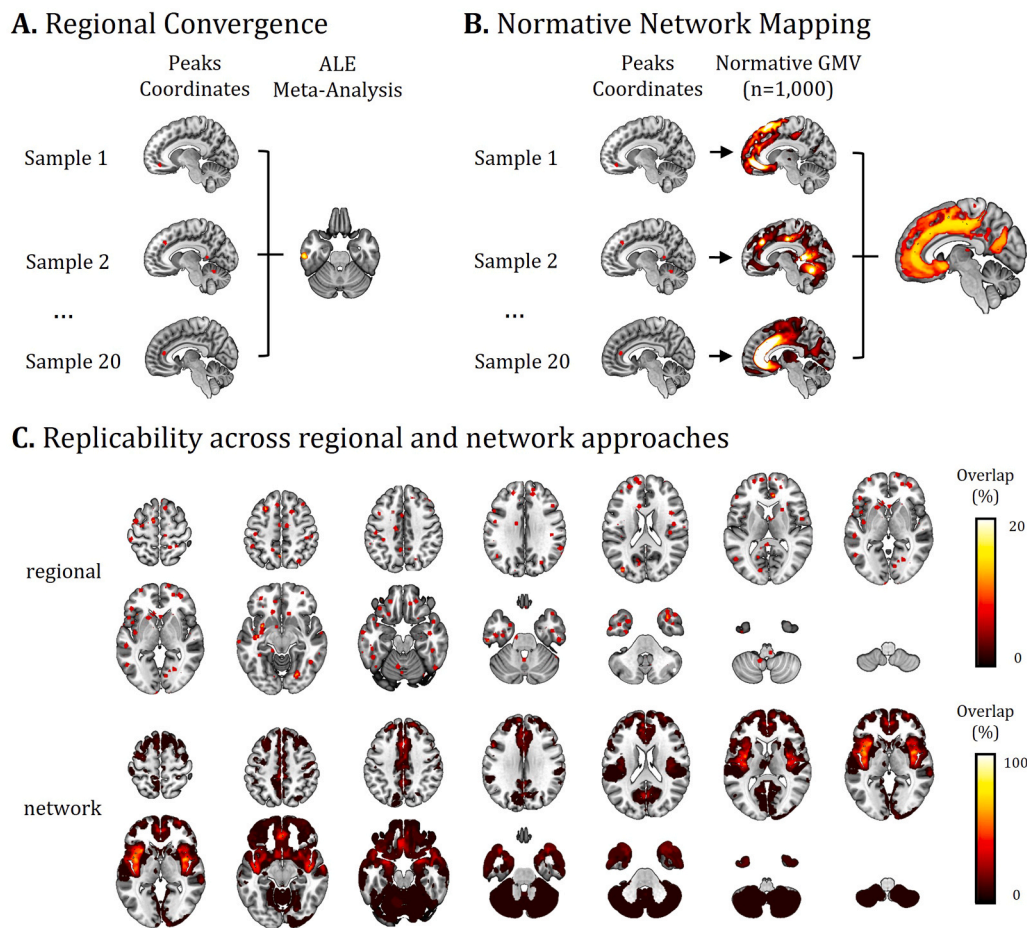


Fig. 2. Spatial Convergence across neuroimaging studies on Psychopathy. **A.** Activation Likelihood Estimation meta-analysis was conducted on peak coordinates of 20 samples examining GMV in Psychopathy, which revealed a small cluster in the left inferior temporal gyrus. **B.** Network mapping was subsequently performed to identify a convergent network across the 20 samples. **C.** Displays the differences in overlap between traditional approaches involving spatial convergence based on peak coordinates, and our framework assessing spatial convergence at a network level. The former resulted in low *regional* replicability (up to 15%) (upper row), and the latter produced high *network* replicability between samples (up to 70% of studies) (lower row).

psychopathic brain scores when examining basic personality dimensions (Supplementary Results, [Supplementary Fig 4](#), [Supplementary Table 11](#))

4. Discussion

The present study sought to investigate whether heterogeneous grey matter alterations associated with psychopathy are organized within a common structural network. First, we conducted an updated meta-analysis of structural neuroimaging samples examining the associations between GMV and psychopathy. We observed only a small effect in the left inferior temporal gyrus, driven by 3 of the 20 included samples. Consistent with our hypothesis, we showed that even though peak locations were highly heterogeneous, their structural covariance profile did robustly map onto a common structural network. This network was characterized by key hubs of the ventral attention and default mode network, with overlap reaching up to 70% across samples. These included the insula, extending from anterior to posterior regions into the lateral OFC/ventrolateral PFC, the posterior midcingulate cortex, the medial orbitofrontal cortex and amygdala to a lesser extent. External validation analyses were subsequently conducted to examine the behavioural and clinical relevance of the identified structural covariance network. First, in an exploratory analysis, we found that this network showed greater involvement in patients whose focal brain lesions preceded the onset of antisocial behaviours, particularly among those presenting with affective deficits and irresponsibility. Yet, given

the retrospective and selected nature of lesion case studies, these findings should be interpreted cautiously and considered preliminary. Next, we examined whether this network reflected meaningful individual differences in psychopathic traits using dimensional personality measures across humans and chimpanzees. In both species, variation within this network was associated with psychopathic and related personality traits, although the relative contributions of specific TriPM and FFM dimensions differed across species. These findings highlight the utility of a network-based framework for understanding psychopathic traits (Dugré and De Brito, 2025), and suggest that the link between this structural covariance network and socio-affective processes and behavioural control may have been conserved over the course of evolution.

Decades of research on the neuroanatomical correlates of psychopathy have identified numerous cortical and subcortical regions implicated in morality and decision-making, particularly the PFC and the amygdala (Blair, 2005; Damasio et al., 1990b; Raine, 2019). Despite recent concerns regarding the reliability of neuroimaging findings in this field (Deming et al., 2024, 2022; Jalava et al., 2023), our meta-analysis provides evidence for GMV alterations across several paralimbic regions, including the cingulate and insular cortices spanning anterior to posterior regions. These findings are consistent with the paralimbic dysfunction model of psychopathy (Baskin-Sommers et al., 2016; Kiehl, 2006). Overall, our findings suggest that these regions are embedded within a distributed structural network, providing a framework for understanding how anatomical variation across individuals is organized.

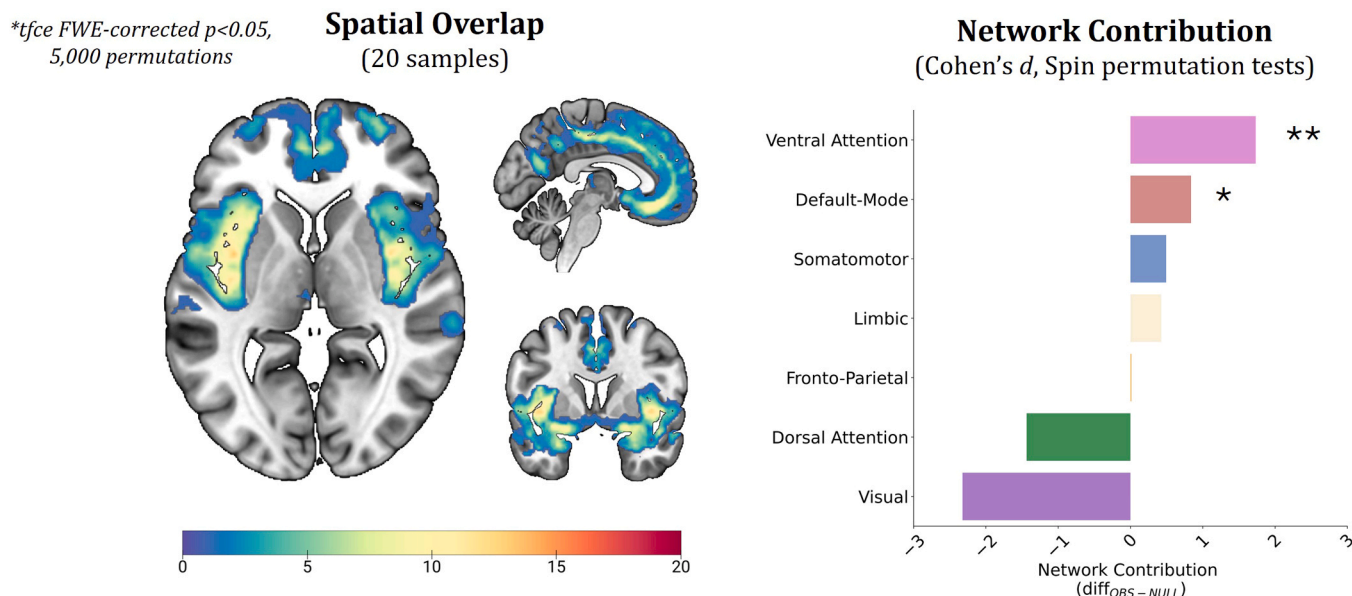


Fig. 3. Neural architecture of the psychopathy network. Left Panel. The Psychopathy Network was formed by the following key hubs: posterior and middle insula extending to superior temporal gyrus, anterior insula extending to lateral OFC/ventrolateral PFC, posterior midcingulate cortex, medial OFC/vmPFC, posterior OFC, and amygdala. Right Panel. This network was primarily driven by stronger structural covariance from nodes of the ventral attention/salience and default mode networks. The bar plot shows the mean difference between each network's observed mean and the mean network value across 5000 spatial autocorrelation-preserving null maps (* $P_{\text{spin}} < 0.01$, ** $P_{\text{spin}} < 0.001$).

within interconnected brain systems. Yet, the mechanisms driving these structural patterns in psychopathy remain largely unclear and warrant further investigation. For example, structural covariance is heavily influenced by genetic factors (Alexander-Bloch et al., 2013; Schmitt et al., 2010), which may be partially explained by regions sharing similar gene expression profiles (Romero-Garcia et al., 2018). Although previous studies have identified associations between specific genetic markers and psychopathy (Palumbo et al., 2022; Tiihonen et al., 2020), future research should investigate the extent to which variations in these markers influence the structural development and covariance of paralimbic regions.

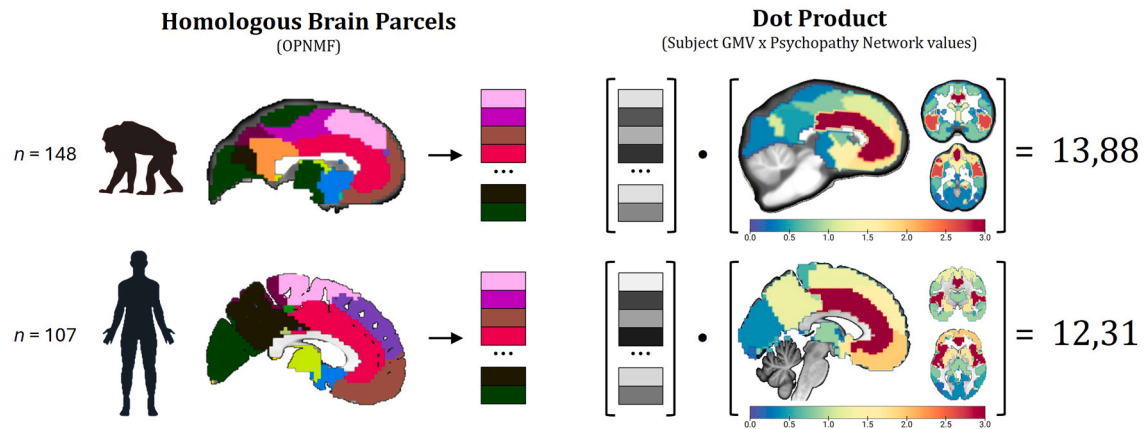
Next, we examined the extent to which variation in this structural network was related to severity in psychopathic traits across humans and chimpanzees. Consistent with previous work implicating paralimbic regions in personality traits related to impulse control and socio-affective processes in both humans (DeYoung et al., 2010; Kapogiannis et al., 2013) and chimpanzees (Latzman et al., 2015b; Rilling et al., 2012), we found that dimensions of the Triarchic Model explained a substantial proportion of variance in psychopathy brain scores. Moreover, dominance analyses revealed cross-species differences in relative contribution of specific Triarchic dimensions. For example, meanness and disinhibition made greater contributions in chimpanzees, whereas disinhibition made a substantial contribution in humans. Similarly, extraversion and dominance accounted for the greatest proportion of explained variance in psychopathy brain scores in chimpanzees, whereas conscientiousness accounted for the largest proportion in humans. One potential explanation for these differences lies in adaptations to changing socio-environmental demands over the course of evolution. Notably, many of the brain regions identified in the current study are involved in social cognition and are among those that underwent the greatest expansion during human evolution (Vickery et al., 2024). Evolutionary pressures toward self-control and norm-following may have driven a reconfiguration in brain connectivity among multimodal association areas (Ardesch et al., 2019), altering connectivity within networks supporting functions that rely heavily on the default mode network, such as language and social cognition (van den Heuvel et al., 2023). Such evolutionary differences, reflecting a shift from greater involvement of salience processing and visual detection in

chimpanzees toward more internally oriented processing in humans (van den Heuvel et al., 2023), may help explain differences in how similar brain patterns relate to behaviour across species. Nevertheless, it is also possible that these differences reflect restricted variability in disinhibition among chimpanzees due to environmental constraints (i.e., captivity), and reduced variability in meanness among community-based human samples, potentially reflecting self-selection biases in recruitment (e.g., volunteer or university-based recruitment). Other possibilities include measurement issues that could arise from using the same label for distinct phenomena (*jingle fallacy*, (Kelley, 1927), as shown by a moderate correlation in disinhibition factor score between species (Latzman et al., 2015b), and/or the use of a five-factor proxy of the TriPM in humans. While more extensive research is needed, our study provides evidence that our psychopathy structural network captures individual differences in psychopathic traits across human and chimpanzees.

5. Limitations

Several limitations need to be acknowledged. First, voxel-based morphometry studies examining GMV in psychopathy vary substantially in terms of demographics, clinical characteristics, MRI protocols, and statistical approaches. Such methodological heterogeneity may have contributed to an underestimation of the true replicability of GMV findings across studies. Second, our subanalyses on psychopathy dimensions (i.e., Factor 1 and Factor 2) were non-significant and the interpretation of the findings should be approached with caution given the small number of included samples. We encourage neuroimaging researchers to measure the neuroanatomical correlates of psychopathy facets to gain a more fine-grained understanding of their underlying structural correlates. Third, the weak effect observed in the traditional coordinate-based meta-analysis may reflect limited statistical power (Frahm et al., 2023). Fourth, we used GMV data of a large normative sample of healthy individuals ($n = 1000$, 50% females) to examine whether heterogeneous brain regions identified across studies mapped onto a common structural network. It is possible that differences in demographics between the included case samples and the normative sample may have resulted in some variations in network mapping. Fifth,

A. Modelling Psychopathic Brain Scores Across Species



B. Cross-Species Validation of Psychopathic Brain Scores

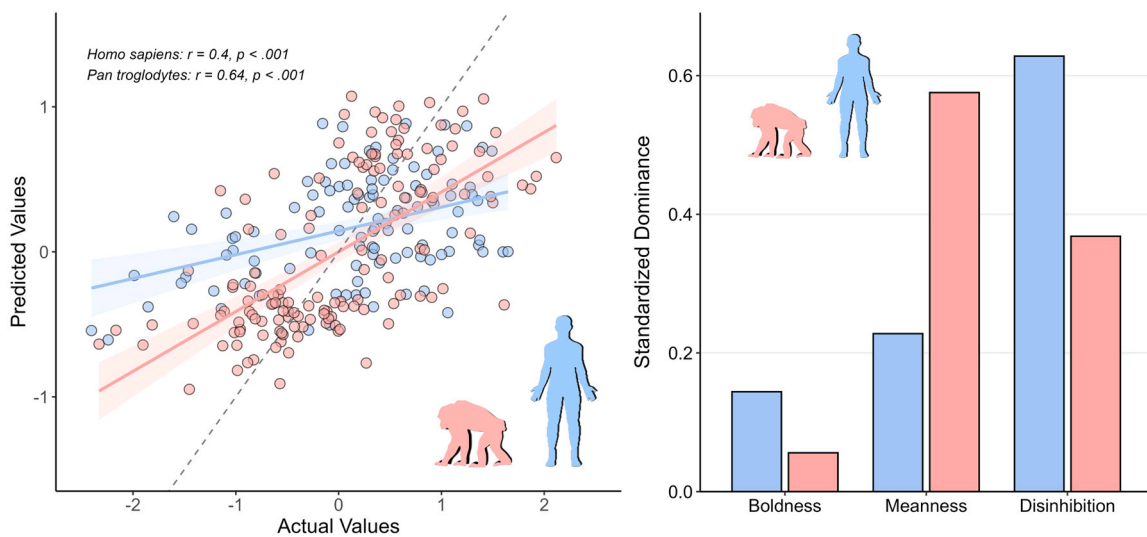


Fig. 4. External validation of the psychopathy network across species. Panel A illustrates the workflow used to compute psychopathic brain scores in chimpanzees (top row) and humans (bottom row). Briefly, the psychopathy network t-map was parcellated into 17 regions recently identified as homologous between humans and chimpanzees (Vickery et al., 2024). The resulting vector was used in a dot product with subject-specific raw gray matter volumes, parcellated using the same 17 regions, to generate a composite psychopathic brain score. Panel B shows the relationship between predicted and observed values (left panel), assessing how well TriPM subscales predict unique variance in psychopathic brain scores. The right panel depicts results from dominance analysis, revealing cross-species differences in the relative contribution of TriPM factors to prediction of psychopathic brain scores.

recent work has raised concerns about the specificity of lesion-network mapping findings (van den Heuvel et al., 2026), suggesting that lesion-derived networks may not be specific to the symptoms of interest. However, this concern has been challenged by two independent groups (Meng et al., 2026; Siddiqi et al., 2026), who emphasized the importance of conducting specificity analyses when interpreting lesion-network maps. In our study, exploratory analyses revealed that the structural covariance network showed specificity for lesions that preceded the onset of antisocial behaviours compared with control lesions. These findings provide evidence against the notion that lesion-network mapping generates similar network maps regardless of the clinical phenotype or input data. Yet, lesion data were included based on the broad criterion that individuals developed antisocial behaviours following focal brain lesions, which does not capture the full range of psychopathic traits. Although explicit measures of psychopathy were not available in the original case descriptions, we manually annotated reported symptoms of antisocial personality disorder. We found that cases reporting limited prosocial emotions and irresponsibility showed greater similarity to the structural covariance network than cases without these

features, suggesting that the observed association was not driven solely by antisocial behaviours. We acknowledge that these annotations were conducted by a single author (JRD) and should therefore be interpreted with caution. Further investigations using larger clinical samples and comprehensive assessments of psychopathic traits are warranted. We also acknowledge the potential biases inherent to retrospective cases studies. Longitudinal studies are warranted to provide a prospective and less biased assessment of the association between focal brain lesions and subsequent antisocial behavioural outcomes. Sixth, the meta-analysis primarily included studies assessing psychopathy using the PCL-R in correctional or forensic populations, whereas our cross-species analyses relied on dimensional personality measures from community samples (i.e., TriPM, Five-Factor Model, and Short Dark Triad). Although these frameworks show conceptual overlap, they differ in theoretical structure and emphasis. Future research should directly assess the extent to which different psychopathy models map onto distinct neurobiological profiles. In the present study, we used the original TriPM factor structure to allow direct comparison with chimpanzee data, which was previously validated in this sample (Latzman et al., 2016). We acknowledge that the

TriPM has been subject to ongoing psychometric debate, particularly regarding its factor structure (Collison et al., 2021; Patrick et al., 2021; Roy et al., 2021). There is also continuing debate regarding the centrality of boldness in psychopathy (Lilienfeld et al., 2016; Sleep et al., 2019). Notably, our cross-species analyses revealed that boldness may not be central to the explained variance in our identified brain network. Yet, dominance dimension of the five-factor model in chimpanzees showed a significant association with the psychopathy brain scores, therefore highlighting the importance of including different conceptualizations. In line with this view, we consider that comparing different conceptualizations of psychopathy is valuable for advancing the field (Patrick et al., 2021), without implying that any single framework fully captures the construct or invalidates the present findings. Seventh, in the current meta-analysis, assessment method and sample type were completely confounded. Specifically, all studies using clinician-rated instruments (e.g., the PCL-R/PCL-SV) were conducted in clinically ascertained samples, whereas studies using self-report measures were conducted exclusively in community samples. Although we observed high spatial similarity between the neural correlates identified using clinician-rated and self-report measures, the remaining differences may reflect characteristics of the underlying populations rather than the assessment instruments themselves. Future neuroimaging studies administering both clinician-rated and self-report measures within the same sample should aim to disentangle the independent contributions of assessment method and sample characteristics. Moreover, it remains unclear whether structural covariance networks derived separately for specific assessment methods and/or sample types would exhibit stronger generalizability to independent samples than our current network. Our findings provide preliminary evidence that the identified network, derived primarily from clinically ascertained samples assessed with the PCL-R, generalizes to independent community samples assessed with self-report measures. Nevertheless, future meta-analyses with a larger number of studies should directly evaluate whether assessment-specific or sample-specific networks differs significantly and/or offer improved generalizability. Eighth, we relied on a meta-analytic rather than a mega-analytic approach which could have overestimated the replicability using group-level estimates. International, large-scale collaborative initiatives such as the ENIGMA-Antisocial Behaviour Working Group, in which individual-level data are shared, may provide a more precise understanding of the neurobiological substrates of Psychopathy. Seventh, given the absence of TriPM measure for our human sample, we relied on a five-factor composite score of the TriPM. While this statistical approach is a proxy measure of the actual TriPM, future research may seek to replicate our findings using the actual TriPM scale. Ninth, the association between the Short Dark Triad and brain scores provides convergent support for the TriPM findings, reducing the likelihood that these results reflect chance, although the measure itself has notable psychometric limitations (Glenn and Sellbom, 2015; Miller et al., 2019). Future studies should seek to replicate our findings using other measures and/or other populations such as the PCL-R. Tenth, the chimpanzee personality data are based on ratings provided by humans familiar with the individuals living in captivity, which may limit the generalizability of these findings. Indeed, captive environments can constrain behavioural repertoires relative to wild settings (e.g., due to housing conditions, management practices, and research procedures). As a result, the observed trait ratings may not capture the full breadth of naturalistic chimpanzee behaviour as expressed in wild populations.

6. Conclusion

Over the past decades, neuroimaging meta-analyses and systematic reviews on the structural brain correlates of psychopathy have yielded inconsistent findings (Aoki et al., 2014; De Brito et al., 2021b; Deming and Koenigs, 2020; Johanson et al., 2019), raising concerns about the reliability of such findings (Deming et al., 2024, 2022; Jalava et al., 2023). Here, we show for the first time that heterogeneous peak

locations across samples do in fact converge onto a common structural covariance network of brain volume. Crucially, we demonstrate that this network, composed mainly of paralimbic regions, is strongly associated with the severity of psychopathic traits in both humans and chimpanzees. These findings underscore the relevance of chimpanzee personality research for understanding human mental health (Latzman et al., 2017), including severe personality disturbances such as psychopathy. In response to the growing scepticism about the reliability of neuroimaging findings, adopting a network-based approach offers promising conceptual and methodological advancements for our understanding of the neurobiology of psychopathy.

Authors contributions

J.R.D. performed the systematic search, conducted all analyses and wrote the manuscript. S.A.D.B. assisted with the systematic search, interpreted the results and contributed to manuscript preparation. S.V., R.D.L., W.D.H., and F.H. contributed to chimpanzees' data collection and aggregation, and contributed to manuscript preparation. All authors revised and approved the final version of the manuscript.

Code Availability

Coordinate-based meta-analysis was conducted using JALE (<https://github.com/juaml/JALE>), a python package for conducting ALE (Activation Likelihood Estimation) meta-analyses (Eickhoff et al., 2012), <http://www.brainmap.org/ale>). Structural Network Mapping was conducted using CAT12 (Gaser et al., 2024), Computational Anatomy Toolbox; www.neuro.uni-jena.de/cat/ in SPM12 (Statistical Parametric Mapping; www.fil.ion.ucl.ac.uk/spm/software/spm12/) following similar steps as in functional network mapping (Dugré and De Brito, 2025; Stubbs et al., 2023; Wall et al., 2025, https://github.com/nimlab/NMH_Stubbs2023).

Declaration of Competing Interest

The authors declare no potential conflicts of interest.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.neubiorev.2026.106929](https://doi.org/10.1016/j.neubiorev.2026.106929).

Data Availability

The chimpanzee sample (www.chimpanzeebrain.org) can be provided by NCBR pending scientific review and a completed material transfer agreement. Request for the chimpanzee data should be submitted to: NCBR. The two human neuroimaging datasets, Brain Genomics Superstruct Project (<https://dataverse.harvard.edu/dataset/GSP>) and Mind-Brain-Body dataset from the Max Planck Institute for Human and Cognitive and Brain Sciences (https://fcon_1000.projects.nitrc.org/indi/retro/MPI_LEMON.html),

are openly available.

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