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Psychopathy and medial frontal cortex: A systematic review reveals predominantly null relationships

Philip Deming [a,*], Stephanie Griffiths [b,c], Jarkko Jalava [d], Michael Koenigs [e], Rasmus Rosenberg Larsen [f]

[a] Department of Psychology, Northeastern University, Boston, MA, United States
[b] Department of Psychology, Okanagan College, Penticton, BC, Canada
[c] Werklund School of Education, University of Calgary, Calgary, AB, Canada
[d] Department of Interdisciplinary Studies, Okanagan College, Penticton, BC, Canada
[e] Department of Psychiatry, University of Wisconsin-Madison, Madison, WI, United States
[f] Forensic Science Program and Department of Philosophy, University of Toronto Mississauga, Mississauga, ON, Canada

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ABSTRACT

Theories have posited that psychopathy is caused by dysfunction in the medial frontal cortex, including ventromedial prefrontal cortex (vmPFC), anterior cingulate cortex (ACC), and dorsomedial prefrontal cortex (dmPFC). Recent reviews have questioned the reproducibility of neuroimaging findings within this field. We conducted a systematic review to describe the consistency of magnetic resonance imaging (MRI) findings according to anatomical subregion (vmPFC, ACC, dmPFC), experimental task, psychopathy assessment, study power, and peak coordinates of significant effects. Searches of PsycInfo and MEDLINE databases produced 77 functional and 24 structural MRI studies that analyzed the medial frontal cortex in relation to psychopathy in adult samples. Findings were predominantly null (85.4 % of 1573 tests across the three medial frontal regions). Studies with higher power observed null effects at marginally lower rates. Finally, peak coordinates of significant effects were widely dispersed. The evidence failed to support theories positing the medial frontal cortex as a consistent neural correlate of psychopathy. Theory and methods in the field should be revised to account for predominantly null neuroimaging findings.

1. Introduction

The medial frontal cortex has long been considered a potential neural correlate of psychopathy, which is defined by a deceitful interpersonal style, callousness towards the welfare of others, a reckless and irresponsible lifestyle, and repeated and varied criminal behavior (Crego and Widiger, 2015; Hart and Cook, 2012). Psychopathy has been identified as a risk factor for crime (Gillespie et al., 2023; Monahan et al., 2001; Reidy et al., 2015) and recidivism (J. R. Anderson et al., 2018; Leistico et al., 2008), and psychopathic persons are therefore commonly believed to place a disproportionate burden on society in terms of the financial cost and human toll of their behavior (Gatner et al., 2023; Kiehl and Hoffman, 2011; Reidy et al., 2015). While the exact social cost of psychopathy is difficult to ascertain and perhaps a contested issue (Verona and Joyner, 2023), reducing these costs has been a primary motivation for research into potential neurobiological causes of psychopathy (Glenn and Raine, 2014; Nadelhoffer et al., 2012). Discovering such causes could potentially assist the development of effective treatment programs, yet recent work has questioned the consistency of findings in this field (Deming et al., 2022; Griffiths and Jalava, 2017; Jalava et al., 2021, 2023; Koenigs et al., 2011).

Perhaps the first evidence linking medial frontal cortical function to psychopathic behavior came from the famous case of Phineas Gage, a railway worker who suffered extensive damage to the ventromedial prefrontal cortex (vmPFC; Fig. 1) in a railway construction accident. Gage survived, but according to some anecdotal reports his personality changed markedly (cf. Schleim, 2022). The once dependable Gage was nearly unrecognizable to his friends following the accident, now showing signs of disrespectful, impulsive, and irresponsible behavior somewhat consistent with the definition of psychopathy (Damasio et al., 1994; Harlow, 1868). A century later, the profile of socioemotional dysfunction that typically arises following vmPFC injury was termed

* Corresponding author.
E-mail address: p.deming@northeastern.edu (P. Deming).

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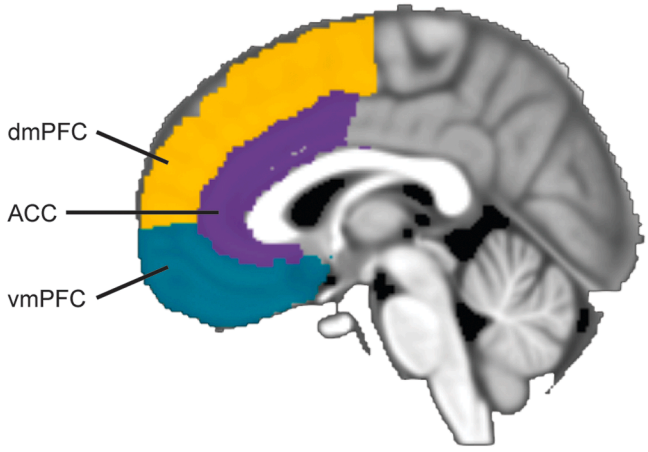

Fig. 1. The medial frontal cortex consisting of ventromedial prefrontal cortex (vmPFC; teal), anterior cingulate cortex (ACC; purple), and dorsomedial prefrontal cortex (dmPFC; gold). Region of interest masks were derived from the Brainnetome Atlas (Fan et al., 2016).

"pseudopsychopathy" (Blumer and Benson, 1975) or "acquired sociopathy" (Damasio, 1994).

Several theories have posited a vital role for vmPFC in the etiology of psychopathy, founded primarily in evidence from neuropsychological assessments of human patients with vmPFC damage and from neuroimaging studies of healthy human participants. This evidence has established a role for vmPFC in decision-making (e.g., representing the value of stimuli and outcomes), emotion (e.g., representing negative affect, regulating negative affect and pain), and social cognition (e.g., empathy; Hiser and Koenigs, 2018). The *integrated emotion systems* theory proposed that psychopathy might be caused by vmPFC dysfunction, impairing the capacity to quickly alter behavior following contingency changes (i.e., response reversal), leading to an increased risk for frustration-based aggression (Blair, 2005). Blair (2007) further argued that psychopathy could be associated with impaired representation of valenced outcomes due to vmPFC dysfunction, leading to impairments in decision-making, especially moral decision-making (cf. Sackris, 2022). Similarly, the *paralimbic hypothesis* proposed that vmPFC dysfunction could explain the traits conceptually associated with psychopathy, such as impulsive decision-making and lack of empathy (Kiehl, 2006). Each of these theories claimed support from studies showing that psychopathic people perform similarly to patients with vmPFC damage on tasks measuring response reversal (Mitchell et al., 2006, 2002), moral decision-making (Anderson et al., 1999; Koenigs et al., 2012), economic decision-making (Koenigs et al., 2010), and impulsivity (Lapierre et al., 1995).

Notably, the *paralimbic hypothesis* attributed psychopathy to dysfunction not just within vmPFC but within a large swath of cortex and subcortex, including another medial frontal region, the anterior cingulate cortex (ACC; Fig. 1). Among healthy individuals, ACC has been implicated in a host of affective (e.g., emotion (Lindquist et al., 2012), emotion regulation (Ochsner et al., 2012; Stevens, 2011), pain (Wager et al., 2013)) and executive functions (e.g., action selection (Rushworth, 2008), error monitoring (Alexander and Brown, 2019), cognitive control (Menon and Uddin, 2010; Shenhav et al., 2013)). The *paralimbic hypothesis* proposed that psychopathy may be related to ACC dysfunction, which might cause impairments in empathy, error monitoring, and response inhibition (Kiehl, 2006). Though ACC lesions are rare, patients often present with symptoms associated with psychopathy, including apathy (Kumral et al., 2019; Mesulam, 2000), difficulty empathizing (Hornak, 2003), impaired error monitoring (Maier et al., 2015; Swick and Turken, 2002), and impaired response inhibition (Degos et al., 1993). As more direct evidence, our previous meta-analysis found that psychopathy was related to underactivity of the dorsal ACC across studies (Deming and Koenigs, 2020). We proposed that dysfunction of the dorsal ACC, a key node of the salience network, may contribute to impairments in detecting salient cues and allocating attentional resources to process those cues (Deming et al., 2023; Deming and Koenigs, 2020).

Though relatively less theoretical work has linked psychopathy to a third medial frontal region, the dorsomedial prefrontal cortex (dmPFC; Fig. 1), there is reason to consider this region as a potential key neural correlate as well. Evidence from healthy people has implicated dmPFC broadly in social cognition (e.g., thinking about oneself (Whitfield-Gabrieli et al., 2011), thinking about other people (Denny et al., 2012), and social interactions (Skerry and Saxe, 2014; Wagner et al., 2016)). Additionally, dmPFC is a node within the default mode network, a constellation of regions whose activity tends to decrease when a person engages in an externally-focused task (Raichle, 2015). Our previous meta-analysis found that psychopathy was related to dmPFC overactivity across a variety of tasks (Deming and Koenigs, 2020). This finding aligned with a proposal made by Freeman and colleagues, that psychopathic individuals fail to adaptively inhibit dmPFC activity (and activity within other medial default mode network regions) when engaged in externally-focused tasks (Freeman et al., 2015). However, an earlier meta-analysis contradicted this finding: Poeppl et al. (2018) found that psychopathy was related to dmPFC *under*activity across studies and proposed this may contribute to psychopathic individuals' lack of empathy and remorse.

In fact, inconsistency is characteristic of much of the empirical evidence that has been cited in support of neurobiological theories of psychopathy. Patients with damage to vmPFC or ACC rarely present with the full constellation of traits associated with psychopathy (Kiehl, 2006). Furthermore, recent systematic reviews suggest that other potential neural correlates such as the amygdala are not as consistently related to psychopathy across studies as previously thought (Deming et al., 2022; Jalava et al., 2021). Direct replications of neuroimaging evidence in this field are lacking (Jalava et al., 2023), perhaps partly because psychopathy is defined in numerous different ways across heterogeneous samples (Koenigs et al., 2011). Though our previous meta-analysis found relationships between psychopathy and activity of the ACC and dmPFC, the meta-analysis included only studies that analyzed the whole brain (i.e., excluding region of interest analyses). Thus, these meta-analytic findings do not represent the entire neuroimaging literature on psychopathy. In sum, inconsistencies in the neuropsychological and neuroimaging literature have made for an unclear picture of the relationship between psychopathy and the structure and function of the medial frontal cortex. No systematic review to date has quantified the consistency of this relationship across studies.

The current study aimed to address this gap. We systematically reviewed magnetic resonance imaging (MRI) studies of psychopathy in adult samples to quantify the consistency of null, negative (i.e., reduced activity or volume), and positive (i.e., increased activity or volume) relationships between psychopathy and medial frontal cortex across studies. Our review included any study that had the potential to observe a relationship between psychopathy and task-based activity or gray matter structure of vmPFC, ACC, or dmPFC, by examining these regions either as part of the whole brain or as a focal region of interest (ROI). To examine the context specificity of medial frontal dysfunction, we examined the consistency of findings within specific experimental tasks, such as face processing tasks. Similarly, we examined the consistency of findings as a function of the type of psychopathy assessment employed (i.e., clinician-rated vs. self-report), and in relation to study power as indexed by sample size. Lastly, we examined the spatial consistency of

medial frontal findings by extracting the peak coordinates of significant medial frontal clusters. We conducted each of these examinations for findings related to total psychopathy scores, as well as findings related to specific clusters of psychopathic traits, including interpersonal/affective (e.g., grandiosity, deceitfulness, shallow affect, lack of empathy) and lifestyle/antisocial traits (e.g., impulsivity, irresponsibility, varied criminal behavior).

2. Methods

We performed a PRISMA (Page et al., 2021) database and manual search for MRI studies investigating task-based function (blood oxygen level dependent response; BOLD) and gray matter structure of the medial frontal regions in relation to psychopathy in adult samples. The search was conducted on March 11, 2023 and updated on September 11, 2023. The pre-registration and complete dataset for this study can be accessed via Open Science Framework: https://osf.io/juvkm/?view_only=e0ffce18332e49c3a4b552bbbc973042.

2.1. Inclusion and exclusion criteria

Studies were deemed eligible for coding if they met the following inclusion criteria: (1) analyzed task-based BOLD response or gray matter structure of the medial frontal regions using whole-brain or ROI approach, (2) tested the main effect of a validated measure of psychopathy, (3) reported group statistics rather than single-case results, (4) recruited adult samples defined by age $\geq$ 18, non-overlapping with youth samples defined as age $<$ 18, (5) was published as academic peer-reviewed experimental research, and (6) full-text available in English. In eligible studies, we only included analyses that examined any variation in MRI signal and levels of psychopathy, including continuous analyses (e.g., correlations with psychopathy assessment scores) and group-based analyses (e.g., low vs. high, low vs. mid psychopathy groups). We excluded analyses that were based on ad-hoc or non-clinical psychopathy groupings, for example, "successful vs. unsuccessful", "low-anxious vs. high-anxious", or "primary vs. secondary" psychopathy. We also excluded interaction analyses between task conditions (e.g., Task (Ultimatum Game vs. Dictator Game) x Fairness (Unfair vs. Fair)).

2.2. Search protocol and results

One author executed a full-text, English language-only search for studies published between 1990 and 2023 in PsycInfo and MEDLINE (via the OVID search engine). The search was conducted using a Boolean combination of terms: (psychopathic OR psychopathy OR antisocial) AND (MRI OR magnetic resonance imaging OR neuroimaging). All database search results were imported into Endnote 20 (Clarivate Analytics). Furthermore, a manual search was conducted by another author, screening a recently published review for records that were not identified in the database search (i.e., Deming et al., 2022)

The search returned a total of 635 records (PsycInfo = 340, MEDLINE = 295). We excluded 234 records by screening the titles for duplicates or obvious irrelevance. By scanning the abstract from the remaining 401 records, we further excluded 217 entries for the following reasons: did not assess psychopathy (n = 106), did not analyze BOLD or gray matter structure (n = 55), and not on adult samples (n = 56). A full-text review was conducted of the remaining 184 entries, after which 99 were excluded for the following reasons: did not assess psychopathy (n = 52), did not analyze BOLD or gray matter structure (n = 23), not on adult samples (n = 6), or had other issues (n = 18) (e.g., case study, not peer-reviewed, incomplete reporting, etc.). An additional 16 records were retrieved from scanning recently published review studies and meta-analysis, bringing the total number of included studies to 101. The PRISMA workflow is displayed in Fig. 2.

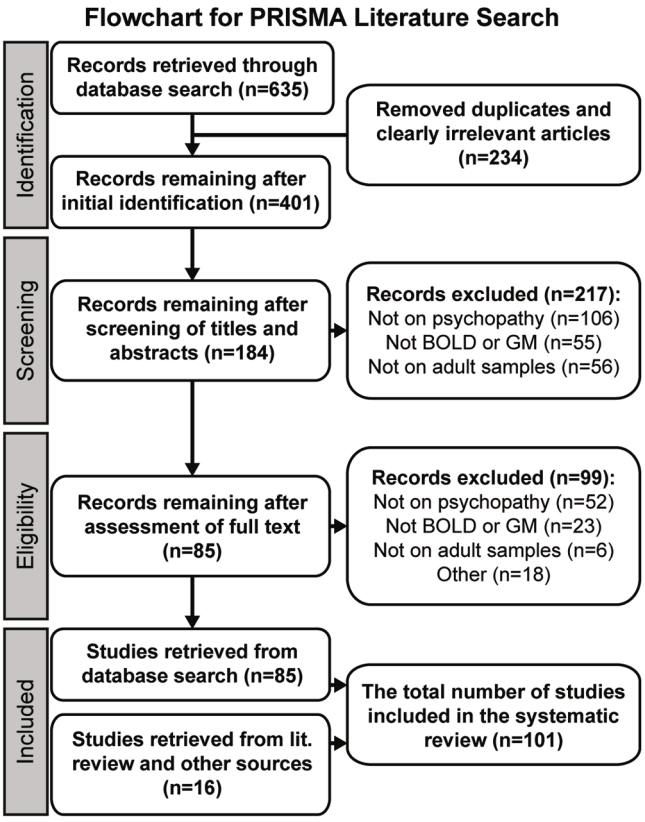

Fig. 2. PRISMA flow chart of the literature search process. Abbreviations: BOLD = blood oxygen level dependent response, GM = gray matter structure.

2.3. Definition of regions of interest

We defined ROIs based on neuroanatomical boundaries. For our primary analyses, we examined large ROIs (covering several subregions with different cytoarchitectural profiles; Fig. 1) that were germane to neurobiological theories of psychopathy (e.g., Blair, 2005; Kiehl, 2006). Primary regions of interest were vmPFC, defined as the medial portion of frontal cortex anterior to the cingulate sulcus and inferior to the genu of the corpus callosum, covering medial portions of Brodmann areas (BAs) 11, 12, and 10 (inferior to the genu of the corpus callosum); ACC, defined as the portion of the cingulate gyrus/sulcus anterior to the paracentral sulcus, covering BAs 24, 25, 32 and 33; and dmPFC, defined as the medial portion of frontal cortex superior to the genu of the corpus callosum and anterior to the paracentral sulcus, covering medial portions of BAs 6, 8, 9, and 10 (superior to the genu of the corpus callosum). Note that our definition of dmPFC extends more posteriorly than some other definitions (i.e., into BA 6; Clairis and Lopez-Persem, 2023). To account for heterogeneous neuroanatomical labeling across studies, we developed a list of relevant labels for each medial frontal region and coded all findings for the listed labels (see Table S1).

To examine the spatial consistency of peak coordinates, we derived masks of these primary ROIs, as well as subregions within these ROIs, from the Brainnetome Atlas (Fan et al., 2016) in AFNI 21.0 (Cox, 1996). The Brainnetome Atlas comprises 210 cortical regions that were defined based on functional connectivity. The atlas's medial frontal cortical regions approximate Brodmann areas (which follow cytoarchitectural boundaries). For the vmPFC mask, we aggregated Brainnetome Atlas regions 11 m (covering medial BA 11, medial anterior BA 10, and anterior BA 12), 13 (posterior BA 12 and BA 25), and 14 m (medial posterior BA 10 and superior BA 12). For the ACC mask, we aggregated atlas regions 24rv (BA 33 and inferior BA 24), 24 cd (superior BA 24), 32p (superior BA 32) and 32sg (inferior BA 32). Lastly, for the dmPFC mask, we aggregated atlas regions 6 m (medial BA 6), 8 m (medal BA 8),

9 m (medial BA 9), and 10 m (medial superior BA 10). The constituent subregions are displayed in Fig. S1.

2.4. Data coding

All included studies were double-coded. Four authors performed the coding, with each person responsible for coding 50 % of the eligible studies. Disagreements between two coders were resolved through group-decision by all four coders. Studies were coded for the correlation and/or group-based differences between task-based BOLD response or gray matter structure and three psychopathy measures: total psychopathy, interpersonal/affective traits, and lifestyle/antisocial traits. Measures of total psychopathy assessed the full set of psychopathic traits (i.e., all subsets of traits for that measure). Measures of interpersonal/affective traits assessed subsets of psychopathic traits related to deceitful interpersonal style and/or shallow emotionality, while measures of lifestyle/antisocial traits assessed subsets of psychopathic traits related to impulsive and/or irresponsible lifestyle and criminal behavior. We coded analyses of Psychopathy Checklist-Revised (PCL-R) facets (interpersonal, affective, lifestyle, antisocial) only if the study did not also report analyses of the superordinate PCL-R factors (interpersonal/affective, lifestyle/antisocial). Statistical contrasts were coded such that "positive" activity indicated greater activity in the psychological function of interest (e.g., Fearful Faces > Neutral Faces). We coded all variations of functional main effects contrasts and structural measures. For instance, most functional studies analyzed multiple task-based contrasts (e.g., Fearful Faces > Neutral Faces, Reward > No Reward, etc.), and some structural studies reported various measures of gray matter structure (e.g., volume, concentration).

In adhering to these planned analyses, the final dataset was generated by tracking null, positive, and negative effects using the following definitions. Analyses that did not reach statistical significance (i.e., $p < .05$; $p_{FWE} < .05$ for voxel-wise analyses) were coded as null. Analyses that found a significant increase (i.e., $p < .05$; $p_{FWE} < .05$ for voxel-wise analyses) in volume/task-based activity in psychopathic samples relative to non-psychopathic samples, or a significant positive correlation between psychopathy scores and volume/task-based activity were coded as positive effects. Analyses that found a significant decrease (i.e., $p < .05$; $p_{FWE} < .05$ for voxel-wise analyses) in volume/task-based activity in psychopathic samples relative to non-psychopathic samples, or a significant negative correlation between psychopathy scores and volume/task-based activity were coded as negative effects. To examine the consistency of findings across studies, we summed the number of studies that reported at least one null, negative, or positive finding. Percent was calculated by dividing the sums by the total number of studies that analyzed the measure of interest (e.g., total psychopathy related to vmPFC activity). As each study could report a mixture of findings (e.g., null and negative), it was possible for cumulative percentages to exceed 100 %. In supplemental analyses, we calculated the percentage of *individual tests* (rather than studies) that yielded null, negative, or positive findings (see Supplemental Materials).

2.5. Examination of peak coordinates

To characterize the spatial consistency of psychopathy findings in medial frontal cortical regions, we extracted the peak coordinates of clusters that were both significantly related to psychopathy and labeled as falling within one of the three medial frontal regions of interest. Fewer than five studies of gray matter structure reported peak coordinates of significant clusters within the vmPFC, ACC, or dmPFC. Therefore, we only examined peak coordinates reported by fMRI studies of medial frontal cortical activity. Peak coordinates were coded as negative (reduced activity) or positive (increased activity) as described above. Talairach coordinates were converted to Montreal Neurological Institute (MNI) template space using the MNI-Talairach Tool of the BioImage Suite (Lacadie et al., 2008). Around each peak coordinate we created a sphere with a radius of 6 mm (a typical smoothing kernel for analyses of cortical BOLD activity; Weibull et al., 2008). We then mapped the spheres onto Brainnetome atlas masks of the vmPFC, ACC, and dmPFC, as well as each of the constituent subregions. Finally, we calculated the percent overlap between each peak coordinate and each medial frontal mask (3dABoverlap in AFNI). Peak coordinates overlapping ≥ 50.0 % with the mask were determined to be within the ROI, whereas peak coordinates overlapping < 50.0 % with the mask were determined to be primarily outside the bounds of the ROI.

2.6. Examination of experimental tasks

To examine the consistency of findings relating psychopathy to medial frontal cortical activity during similar psychological tasks, we labeled the experimental task(s) used by each study. Task labels reflected the stimuli and task demands within each study and captured similarities across studies. We examined the consistency of findings separately for tasks that were employed by at least five studies across regions of interest, including "prototypical facial emotion expressions," "moral" tasks, and "empathy" tasks. Prototypical facial emotion expressions tasks were those that required viewing facial configurations typically associated with discrete emotions (e.g., fear, sadness). Moral tasks were those that required making judgments about or rating the moral severity of morally-laden scenarios, or that portrayed moral dilemmas or images but did not require a judgment. Empathy tasks were those that required participants to identify another person's emotional state using information other than (or in addition to) facial configuration.

2.7. Examination of psychopathy assessment

We examined the consistency of findings related to the type of psychopathy assessment tool. Psychopathy assessments were grouped into two categories: clinician-rated assessments in the Psychopathy Checklist (PCL) family, including the Psychopathy Checklist-Revised (Hare, 2003) and Psychopathy Checklist: Screening Version (Hart et al., 1995); and self-report assessments, including the Levenson Self-Report Psychopathy Scale (Levenson et al., 1995), NEO Five Factor Inventory (Costa and Macrae, 1992), NEO Triarchic Scale (Drislane et al., 2018), Psychopathic Personality Inventory (Lilienfeld and Andrews, 1996), Psychopathic Personality Inventory-Revised (Lilienfeld and Widows, 2005), Psychopathic Personality Inventory-Short Form (Tonnaer et al., 2013), Short Dark Triad (Jones and Paulhus, 2014), Self-Report Psychopathy Scale-Short Form (Paulhus et al., 2015), Inventory of Callous-Unemotional Traits (Frick, 2004), Triarchic Psychopathy Measure (Patrick, 2010), and Youth Psychopathy Inventory-Short Version (Van Baardewijk et al., 2010). We compared the consistency of findings from studies that assessed psychopathy via PCL vs. self-report. Additionally, there was substantial overlap between psychopathy assessment and study population. That is, nearly every study that assessed psychopathy via the PCL recruited participants from prisons, jails, detention centers, and forensic hospitals (i.e., forensic samples). Similarly, most studies that assessed psychopathy via a self-report measure recruited participants from the general public (i.e., community samples). We report the consistency of findings within forensic vs. community samples in the Supplemental Materials.

2.8. Examination of study power

Small sample size is a common shortcoming of neuroimaging studies that hinders study power (Button et al., 2013; Cremers et al., 2017; Marek et al., 2022; Poldrack et al., 2017; Szucs and Ioannidis, 2020). We therefore used sample size as an approximation of study power in order to examine the potential effects of study power on the consistency of medial frontal cortical findings. We separately examined the top one-third and bottom one-third of studies based on sample size (following the method of Deming et al., 2022).

2.9. Supplemental examinations

In addition to our primary analyses, we examined whether several measures of study quality (i.e., statistical choices) or MRI field strength was related to the consistency of null and significant findings. Details of these supplemental examinations are in Supplemental Materials.

3. Results

A total of 101 studies of psychopathy were included in the review. Seventy-seven studies reported 1322 total tests of task-based BOLD activity across the three medial frontal regions. The majority of these tests (85.9 %) yielded null relationships between psychopathy and medial frontal cortical activity. Furthermore, 98.7 % of functional studies observed at least one null effect, while 42.9 % observed only null effects. Twenty-four studies reported 251 total tests of gray matter structure across the three medial frontal regions. The majority of structural tests (83.3 %) yielded null effects. Additionally, 95.8 % of structural studies observed at least one null effect, while 45.8 % observed only null effects. In the following subsections we report specific examinations of functional and structural studies within each medial frontal region.

3.1. Functional studies of ventromedial prefrontal cortex

3.1.1. Results across all studies

A total of 68 studies reported 455 total tests of task-based BOLD activity in the vmPFC in relation to a measure of psychopathy (Table 1). The majority of these tests (89.2 %) yielded null effects. The majority of studies of vmPFC activity found at least one null relationship (≥ 93.3 % of studies for each psychopathy measure; Fig. 3). Negative relationships with total psychopathy (13.3 %), interpersonal/affective traits (14.7 %), and lifestyle/antisocial traits (25.0 %) were more common than positive relationships with these measures (10.0 %, 8.8 %, and 15.6 %, respectively).

3.1.2. Peak coordinates of results across all studies

We extracted 22 peak coordinates of significant vmPFC clusters related to total psychopathy from 10 studies (Table 2, Fig. 4). Of the 14 peak coordinates negatively related to total psychopathy, only four overlapped ≥ 50.0 % with the vmPFC mask, while nine overlapped 0.1–49.9 %, and one overlapped 0.0 % with the vmPFC mask. These peak coordinates were scattered across the medial frontal cortex, rather than consistently localized to a vmPFC subregion (areas 11 m, 13, or 14 m). Similarly, only three of the eight peak coordinates positively related to total psychopathy overlapped ≥ 50.0 % with the vmPFC mask, while four overlapped 0.1–49.9 %, and one overlapped 0.0 % with the vmPFC mask. Again, these peak coordinates were not consistently localized to a vmPFC subregion. Additionally, we extracted 32 peak coordinates of significant vmPFC clusters related to interpersonal/affective and lifestyle/antisocial traits from 15 studies. Patterns for these coordinates resembled those for the peak coordinates related to total psychopathy (see Supplemental Materials).

3.1.3. Results within tasks

Thirteen studies reported 93 total tests of vmPFC activity in prototypical facial expressions tasks, 14 studies reported 69 total tests in moral tasks, and ten studies reported 63 total tests in empathy tasks. Overall, the results within the most common experimental tasks paralleled the results across studies, with the exception of empathy tasks. The majority of tests (≥ 81.7 % for each task category) yielded null effects. The majority of studies within each task found at least one null relationship (≥ 66.7 % of studies for each psychopathy measure for each task category; Fig. 3). Within empathy tasks, a greater proportion of studies found a significant negative relationship with total psychopathy (37.5 %) and lifestyle/antisocial traits (50.0 %) than we observed across studies. Within prototypical facial expressions tasks, studies were more likely to observe a negative relationship with total psychopathy (30.0 %), interpersonal/affective traits (25.0 %) and lifestyle/antisocial traits (33.3 %). Similar to the full review, few studies that employed the most common experimental tasks found a positive relationship (≤ 16.7 % of studies for each psychopathy measure). For each of these tasks, results of specific contrasts are presented in Supplemental Materials.

3.1.4. Results within self-report and PCL assessments

A total of 25 studies reported 138 total tests of vmPFC activity in relation to a self-report psychopathy assessment. These results mirrored the results from the full review across studies. The majority of tests (90.6 %) yielded null effects. The majority of studies found at least one null relationship (≥ 84.6 % of studies for each psychopathy measure; Fig. 3). Studies that used self-report assessments were equally likely to observe a negative (23.1 %) or positive (23.1 %) relationship between vmPFC activity and lifestyle/antisocial traits. Similar to the full review, few studies that used self-report assessments observed a significant negative relationship with total psychopathy or interpersonal/affective traits (≤ 13.3 % for each measure) or a positive relationship with these two measures (≤ 13.3 % for each measure).

A total of 46 studies reported 317 tests of vmPFC activity in relation to a PCL assessment. These results also mirrored the results from the full review across studies. The majority of tests (88.6 %) yielded null effects. The majority of studies found at least one null relationship (≥ 93.3 % of studies for each psychopathy measure; Fig. 3). Fewer studies observed a significant negative relationship (≤ 25.0 % for each psychopathy measure) or positive relationship (≤ 10.0 % for each psychopathy measure).

3.1.5. Results of lowest- and highest-powered studies

The lowest-powered studies consisted of 24 studies that reported 203 total tests of vmPFC activity. The studies included in the full review had a mean sample size of $N = 63.1$ (range: 9–311), whereas the lowest-powered studies had a mean sample size of $N = 22.1$ (range: 9–33). The majority of tests (93.6 %) yielded null effects. All of the lowest-powered studies found at least one null relationship (100.0 % of studies for each psychopathy measure, Fig. 3). In contrast to the full review, nearly half of studies found a negative relationship with lifestyle/antisocial traits (44.4 %), although few found a negative relationship with total psychopathy (8.7 %) interpersonal/affective traits (22.2 %). Similar to the full review, few studies found a positive relationship (≤ 11.1 % of studies for each psychopathy measure).

The highest-powered studies consisted of 23 studies that reported 180 total tests of vmPFC activity, with a mean sample size of $N = 123.0$ (range: 57–311). The majority of tests (81.7 %) yielded null effects. The majority of the highest-powered studies found at least one null relationship (≥ 80.0 % of studies for each psychopathy measure, Fig. 3). Negative relationships with total psychopathy (25.0 %) were slightly more common within the highest-powered studies than in the full review across studies. Positive relationships with total psychopathy

Table 1
Studies that analyzed task-based activity in the vmPFC in relation to psychopathy.

Study	Pop.	N	Assessment	Task	Relationship Between vmPFC Activity and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Contreras-Rodríguez et al., 2014)	F	44	PCL-R	Prototypical facial expressions	0	6	0	0	3	0	0	3	0
(Decety et al., 2014)	F	70	PCL-R	Prototypical facial expressions	4	0	0	4	0	0	2	2	0
(Deeley et al., 2006)	F	24	PCL-R	Prototypical facial expressions	0	2	0						
(Dolan and Fullam, 2009)	F	24	PCL:SV	Prototypical facial expressions	0	10	0	0	10	0	3	6	1
(Gordon et al., 2004)	C	20	PPI	Prototypical facial expressions	0	3	0	0	3	0	0	3	0
(Pardini, 2010)	C	42	SRP	Prototypical facial expressions				0	2	0	0	1	0
(Sethi et al., 2018)	C	232	SRP-SF	Prototypical facial expressions				1	3	0			
(Szabó et al., 2017)	C	41	ICU	Prototypical facial expressions	0	3	0						
(Tully et al., 2023)	F	58	PCL-R	Prototypical facial expressions	0	4	0						
(Mier et al., 2014)	F	29	PCL-R	Prototypical facial expressions, empathy	0	4	0						
(Decety et al., 2013b)	F	70	PCL-R	Prototypical facial expressions, empathy	2	0	0	1	1	0	1	1	0
(Sun et al., 2022)	F	58	PCL-R, LSRP	Prototypical facial expressions, vocal expressions	1	9	0	0	5	0			
(Fede et al., 2016)	F	235	PCL-R	Moral judgment	0	6	0	0	2	0	0	2	0
(Glenn et al., 2009)	C	17	PCL-R	Moral judgment	0	1	0	1	1	0	0	2	0
(Harenski et al., 2010)	F	72	PCL-R	Moral judgment	0	5	0	0	2	0	1	1	0
(Harenski et al., 2014)	F	157	PCL-R	Moral judgment	0	2	0	0	2	0	0	2	0
(Marsh and Cardinale, 2014)	C	33	PPI-R	Moral judgment	0	6	0						
(Yoder et al., 2015a)	F	88	PCL-R	Moral judgment	0	2	0						
(Zijlmans et al., 2018)	F	100	YPI-SV	Moral judgment	0	0	1	0	1	1	0	1	0
(Seara-Cardoso et al., 2016a)	C	56	SRP-SF	Moral judgment				0	8	0	0	7	1
(Ueltzhöffer et al., 2023)	C	52	SD3	Moral judgment	0	2	0						
(Caldwell et al., 2015)	F	311	PCL-R	Viewing moral images				0	1	0	0	1	0
(Cheng et al., 2021)	C	56	PPI-R	Viewing moral images	0	1	0						
(Harenski et al., 2009)	C	10	PPI	Viewing moral images, emotion regulation	0	5	0						
(Pujol et al., 2012)	F	44	PCL-R	Viewing moral dilemmas, Stroop	0	2	0						
(Decety et al., 2013a)	F	121	PCL-R	Empathy	1	1	0	0	1	0	1	0	0
(Decety et al., 2015)	F	155	PCL-R	Empathy	0	8	0	0	4	0	0	4	0
(Deming et al., 2020)	F	94	PCL-R	Empathy	0	10	0	0	4	0	0	3	0
(Meffert et al., 2013)	F	54	PCL-R	Empathy	1	3	0						
(Yoder et al., 2021)	F	107	PCL-R	Empathy	0	4	0	0	4	0	0	4	0
(Veit et al., 2010)	F	9	PCL:SV, LSRP	Empathy	0	1	0	0	2	0	1	1	0
(Geurts et al., 2016)	F	34	PCL-R	Reward anticipation	0	2	0						
(Bjork et al., 2012)	C	31	PPI	Reward processing	0	2	1	0	3	0	0	3	0
(Brislin et al., 2022)	C	158	NEO-TRI	Reward processing				0	2	0	0	2	0
(Pujara et al., 2014)	F	41	PCL-R	Reward processing	0	1	0						
(Gregory et al., 2015)	F	32	PCL-R	Reward/punishment processing	0	4	0						
(Birbaumer et al., 2005)	F	20	PCL-R	Fear conditioning	1	3	0						
(Schneider et al., 2000)	F	24	PCL-R	Fear conditioning	0	4	0						
(Schultz et al., 2016)	F	50	PCL-R	Fear conditioning	0	1	0						
(Veit et al., 2002)	F	15	PCL-R	Fear conditioning	0	3	0						
(Larson et al., 2013)	F	49	PCL-R	Fear conditioning, attention	0	1	0						
(Geurts et al., 2022)	F	33	PCL-R	Aversive conditioning	0	1	0						
(Fullam et al., 2009)	C	24	PPI	Deception	0	1	0	1	2	0	0	4	0
(Glenn et al., 2017)	C	16	PCL-R	Deception	0	10	0	0	20	0	0	20	0
(Abe et al., 2018)	F	43	PCL-R	Deception	0	5	0						
(Shao and Lee, 2017)	C	52	PPI-R	Deception	0	3	0						
(Anderson et al., 2017)	F	120	PCL-R	Viewing emotional images	0	3	1	0	3	1	0	3	1
(Shane and Groat, 2018)	F	67	PCL-R	Viewing emotional images	4	4	1						
(Muller et al., 2003)	F	12	PCL-R	Viewing emotional images	0	2	0						
(Kiehl et al., 2001)	F	16	PCL-R	Remembering emotional words	0	6	0						
(Sadeh et al., 2013)	C	49	NEO-FFI	Emotion word Stroop				0	2	0	0	1	1
(Volman et al., 2016)	F	34	PCL-R	Emotional control	0	1	0						
(Müller et al., 2008b)	F	22	PCL-R	Discriminating cues after emotion induction	0	2	0						
(Nummenmaa et al., 2021)	F, C	38, 100	PCL-R, LSRP	Viewing violent scenes	0	0	2	0	0	1	0	0	1
(Yoder et al., 2015b)	C	43	PPI-R	Viewing violent scenes				0	1	0	0	1	0
(Deming et al., 2018)	F	57	PCL-R	Self/other processing	0	3	0	0	3	0	0	3	0
(Overgaauw et al., 2020)	C	38	PPI-SF	Self/other processing	0	4	0						
(Cope et al., 2014)	F	137	PCL-R	Viewing drug images	0	1	0	0	1	0	0	1	0
(Denomme et al., 2018)	C	105	PCL-R	Viewing drug images	0	2	0	0	1	0	0	1	0
(Rilling et al., 2007)	C	30	PPI, LSRP	Social cooperation	1	10	0	0	6	0	1	6	0
(Osumi et al., 2012)	C	20	LSRP	Frustration	0	1	0						

(continued on next page)

Table 1 (*continued*)

Study	Pop.	N	Assessment	Task	Relationship Between vmPFC Activity and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(da Cunha-Bang et al., 2017)	F	44	PCL-R	Aggression	0	1	0						
(Vieira et al., 2014)	C	35	TriPM	Cognitive load	0	2	0						
(Rodman et al., 2016)	F	46	PCL-R	Cognitive control	0	2	0						
(Vanova et al., 2022)	C	22	TriPM	Lexical decision-making	0	8	0						
(N. E. Anderson et al., 2018)	F	168	PCL-R	Salience processing	0	4	0	0	4	0	0	4	0
(Sheng et al., 2010)	C	19	PPI-R	Speech				0	3	0	1	3	0
(Sommer et al., 2010)	F	28	PCL-R	Theory of mind	0	3	1						
Total					**15**	**200**	**7**	**8**	**110**	**3**	**11**	**96**	**5**
Percent					**7 %**	**90 %**	**3 %**	**7 %**	**91 %**	**2 %**	**10 %**	**86 %**	**4 %**

Note: Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: - = negative relationship, + = positive relationship, vmPFC = ventromedial prefrontal cortex, Pop. = population, F = Forensic, C = Community, ICU = Inventory of Callous-Unemotional Traits, LSRP = Levenson Self-Report Psychopathy Scale, NEO-FFI = NEO Five Factor Inventory, NEO-Tri = NEO Triarchic Scale, PCL-R = Psychopathy Checklist-Revised, PCL:SV = Psychopathy Checklist: Screening Version, PPI = Psychopathic Personality Inventory, PPI-R = Psychopathic Personality Inventory-Revised, PPI-SF = Psychopathic Personality Inventory-Short Form, SD3 = Short Dark Triad, SRP = Self-Report Psychopathy Scale, SRP-SF = Self-Report Psychopathy Scale-Short Form, TriPM = Triarchic Psychopathy Measure, YPI-SV = Youth Psychopathy Inventory-Short Version.

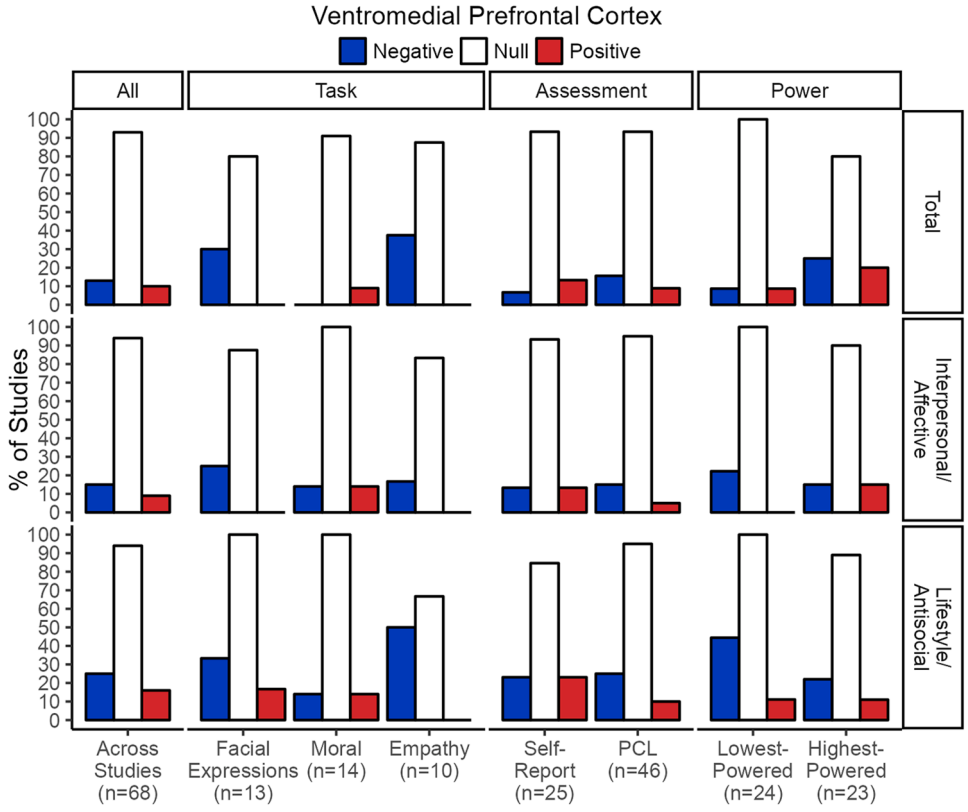

Fig. 3. Summary of studies that analyzed task-based activity in the ventromedial prefrontal cortex in relation to psychopathy. Cumulative percentages may exceed 100 % because many studies reported a mixture of findings (e.g., null and negative). Abbreviations: n = total number of studies included in examination.

(20.0 %) and interpersonal/affective traits (15.0 %) were also slightly more common within the highest-powered studies than in the full review. Few of the highest-powered studies found a positive relationship between vmPFC activity and lifestyle/antisocial traits (11.1 %).

3.2. Structural studies of ventromedial prefrontal cortex

3.2.1. Results across all studies

A total of 23 studies reported 98 total tests of gray matter structure in the vmPFC in relation to psychopathy (Table 3). Most studies (17/23) examined gray matter volume (GMV) in the vmPFC, six examined cortical thickness, three examined gray matter density (GMD), one examined gray matter concentration (GMC), and one examined cortical gyrification, with four studies examining more than one measure of gray matter structure. The majority of tests (75.5 %) yielded null effects. The majority of studies observed at least one null relationship (≥ 81.8 % of studies for each psychopathy measure; Fig. 5). Nearly one third of studies found a negative relationship with total psychopathy (31.8 %). Fewer studies found a negative relationship with interpersonal/affective traits (15.4 %), and no studies found a negative relationship with lifestyle/antisocial traits (0.0 %). Few studies of vmPFC gray matter structure observed a positive relationship (≤ 15.4 % for each

Table 2
Overlap between ventromedial prefrontal cortex and peak coordinates associated with total psychopathy.

H	Cluster label in original paper	MNI Coordinates	N	Ventromedial Prefrontal Cortex Overlap (%)			
				Whole	Area 11m	Area 13	Area 14m
Negative findings (Reduced activity)							
R	ventromedial orbitofrontal cortex	15, 24, −12	20	18.7	0.0	18.7	0.0
R	ventromedial prefrontal cortex	2, 16, −28	70	13.2	0.0	13.2	0.0
R	ventromedial prefrontal cortex	8, 30, −10	70	42.9	0.0	3.1	39.8
R	orbitofrontal cortex	14, 58, −2	121	23.1	0.4	0.0	22.7
R	ventromedial prefrontal cortex	8, 65, −10	70	**97.1**	**94.6**	0.0	2.5
R	medial orbitofrontal cortex	18, 20, −18	70	28.3	0.0	28.3	0.0
L	medial orbitofrontal cortex	−15, 32, −22	70	37.5	11.5	26.0	0.0
R	ventromedial prefrontal cortex	10, 18, −22	70	**87.2**	0.0	**87.2**	0.0
R	ventromedial prefrontal cortex	10, 68, −15	70	**55.2**	**55.2**	0.0	0.0
R	ventromedial prefrontal cortex	8, 30, −10	70	42.9	0.0	3.1	39.8
R	medial orbitofrontal cortex	10, 38, −8	70	**51.6**	0.0	0.0	**51.6**
L	medial orbitofrontal cortex	−12, 42, −10	70	45.1	0.0	0.9	44.3
L	mid orbital gyrus	−15, 63, −3	54	11.3	6.9	0.0	4.4
R	orbital/superior frontal gyrus	21, 57, −6	67	0.0	0.0	0.0	0.0
Positive findings (Increased activity)							
L	frontal mid orbital	−9, 36, −15	120	**55.5**	10.8	32.6	12.1
R	orbitofrontal cortex	1, 46, 2	38	11.6	0.0	0.0	11.6
R	medial frontal cortex	15, 48, −3	67	40.8	0.0	0.0	40.8
R	medial frontal cortex	12, 36, −15	67	35.1	1.3	13.6	20.1
L	medial frontal cortex	−12, 24, −12	67	27.7	0.0	27.7	0.0
L	medial frontal cortex	−18, 57, 6	67	0.0	0.0	0.0	0.0
L	orbitofrontal cortex	−2, 44, −20	28	**97.2**	**69.9**	0.0	27.3
L	ventromedial prefrontal cortex	−4, 54, −5	100	**81.9**	0.0	0.0	**81.9**

Peak coordinates that overlapped ≥ 50.0 % with the ventromedial prefrontal cortex mask are in bold.
Abbreviations: H = hemisphere, L = left, R = right

psychopathy measure).

3.2.2. Results within self-report and PCL assessments

Five studies reported 19 total tests of vmPFC gray matter structure in relation to a self-report psychopathy assessment. The majority of tests (89.5 %) yielded null effects. All studies that examined total psychopathy (100.0 %) or lifestyle/antisocial traits (100.0 %) observed at least one null relationship with gray matter structure (Fig. 5). One study observed a null relationship with interpersonal/affective traits (50.0 %), while one study observed a negative relationship with interpersonal/affective traits (50.0 %). No study found a positive relationship (0.0 % for each psychopathy measure).

A total of 19 studies reported 79 total tests of vmPFC gray matter structure in relation to a PCL assessment. Results largely paralleled the results from the full review. The majority of tests (72.2 %) yielded null effects. Most studies that used PCL assessments found at least one null relationship (≥ 77.8 % of studies for each psychopathy measure; Fig. 5). Additionally, over one third of these studies found a negative relationship with total psychopathy (38.9 %).

3.2.3. Results of lowest- and highest-powered studies

The lowest-powered studies consisted of eight studies that reported 25 total tests of vmPFC gray matter structure. The studies included in the full review had a mean sample size of $N = 88.6$ (range: 26–716), whereas the lowest-powered studies had a mean sample size of $N = 34.8$ (range: 26–39). The results of the lowest-powered studies paralleled the results of the full review. The majority of tests (84.0 %) yielded null effects. Most studies observed at least one null relationship (≥ 75.0 % of studies for each psychopathy measure; Fig. 5).

The highest-powered studies consisted of eight studies that reported 54 total tests of vmPFC gray matter structure, with a mean sample size of $N = 182.0$ (range: 66–716). Results also paralleled the results of the full review, with the exception that the highest-powered studies were less likely to find a negative relationship between vmPFC gray matter structure and total psychopathy (12.5 % of studies; Fig. 5). The majority of tests (83.3 %) yielded null effects.

3.3. Functional studies of anterior cingulate cortex

3.3.1. Results across all studies

A total of 66 studies reported 434 total tests of task-based BOLD activity in the ACC in relation to a measure of psychopathy (Table 4). The majority of these tests (83.2 %) yielded null effects. The majority of studies found at least one null relationship (≥ 86.7 % of studies for each psychopathy measure; Fig. 6). One quarter of studies found a negative relationship with total psychopathy (27.6 %), while slightly fewer found a negative relationship with interpersonal/affective traits (21.2 %) or lifestyle/antisocial traits (23.3 %). Less than one fifth of studies of ACC activity observed a positive relationship (≤ 18.2 % for each psychopathy measure).

3.3.2. Peak coordinates of results across all studies

We extracted 38 peak coordinates of significant ACC clusters related to total psychopathy from 15 studies (Table 5, Fig. 7). Of the 21 peak coordinates negatively related to total psychopathy, eight overlapped ≥ 50.0 % with the ACC mask, eight overlapped 0.1–49.9 %, and five overlapped 0.0 % with the ACC mask. Three of these peak coordinates overlapped ≥ 50.0 % with area 32p and one overlapped ≥ 50.0 % with area 32sg, while the remaining peak coordinates did not overlap ≥ 50.0 % with any ACC subregion. Overall, the peak coordinates negatively related to psychopathy were scattered across medial frontal cortex. Of the 17 peak coordinates positively related to total psychopathy, only four overlapped ≥ 50.0 % with the ACC mask, eight overlapped 0.1–49.9 %, and five overlapped 0.0 % with the ACC mask. The peak coordinates positively related to total psychopathy were similarly scattered across medial frontal cortex, rather than localized to any ACC subregion. Additionally, we extracted 28 peak coordinates of significant ACC clusters related to interpersonal/affective traits and lifestyle/antisocial traits from 16 studies. Patterns for these peak coordinates resembled those for the peak coordinates related to total psychopathy (see Supplemental Materials).

3.3.3. Results within tasks

Twelve studies reported 95 total tests of ACC activity in prototypical facial expressions tasks, ten studies reported 57 total tests in moral tasks,

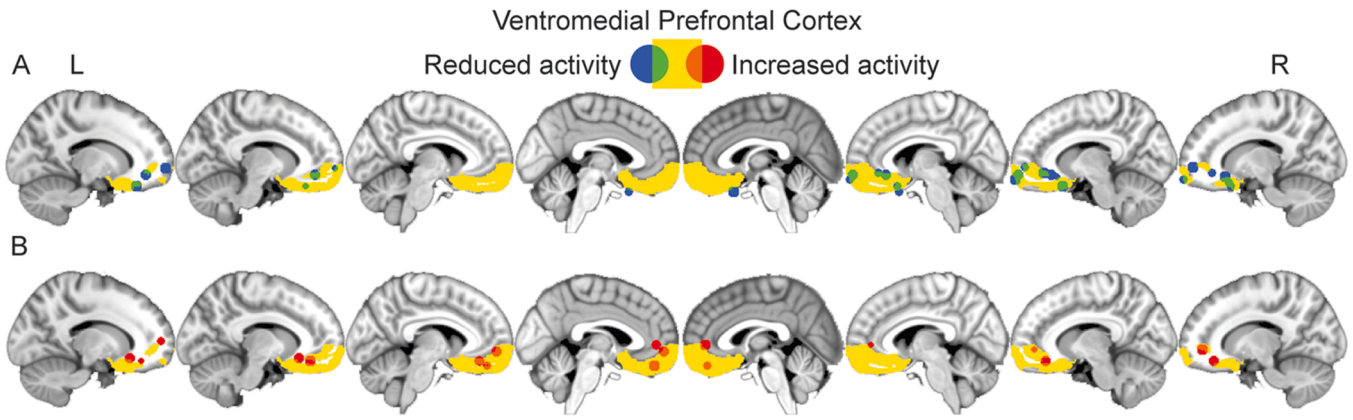

Fig. 4. Peak coordinates of ventromedial prefrontal cortex clusters that were A) negatively related and B) positively related to total psychopathy. In each panel, sagittal slices are displayed from left to right at x = −14, −10, −6, −2, 2, 6, 10, 14.

Table 3
Studies that analyzed vmPFC structure in relation to psychopathy.

Study	Pop.	N	Assessment	Structural Measure	Relationship Between vmPFC Structure and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Beckwith et al., 2018)	C	155	PPI	GMV	0	1	0						
(Bertsch et al., 2013)	F	39	PCL-R	GMV	0	4	0						
(Cantor et al., 2015)	F	56	LSRP	GMV	0	1	0						
(Cope et al., 2012)	F	66	PCL-R	GMV	0	2	0	0	1	1	0	2	0
(Gregory et al., 2012)	F	44	PCL-R	GMV	3	0	0						
(Hofhansel et al., 2020)	F	26	PCL-R	GMV	0	1	0	0	1	0	0	1	0
(Korponay et al., 2017)	F	124	PCL-R	GMV	0	3	1	0	4	0	0	3	3
(Laakso et al., 2002)	F	57	PCL-R	GMV	1	1	0	0	1	0	0	1	0
(Leutgeb et al., 2015)	F	40	PCL-R	GMV				0	1	0	0	0	2
(Müller et al., 2008a)	F	34	PCL-R	GMV	0	2	0	0	1	0	0	1	0
(Pera-Guardiola et al., 2016)	F	39	PCL-R	GMV	0	1	0	0	2	0	0	2	0
(Tiihonen et al., 2008)	F	37	PCL-R	GMV	2	0	0						
(Vieira et al., 2015)	C	35	PPI-R, TriPM	GMV	0	2	0						
(Chester et al., 2023)	C	97	SRP-SF	GMV, CT	0	4	0	0	4	0	0	4	0
(Lam et al., 2017)	F	67	PCL-R	GMV, CT	0	2	0	0	2	0	0	2	0
(Yang et al., 2010)	C	53	PCL-R	GMV, CT	2	2	0						
(Calzada-Reyes et al., 2021)	F	132	PCL-R	GMV, GMD, CT	0	3	0	0	2	0	0	2	0
(Kolla et al., 2017)	F	38	PCL-R	CT	0	1	0						
(Ly et al., 2012)	F	52	PCL-R	CT	0	1	0						
(de de de Oliveira-Souza et al., 2008)	C	30	PCL:SV	GMC	1	1	0	1	0	0	0	1	0
(Boccardi et al., 2011)	F	51	PCL-R	GMD	3	0	0						
(Nummenmaa et al., 2021)	F, C	38, 100	PCL-R, LSRP	GMD	2	0	0	2	0	0	0	1	0
(Miskovich et al., 2018)	F	716	PCL-R	Cortical gyrification	0	1	0	0	1	0	0	1	0
Total					**14**	**33**	**1**	**3**	**20**	**1**	**0**	**21**	**5**
Percent					**29 %**	**69 %**	**2 %**	**13 %**	**83 %**	**4 %**	**0 %**	**81 %**	**19 %**

Note: Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: - = negative relationship, + = positive relationship, vmPFC = ventromedial prefrontal cortex, Pop. = population, F = Forensic, C = Community, GMV = gray matter volume, CT = cortical thickness, GMD = gray matter density, GMC = gray matter concentration, LSRP = Levenson Self-Report Psychopathy Scale, PCL-R = Psychopathy Checklist-Revised, PCL:SV = Psychopathy Checklist: Screening Version, PPI = Psychopathic Personality Inventory, PPI-R = Psychopathic Personality Inventory-Revised, SRP-SF = Self-Report Psychopathy Scale-Short Form, TriPM = Triarchic Psychopathy Measure.

and nine studies reported 75 total tests in empathy tasks. The results within the most common experimental tasks tended to reflect the results of the full review across studies of ACC activity, with the exception that studies that employed an empathy task were less likely to observe null relationships and more likely to observe significant negative or positive relationships with total psychopathy. The majority of tests (≥ 61.3 % for each task category) yielded null effects. For each task, the majority of studies of ACC activity found at least one null relationship (≥ 66.7 % of studies for each psychopathy measure for each task category; Fig. 6). Within empathy tasks, nearly half of studies found a negative (44.4 %) or positive relationship (44.4 %) with total psychopathy. Within moral tasks, two studies (40.0 %) observed a negative relationship between ACC activity and lifestyle/antisocial traits. For each of these tasks, results of specific contrasts are in Supplemental Materials.

3.3.4. *Results within self-report and PCL assessments*

A total of 23 studies reported 113 total tests of ACC activity in relation to a self-report psychopathy assessment. Results largely paralleled the results from the full review across studies. The majority of tests (86.7 %) yielded null effects. The majority of studies observed at least one null relationship (≥ 81.8 % of studies for each psychopathy measure; Fig. 6). Few of the studies that measured psychopathy via self-report observed a significant negative relationship (≤ 20.0 % for each psychopathy measure) or positive relationship (≤ 27.3 % for each

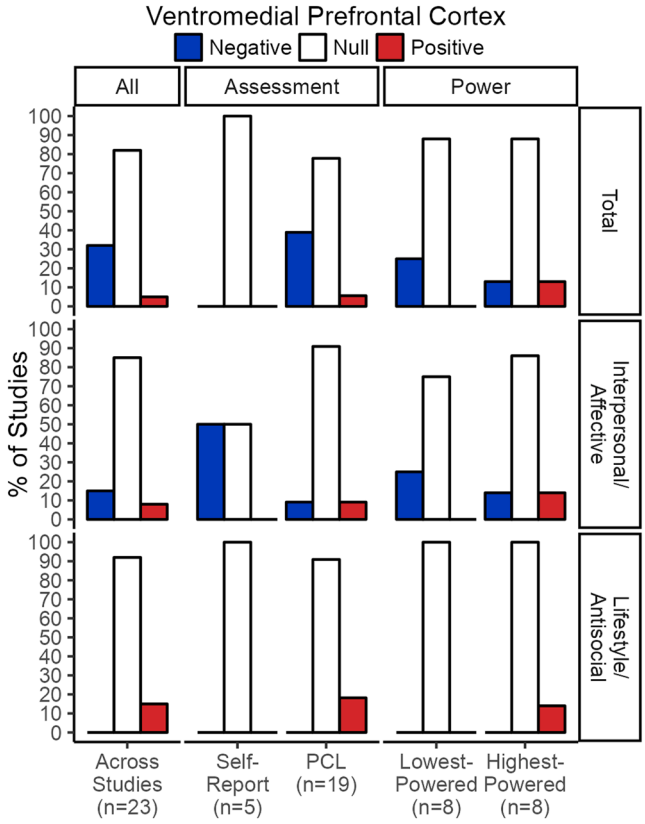

Fig. 5. Summary of studies that analyzed gray matter structure in the ventromedial prefrontal cortex in relation to psychopathy. Cumulative percentages may exceed 100 % because many studies reported a mixture of findings (e.g., null and negative). Abbreviations: n = total number of studies included in examination.

psychopathy measure).

A total of 45 studies reported 321 total tests of ACC activity in relation to a PCL assessment. These results also paralleled the results from the full review across studies, with the exception that these studies were slightly more likely to observe a negative relationship with lifestyle/antisocial traits. The majority of tests (81.9 %) yielded null effects. The majority of studies observed at least one null relationship (≥ 88.4 % of studies for each psychopathy measure; Fig. 6). One third of studies that measured psychopathy via PCL found a negative relationship between ACC activity and lifestyle/antisocial traits (36.8 %), while less than one third found a negative relationship with total psychopathy (30.2 %) or interpersonal/affective traits (25.0 %).

3.3.5. Results of lowest- and highest-powered studies

The lowest-powered studies consisted of 23 studies that reported 176 total tests of ACC activity. The studies included in the full review had a mean sample size of $N = 62.6$ (range: 12–311), whereas the lowest-powered studies had a mean sample size of $N = 24.2$ (range: 12–34). The results of the lowest-powered studies largely reflected the results of the full review. The majority of tests (90.9 %) yielded null effects. All of the lowest-powered studies of ACC activity found at least one null relationship (100.0 % of studies for each psychopathy measure; Fig. 6). The lowest-powered studies were slightly less likely to find a negative relationship (≤ 18.2 % for each psychopathy measure) or positive relationship (≤ 14.3 % for each psychopathy measure) than in the full review.

The highest-powered studies consisted of 23 studies that reported 201 total tests of ACC activity, with a mean sample size of $N = 117.0$ (range: 56–311). In contrast to the full review and to the review of the lowest-powered studies, the highest-powered studies were less likely to observe null relationships and more likely to observe significant negative or positive relationships between ACC activity and psychopathy. Yet, the majority of tests (77.1 %) yielded null effects. The majority of the highest-powered studies found at least one null relationship (≥ 80.0 % of studies for each psychopathy measure; Fig. 6). Negative relationships were the next most common finding for total psychopathy (40.0 %) and lifestyle/antisocial traits (29.4 %). The highest-powered studies were equally likely to observe negative and positive relationships with interpersonal/affective traits (26.3 %). Many of the highest-powered studies observed a positive relationship with total psychopathy (35.0 %), while few observed a positive relationship with lifestyle/antisocial traits (11.8 %).

3.4. Structural studies of anterior cingulate cortex

3.4.1. Results across all studies

A total of 20 studies reported 89 total tests of gray matter structure in the ACC in relation to psychopathy (Table 6). Most studies (14/20) examined GMV in the ACC, four examined cortical thickness, three examined GMD, one examined GMC, and one examined cortical gyrification, with two studies examining more than one measure of gray matter structure. The majority of tests (87.6 %) yielded null effects. The majority of studies of ACC gray matter structure found at least one null relationship (≥ 85.0 % of studies for each psychopathy measure; Fig. 8). One fifth of studies found a negative relationship with total psychopathy (20.0 %), while fewer studies found a relationship with interpersonal/affective traits (9.1 %) or lifestyle/antisocial traits (0.0 %). Nearly one fifth of studies found a positive relationship with lifestyle/antisocial traits (18.2 %), while fewer studies found a positive relationship with total psychopathy (10.0 %) or interpersonal/affective traits (9.1 %).

3.4.2. Results within self-report and PCL assessments

Five studies reported 18 total tests of ACC gray matter structure in relation to a self-report psychopathy assessment. The majority of tests (88.9 %) yielded null effects. All studies that examined total psychopathy (100.0 %) or lifestyle/antisocial traits (100.0 %) observed at least one null relationship (Fig. 8). One study that used self-report assessments found a null relationship (50.0 %), one found a negative relationship (50.0 %), and one found a positive relationship (50.0 %) with interpersonal/affective traits. No study that used self-report assessments found a significant negative or positive relationship with total psychopathy (0.0 %) or lifestyle/antisocial traits (0.0 %).

A total of 16 studies reported 71 total tests of ACC gray matter structure in relation to a PCL assessment. Results paralleled the results from the full review. The majority of tests (87.3 %) yielded null effects. The majority of studies found at least one null relationship (≥ 81.3 % of studies for each psychopathy measure; Fig. 8). One quarter of studies that used PCL assessments found a negative relationship between ACC structure and total psychopathy (25.0 %), while nearly one quarter found a positive relationship with lifestyle/antisocial traits (22.2 %).

3.4.3. Results of lowest- and highest-powered studies

The lowest-powered studies consisted of seven studies that reported 23 total tests of ACC gray matter structure. The studies included in the full review had a mean sample size of $N = 95.0$ (range: 26–716), whereas the lowest-powered studies had a mean sample size of $N = 34.3$ (range: 26–39). All tests (100.0 %) yielded null effects. All of the lowest-powered studies of ACC gray matter structure observed a null relationship (100.0 % of studies for each psychopathy measure; Fig. 8).

The highest-powered studies consisted of seven studies that reported 52 total tests of ACC gray matter structure, with a mean sample size of $N = 199.0$ (range: 72–716). The results of the highest-powered studies largely reflected the results of the full review. The majority of tests (86.5 %) yielded null effects. Most studies observed at least one null relationship (≥ 83.3 % of studies for each psychopathy measure; Fig. 8).

Table 4
Studies that analyzed task-based activity in the ACC in relation to psychopathy.

Study	Pop.	N	Assessment	Task	Relationship Between ACC Activity and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Contreras-Rodríguez et al., 2014)	F	44	PCL-R	Prototypical facial expressions	0	6	0	0	3	0	0	3	0
(Decety et al., 2014)	F	70	PCL-R	Prototypical facial expressions	0	3	1	0	3	1	0	4	0
(Deeley et al., 2006)	F	24	PCL-R	Prototypical facial expressions	0	2	0						
(Dolan and Fullam, 2009)	F	24	PCL:SV	Prototypical facial expressions	0	10	0	0	10	0	1	9	0
(Pardini, 2010)	C	42	SRP	Prototypical facial expressions				0	2	0	0	1	0
(Seara-Cardoso et al., 2016b)	C	30	SRP-SF	Prototypical facial expressions	0	1	0	0	1	0	0	1	0
(Sethi et al., 2018)	C	232	SRP-SF	Prototypical facial expressions				2	2	0			
(Szabó et al., 2017)	C	41	ICU	Prototypical facial expressions	1	2	0						
(Tully et al., 2023)	F	58	PCL-R	Prototypical facial expressions	1	3	0						
(Mier et al., 2014)	F	29	PCL-R	Prototypical facial expressions, empathy	0	4	0						
(Decety et al., 2013b)	F	70	PCL-R	Prototypical facial expressions, empathy	1	0	1	0	1	1	1	1	0
(Sun et al., 2022)	F	58	PCL-R, LSRP	Prototypical facial expressions, vocal expressions	8	12	0	1	4	0			
(Fede et al., 2016)	F	235	PCL-R	Moral judgment	1	5	0	0	2	0	0	2	0
(Harenski et al., 2010)	F	72	PCL-R	Moral judgment	0	5	0						
(Harenski et al., 2014)	F	157	PCL-R	Moral judgment	1	1	0	0	2	0	1	1	0
(Marsh and Cardinale, 2014)	C	33	PPI-R	Moral judgment	0	6	0						
(Yoder et al., 2015a)	F	88	PCL-R	Moral judgment	2	0	2	0	1	1	1	1	0
(Seara-Cardoso et al., 2016a)	C	56	SRP-SF	Moral judgment				0	8	0	0	8	0
(Ueltzhöffer et al., 2023)	C	52	SD3	Moral judgment	0	2	0						
(Caldwell et al., 2015)	F	311	PCL-R	Viewing moral images				1	0	0	0	1	0
(Cheng et al., 2021)	C	56	PPI-R	Viewing moral images	0	1	0						
(Pujol et al., 2012)	F	44	PCL-R	Viewing moral dilemmas, Stroop	0	2	0						
(Decety et al., 2013a)	F	121	PCL-R	Empathy	1	0	1	0	1	1	1	0	1
(Deming et al., 2020)	F	94	PCL-R	Empathy	0	10	0	0	4	0	0	3	0
(Decety et al., 2015)	F	155	PCL-R	Empathy	1	3	4	1	3	0	0	4	0
(Meffert et al., 2013)	F	54	PCL-R	Empathy	1	2	2						
(Molenberghs et al., 2014)	C	48	SRP	Empathy	1	0	0						
(Seara-Cardoso et al., 2015)	C	46	SRP-SF	Empathy	0	1	0	0	1	0	0	0	1
(Yoder et al., 2021)	F	107	PCL-R	Empathy	0	4	0	0	4	0	1	3	0
(Geurts et al., 2016)	F	34	PCL-R	Reward anticipation	0	2	0						
(Bjork et al., 2012)	C	31	PPI	Reward processing	0	1	2	0	2	1	0	1	2
(Pujara et al., 2014)	F	41	PCL-R	Reward processing	0	1	0						
(Birbaumer et al., 2005)	F	20	PCL-R	Fear conditioning	1	3	0						
(Schneider et al., 2000)	F	24	PCL-R	Fear conditioning	0	4	0						
(Schultz et al., 2016)	F	50	PCL-R	Fear conditioning	0	1	0						
(Veit et al., 2002)	F	15	PCL-R	Fear conditioning	0	3	0						
(Larson et al., 2013)	F	49	PCL-R	Fear conditioning, attention	0	1	0						
(Geurts et al., 2022)	F	33	PCL-R	Aversive conditioning	0	1	0						
(Fullam et al., 2009)	C	24	PPI	Deception	0	1	0	0	3	0	0	4	0
(Glenn et al., 2017)	C	16	PCL-R	Deception	0	10	0	0	20	0	0	20	0
(Abe et al., 2018)	F	43	PCL-R	Deception	2	0	0	1	0	0	1	0	0
(Shao and Lee, 2017)	C	52	PPI-R	Deception	0	3	0						
(Shane and Groat, 2018)	F	67	PCL-R	Viewing emotional images	0	6	2						
(Anderson et al., 2017)	F	120	PCL-R	Viewing emotional images	0	4	0	0	4	0	0	4	0
(Muller et al., 2003)	F	12	PCL-R	Viewing emotional images	1	1	0						
(Kiehl et al., 2001)	F	16	PCL-R	Remembering emotional words	6	1	0						
(Sadeh et al., 2013)	C	49	NEO-FFI	Emotion word Stroop				0	2	0	0	1	0
(Volman et al., 2016)	F	34	PCL-R	Emotional control	0	1	0						
(Müller et al., 2008b)	F	22	PCL-R	Discriminating cues after emotion induction	0	2	0						
(Nummenmaa et al., 2021)	F, C	38, 100	PCL-R, LSRP	Viewing violent scenes	0	0	1	0	0	1	0	0	1
(Yoder et al., 2015b)	C	43	PPI-R	Viewing violent scenes				0	1	0	0	1	0
(Deming et al., 2018)	F	57	PCL-R	Self/other processing	0	3	0	0	3	0	0	3	0
(Overgaauw et al., 2020)	C	38	PPI-SF	Self/other processing	0	4	0						
(Nuñez et al., 2005)	C	20	PPI	Self processing				0	6	0	0	8	0
(Cope et al., 2014)	F	137	PCL-R	Viewing drug images	0	1	0	0	1	0	0	1	0
(Denomme et al., 2018)	C	105	PCL-R	Viewing drug images	0	2	0	0	1	0	0	1	0
(Rilling et al., 2007)	C	30	PPI, LSRP	Social cooperation	1	4	1	0	2	0	0	2	0
(Osumi et al., 2012)	C	20	LSRP	Frustration	0	1	0						
(da Cunha-Bang et al., 2017)	F	44	PCL-R	Aggression	0	1	0						
(Rodman et al., 2016)	F	46	PCL-R	Cognitive control	0	2	0						
(Vieira et al., 2014)	C	35	TriPM	Cognitive load	0	2	0						
(Vanova et al., 2022)	C	22	TriPM	Lexical decision-making	0	8	0						

(*continued on next page*)

Table 4 (*continued*)

Study	Pop.	N	Assessment	Task	Relationship Between ACC Activity and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Kiehl et al., 2004)	F	16	PCL-R	Reading concrete and abstract words	0	1	0						
(N. E. Anderson et al., 2018)	F	168	PCL-R	Salience processing	0	4	0	1	3	0	0	4	0
(Schiffer et al., 2014)	F	44	PCL-R	Stroop				1	1	0			
(Sommer et al., 2010)	F	28	PCL-R	Theory of mind	0	4	0						
Total					**30**	**168**	**17**	**8**	**101**	**6**	**7**	**92**	**5**
Percent					**14 %**	**78 %**	**8 %**	**7 %**	**88 %**	**5 %**	**7 %**	**88 %**	**5 %**

Note: Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: - = negative relationship, + = positive relationship, ACC = anterior cingulate cortex, Pop. = population, F = Forensic, C = Community, ICU = Inventory of Callous-Unemotional Traits, LSRP = Levenson Self-Report Psychopathy Scale, NEO-FFI = NEO Five Factor Inventory, NEO-Tri = NEO Triarchic Scale, PCL-R = Psychopathy Checklist-Revised, PCL:SV = Psychopathy Checklist: Screening Version, PPI = Psychopathic Personality Inventory, PPI-R = Psychopathic Personality Inventory-Revised, PPI-SF = Psychopathic Personality Inventory-Short Form, SD3 = Short Dark Triad, SRP = Self-Report Psychopathy Scale, SRP-SF = Self-Report Psychopathy Scale-Short Form, TriPM = Triarchic Psychopathy Measure, YPI-SV = Youth Psychopathy Inventory-Short Version.

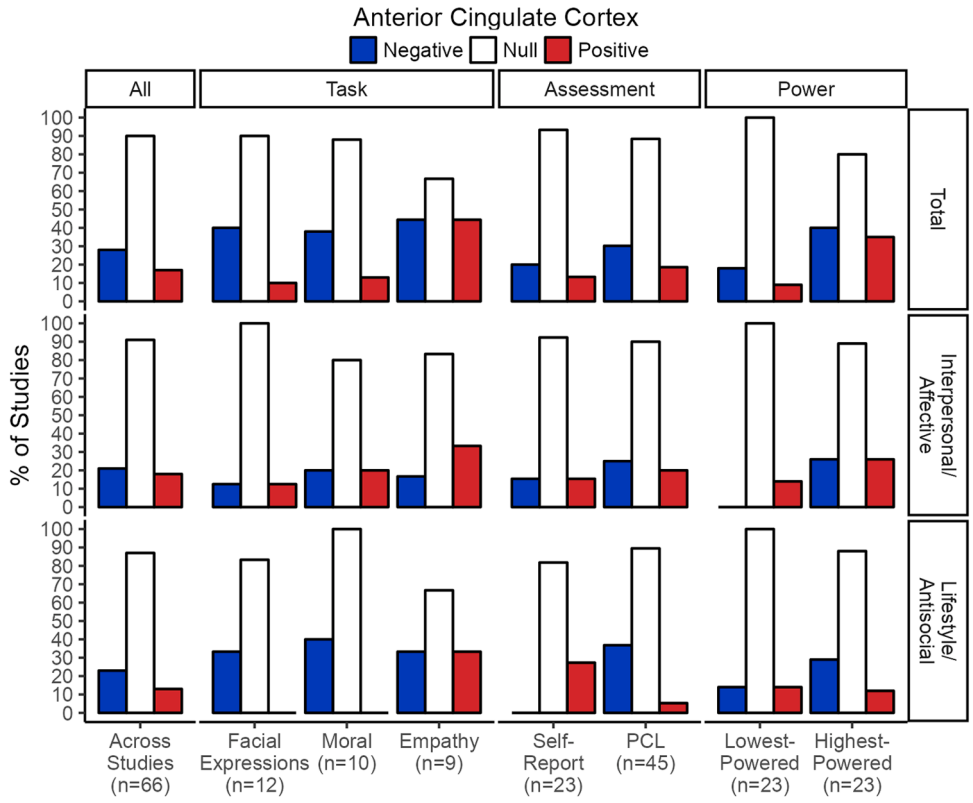

Fig. 6. Summary of studies that analyzed task-based activity in the anterior cingulate cortex in relation to psychopathy. Cumulative percentages may exceed 100 % because many studies reported a mixture of findings (e.g., null and negative). Abbreviations: n = total number of studies included in examination.

3.5. Functional studies of dorsomedial prefrontal cortex

3.5.1. Results across all studies

A total of 67 studies reported 433 total tests of task-based BOLD activity in the dmPFC in relation to psychopathy (Table 7). The majority of these tests (85.0 %) yielded null effects. The majority of studies of dmPFC activity found at least one null relationship (≥ 85.2 % of studies for each psychopathy measure; Fig. 9). One fifth of studies found a positive relationship between dmPFC activity and total psychopathy (21.7 %) and interpersonal/affective traits (20.7 %), although fewer studies found a positive relationship with lifestyle/antisocial traits (14.8 %). Nearly one quarter of studies found a negative relationship with interpersonal/affective traits (24.1 %), while fewer found a negative relationship with total psychopathy (16.7 %) or lifestyle/ antisocial traits (14.8 %).

3.5.2. Peak coordinates of results across all studies

We extracted 53 peak coordinates of significant dmPFC clusters related to total psychopathy from 16 studies (Table 8, Fig. 10). Of the 26 peak coordinates negatively related to total psychopathy, 11 overlapped ≥ 50.0 % with the dmPFC mask, 14 overlapped 0.1–49.9 %, and one overlapped 0.0 % with the dmPFC mask. The peak coordinates negatively related to total psychopathy were not localized to any dmPFC subregion. Of the 27 peak coordinates positively related to total psychopathy, 17 overlapped ≥ 50.0 % with the dmPFC mask, nine overlapped 0.1–49.9 %, and one overlapped 0.0 % with the dmPFC mask.

Table 5
Overlap between anterior cingulate cortex and peak coordinates associated with total psychopathy.

H	Cluster Label in Original Paper	MNI Coordinates	N	Anterior Cingulate Cortex Overlap (%)				
				Whole	Area 24rv	Area 24cd	Area 32p	Area 32sg
Negative findings (Reduced activity)								
L	anterior cingulate cortex	−2, 28, 20	43	**86.9**	15.0	0.0	**67.8**	4.1
L	anterior cingulate cortex	−4, 26, 22	43	**81.9**	14.9	0.0	**66.7**	0.3
R	rostral anterior cingulate	3, 30, 3	20	18.6	18.6	0.0	0.0	0.0
L	dorsal anterior cingulate cortex	−2, 35, 5	70	**62.4**	14.5	0.0	0.0	47.8
R	anterior midcingulate cortex	6, 18, 34	121	**79.4**	0.0	14.1	**65.4**	0.0
L	anterior cingulate cortex	−12, 28, 18	155	10.4	0.0	0.0	10.4	0.0
L/R	anterior cingulate cortex	0, 3, −9	235	0.0	0.0	0.0	0.0	0.0
R	rostral anterior cingulate cortex	6, 27, 18	157	**61.7**	38.2	0.0	16.1	7.4
L/R	rostral anterior cingulate cortex	0, 41, 9	16	**99.2**	1.7	0.0	12.1	**85.5**
L	caudal anterior cingulate cortex	−9, 23, 21	16	24.4	8.7	0.0	15.6	0.0
L	midcingulate gyrus	−12, 27, 30	54	25.1	0.0	0.0	25.1	0.0
L	midcingulate gyrus	−9, −18, 42	54	0.0	0.0	0.0	0.0	0.0
R	midcingulate gyrus	9, −21, 39	54	0.0	0.0	0.0	0.0	0.0
R	midcingulate gyrus	9, 9, 27	54	21.8	21.7	0.1	0.0	0.0
R	midcingulate gyrus	3, 15, 21	54	31.4	28.5	1.9	1.0	0.0
L	gyrus cinguli	−6, 37, 7	12	**70.1**	8.0	0.0	12.4	49.6
R	gyrus cinguli, subgenual cingulate	5, 29, 16	12	**68.3**	38.9	0.0	14.9	14.5
L	cingulate gyrus	−5–2 27	30	16.3	16.3	0.0	0.0	0.0
L	dorsal anterior cingulate	−18, 18, 38	88	0.0	0.0	0.0	0.0	0.0
L	midcingulate gyrus	−8, −15, 39	58	0.0	0.0	0.0	0.0	0.0
R	midcingulate gyrus	11, −5, 35	58	2.2	0.3	1.9	0.0	0.0
Positive findings (Increased activity)								
R	dorsal anterior cingulate cortex	4, 50, 16	70	14.1	0.0	0.0	0.7	13.4
L	dorsal anterior cingulate cortex	−8, 38, 26	70	37.3	0.0	0.0	37.3	0.0
R	anterior midcingulate cortex	2, 16, 28	70	**88.9**	34.1	34.5	20.3	0.0
L	anterior midcingulate cortex	−4, 8, 34	121	**82.0**	15.9	**66.2**	0.0	0.0
R	anterior midcingulate cortex	4, 10, 32	121	**78.6**	20.8	**56.0**	1.8	0.0
L	middle cingulate gyrus	−2, 0, 28	70	35.3	32.6	2.7	0.0	0.0
R	anterior cingulate cortex	12, 34, 14	155	23.2	7.9	0.0	5.1	10.2
R	anterior cingulate cortex	12, 34, 12	155	21.3	9.4	0.0	1.7	10.3
L	anterior cingulate cortex	−10, 28, 20	155	32.5	1.5	0.0	31.1	0.0
L	anterior cingulate cortex	−8, 26, 20	155	43.5	10.6	0.0	32.9	0.0
R	anterior cingulate gyrus	9, 48, 6	54	41.8	0.0	0.0	0.0	41.8
R	middle frontal gyrus, superior frontal gyrus, superior medial gyrus, anterior cingulate cortex	9, 30, 45	54	0.0	0.0	0.0	0.0	0.0
L	superior medial gyrus, anterior cingulate cortex	−9, 39, 33	54	0.0	0.0	0.0	0.0	0.0
R	superior medial gyrus, anterior cingulate cortex	9, 30, 48	54	0.0	0.0	0.0	0.0	0.0
L	anterior cingulate cortex	−18, 18, 18	67	0.0	0.0	0.0	0.0	0.0
R	anterior cingulate cortex	15, 18, 48	67	0.0	0.0	0.0	0.0	0.0
L	subgenual anterior cingulate cortex	−2, 26, −6	88	**73.5**	19.3	0.0	0.0	**54.2**

Peak coordinates that overlapped ≥ 50.0 % with the anterior cingulate mask are in bold.
Abbreviations: H = hemisphere, L = left, R = right

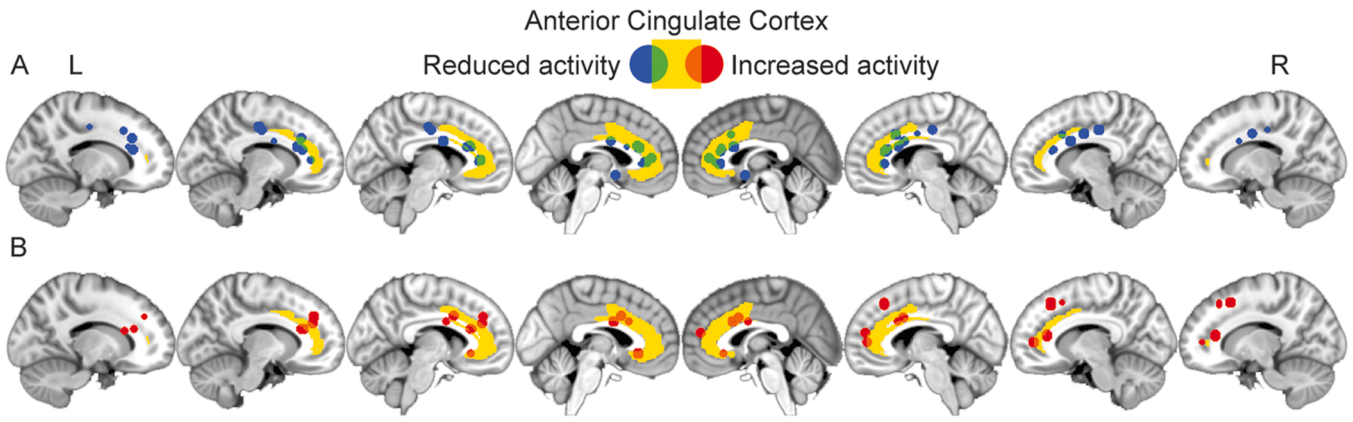

Fig. 7. Peak coordinates of anterior cingulate cortex clusters that were A) negatively related and B) positively related to total psychopathy. In each panel, sagittal slices are displayed from left to right at x = −14, −10, −6, −2, 2, 6, 10, 14.

Table 6
Studies that analyzed ACC structure in relation to psychopathy.

Study	Pop.	N	Assessment	Structural Measure	Relationship Between ACC Structure and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Beckwith et al., 2018)	C	155	PPI	GMV	0	1	0						
(Bertsch et al., 2013)	F	39	PCL-R	GMV	0	4	0						
(Cantor et al., 2015)	F	56	LSRP	GMV	0	1	0						
(Cope et al., 2012)	F	66	PCL-R	GMV	0	1	1	0	2	0	0	2	0
(Glenn et al., 2010)	C	72	PCL-R	GMV	0	6	0	0	3	0	0	3	0
(Gregory et al., 2012)	F	44	PCL-R	GMV	0	2	0						
(Hofhansel et al., 2020)	F	26	PCL-R	GMV	0	1	0	0	1	0	0	1	0
(Korponay et al., 2017)	F	124	PCL-R	GMV	0	4	0	0	4	0	0	5	1
(Müller et al., 2008a)	F	34	PCL-R	GMV	0	2	0	0	1	0	0	1	0
(Pera-Guardiola et al., 2016)	F	39	PCL-R	GMV	0	1	0	0	2	0	0	2	0
(Tiihonen et al., 2008)	F	37	PCL-R	GMV	0	1	0						
(Vieira et al., 2015)	C	35	PPI-R, TriPM	GMV	0	2	0						
(Chester et al., 2023)	C	97	SRP-SF	GMV, CT	0	4	0	0	3	1	0	4	0
(Calzada-Reyes et al., 2021)	F	132	PCL-R	GMV, GMD, CT	1	1	1	0	2	0	0	1	1
(Ly et al., 2012)	F	52	PCL-R	CT	1	0	0						
(Yang et al., 2010)	C	53	PCL-R	CT	0	2	0						
(de de de Oliveira-Souza et al., 2008)	C	30	PCL:SV	GMC	0	2	0	0	1	0	0	1	0
(Boccardi et al., 2011)	F	51	PCL-R	GMD	2	0	0						
(Nummenmaa et al., 2021)	F, C	38, 100	PCL-R, LSRP	GMD	1	0	0	1	0	0	0	1	0
(Miskovich et al., 2018)	F	716	PCL-R	Cortical gyrification	0	1	0	0	1	0	0	1	0
Total					**5**	**36**	**2**	**1**	**20**	**1**	**0**	**22**	**2**
Percent					**12 %**	**84 %**	**5 %**	**5 %**	**91 %**	**5 %**	**0 %**	**92 %**	**8 %**

Note: Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: - = negative relationship, + = positive relationship, ACC = anterior cingulate cortex, Pop. = population, F = Forensic, C = Community, GMV = gray matter volume, CT = cortical thickness, GMD = gray matter density, GMC = gray matter concentration, LSRP = Levenson Self-Report Psychopathy Scale, PCL-R = Psychopathy Checklist-Revised, PCL:SV = Psychopathy Checklist: Screening Version, PPI = Psychopathic Personality Inventory, PPI-R = Psychopathic Personality Inventory-Revised, SRP-SF = Self-Report Psychopathy Scale-Short Form, TriPM = Triarchic Psychopathy Measure.

There was some spatial consistency of dmPFC peak coordinates positively related to psychopathy, with eight of these overlapping ≥ 50.0 % with area 9 m. Yet, peak coordinates also appeared in the other three dmPFC subregions: four overlapped ≥ 50.0 % with area 10 m, two overlapped ≥ 50.0 % with area 6 m, and two overlapped ≥ 50.0 % with area 8 m. Additionally, we extracted 37 peak coordinates of significant dmPFC clusters related to interpersonal/affective traits and lifestyle/antisocial traits from ten studies. Patterns for these peak coordinates resembled those for the peak coordinates related to total psychopathy (see Supplemental Materials).

3.5.3. Results within tasks

Eleven studies reported 107 total tests of dmPFC activity in prototypical facial expressions tasks, 12 studies reported 54 total tests in moral tasks, and nine studies reported 76 total tests in empathy tasks. The results within the most common experimental tasks largely paralleled the results of the full review across studies of dmPFC activity, with the exception that studies that employed empathy tasks were less likely to observe null relationships and more likely to observe significant positive relationships with psychopathy. The majority of tests (≥ 61.8 % for each task category) yielded null effects. For each of the most common tasks, the majority of studies found at least one null relationship (≥ 60.0 % of studies for each psychopathy measure for each task category; Fig. 9). Within empathy tasks, half of studies found a positive relationship with interpersonal/affective traits (50.0 %), and nearly half found a positive relationship with total psychopathy (44.4 %). One third of studies that used empathy tasks found a negative relationship (33.3 % for each psychopathy measure). Nearly one half of studies that used

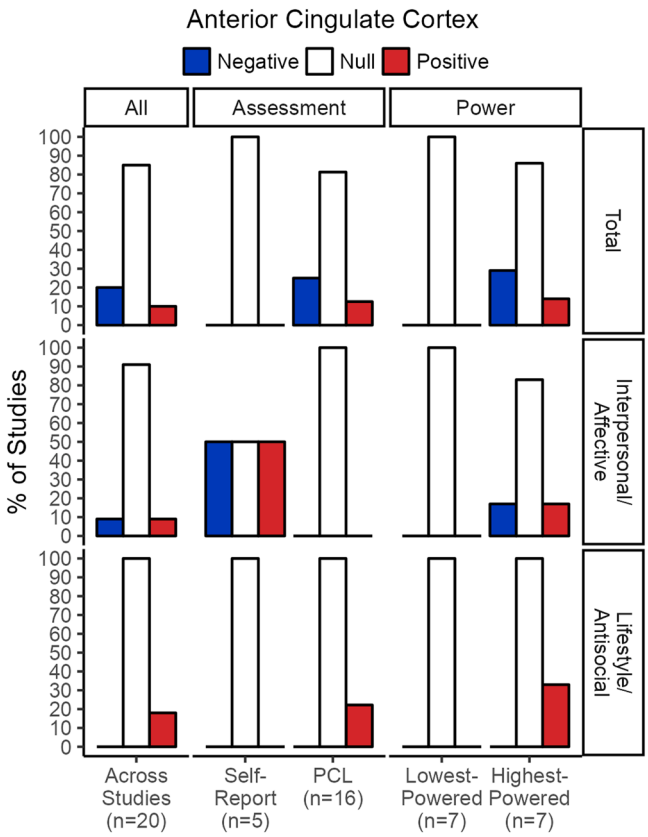

Fig. 8. Summary of studies that analyzed gray matter structure in the anterior cingulate cortex in relation to psychopathy. Cumulative percentages may exceed 100 % because many studies reported a mixture of findings (e.g., null and negative). Abbreviations: n = total number of studies included in examination.

prototypical facial expressions tasks found a positive relationship with lifestyle/antisocial traits (40.0 %). For each of the most common experimental tasks, results of dmPFC activity in specific contrasts are in Supplemental Materials.

3.5.4. Results within self-report and PCL assessments

A total of 25 studies reported 120 tests of dmPFC activity in relation to a self-report psychopathy assessment. Results largely reflected the results from the full review across studies, with the exception that these studies were more likely to observe positive relationships with lifestyle/antisocial traits. The majority of tests (89.2 %) yielded null effects. The majority of studies found at least one null relationship (≥ 80.0 % of studies for each psychopathy measure; Fig. 9). Nearly one third of studies that used self-report assessments found a positive relationship between dmPFC activity and lifestyle/antisocial traits (30.0 %), while one quarter found a positive relationship with interpersonal/affective traits (25.0 %).

A total of 45 studies reported 313 total tests of dmPFC activity in relation to a PCL assessment. These results also reflected the results from the full review across studies. The majority of tests (83.4 %) yielded null effects. The majority of these studies found at least one null relationship (≥ 83.3 % of studies for psychopathy each measure; Fig. 9). One quarter of studies found a negative relationship with interpersonal/affective traits (27.8 %), while one quarter found a positive relationship with total psychopathy (25.0 %).

3.5.5. Results of lowest- and highest-powered studies

The lowest-powered studies consisted of 23 studies that reported 185 total tests of dmPFC activity. The studies included in the full review had a mean sample size of $N = 61.4$ (range: 9–311), whereas the lowest-powered studies had a mean sample size of $N = 22.3$ (range: 9–33). The results of the lowest-powered studies paralleled the findings of the full review across studies of dmPFC activity. The majority of tests (93.5 %) yielded null effects. The majority of studies found at least one null relationship (≥ 87.5 % of studies for each psychopathy measure; Fig. 9). One quarter of the lowest-powered studies observed a positive relationship with interpersonal/affective traits or lifestyle/antisocial traits. Fewer of the lowest-powered studies observed a positive relationship between dmPFC activity and total psychopathy (14.3 %) or a negative relationship (≤ 12.5 % of studies for each psychopathy measure).

The highest-powered studies consisted of 23 studies that reported 188 total tests of dmPFC activity, with a mean sample size of $N = 117.0$ (range: 56–311). Compared to the full review and the lowest-powered studies, the highest-powered studies were less likely to observe null relationships and more likely to observe significant positive or negative relationships between dmPFC activity and psychopathy. Yet, the majority of tests (77.1 %) yielded null effects. The majority of the highest-powered studies found at least one null relationship (≥ 80.0 % of studies for each psychopathy measure; Fig. 9). Nearly one third of the highest-powered studies observed a positive relationship between dmPFC activity and total psychopathy (30.0 %), while fewer studies observed a positive relationship with interpersonal/affective traits (17.6 %) or lifestyle/antisocial traits (13.3 %). Additionally, a greater proportion of the highest-powered studies observed a negative relationship with total psychopathy (25.0 %), interpersonal/affective traits (35.3 %), and lifestyle/antisocial traits (20.0 %) than in the full review.

3.6. Structural studies of dorsomedial prefrontal cortex

3.6.1. Results across all studies

A total of 20 studies reported 64 total tests of gray matter structure in the dmPFC in relation to psychopathy (Table 9). Most studies (14/20) examined GMV in the dmPFC, four examined cortical thickness, three examined GMD, one examined GMC, and one examined cortical gyrification, with two studies examining more than one measure of gray matter structure. The majority of tests (89.1 %) yielded null effects. The majority of studies of dmPFC gray matter structure found at least one null relationship (≥ 84.2 % of studies for each psychopathy measure; Fig. 11). One quarter of studies found a negative relationship with total psychopathy (26.3 %), while no study found a negative relationship with interpersonal/affective traits (0.0 %) or lifestyle/antisocial traits (0.0 %). No study of dmPFC gray matter structure found a positive relationship (0.0 % for each psychopathy measure).

3.6.2. Results within self-report and PCL assessments

Five studies reported 12 total tests of dmPFC gray matter structure in relation to a self-report psychopathy assessment. Results of these studies were uniformly null (Fig. 11).

A total of 16 studies reported 52 total tests of dmPFC gray matter structure in relation to a PCL assessment. Results paralleled the results from the full review. The majority of tests (86.5 %) yielded null effects. The majority of studies that used a PCL assessment observed at least one null relationship (≥ 80.0 % of studies for each psychopathy measure; Fig. 11). One third of these studies observed a negative relationship between dmPFC structure and total psychopathy (33.3 %).

3.6.3. Results of lowest- and highest-powered studies

The lowest-powered studies consisted of seven studies that reported 24 total tests of dmPFC gray matter structure. The studies included in the full review had a mean sample size of $N = 90.3$ (range: 26–716), whereas the lowest-powered studies had a mean sample size of $N = 34.3$ (range: 26–39). The results of the lowest-powered studies mirrored the results of the full review. The majority of tests (91.7 %) yielded null effects. The majority of studies found at least one null relationship (≥ 85.7 % of

Table 7
Studies that analyzed task-based activity in the dmPFC in relation to psychopathy.

Study	Pop.	N	Assessment	Task	Relationship Between dmPFC Activity and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Contreras-Rodríguez et al., 2014)	F	44	PCL-R	Prototypical facial expressions	0	6	0	0	3	0	0	3	0
(Decety et al., 2014)	F	70	PCL-R	Prototypical facial expressions	4	0	0	4	0	0	4	0	0
(Deeley et al., 2006)	F	24	PCL-R	Prototypical facial expressions	0	2	0						
(Dolan and Fullam, 2009)	F	24	PCL:SV	Prototypical facial expressions	0	9	1	0	10	0	0	10	0
(Gordon et al., 2004)	C	20	PPI	Prototypical facial expressions	0	3	0	0	3	0	0	3	0
(Sethi et al., 2018)	C	232	SRP-SF	Prototypical facial expressions				0	4	0			
(Szabó et al., 2017)	C	41	ICU	Prototypical facial expressions	0	3	0						
(Tully et al., 2023)	F	58	PCL-R	Prototypical facial expressions	0	4	0						
(Mier et al., 2014)	F	29	PCL-R	Prototypical facial expressions, empathy	0	4	0						
(Decety et al., 2013b)	F	70	PCL-R	Prototypical facial expressions, empathy	1	0	1	1	0	1	1	1	0
(Sun et al., 2022)	F	58	PCL-R, LSRP	Prototypical facial expressions, vocal expressions	4	6	0	1	4	0			
(Ueltzhöffer et al., 2023)	C	52	SD3	Moral judgment	0	2	0						
(Fede et al., 2016)	F	235	PCL-R	Moral judgment	1	3	0						
(Harenski et al., 2010)	F	72	PCL-R	Moral judgment	0	5	0						
(Harenski et al., 2014)	F	157	PCL-R	Moral judgment	0	2	0	0	2	0	0	2	0
(Marsh and Cardinale, 2014)	C	33	PPI-R	Moral judgment	0	6	0						
(Reniers et al., 2012)	C	24	LSRP	Moral judgment				1	0	1	0	1	0
(Yoder et al., 2015a)	F	88	PCL-R	Moral judgment	0	2	0						
(Seara-Cardoso et al., 2016a)	C	56	SRP-SF	Moral judgment				0	8	0	0	8	0
(Caldwell et al., 2015)	F	311	PCL-R	Viewing moral images				0	1	0	0	1	0
(Cheng et al., 2021)	C	56	PPI-R	Viewing moral images	0	1	0						
(Harenski et al., 2009)	C	10	PPI	Viewing moral images, emotion regulation	0	5	0						
(Pujol et al., 2012)	F	44	PCL-R	Viewing moral dilemmas, Stroop	1	0	1						
(Deming et al., 2020)	F	94	PCL-R	Empathy	0	10	0	0	4	0	0	3	0
(Decety et al., 2013a)	F	121	PCL-R	Empathy	1	0	1	1	0	1	1	0	1
(Decety et al., 2015)	F	155	PCL-R	Empathy	0	5	3	1	2	1	0	4	0
(Meffert et al., 2013)	F	54	PCL-R	Empathy	1	1	2						
(Molenberghs et al., 2014)	C	48	SRP	Empathy	1	0	0						
(Yoder et al., 2021)	F	107	PCL-R	Empathy	0	4	0	0	4	0	0	4	0
(Veit et al., 2010)	F	9	PCL:SV, LSRS	Empathy	0	1	0	0	2	0	2	0	0
(Geurts et al., 2016)	F	34	PCL-R	Reward anticipation	0	2	0						
(Bjork et al., 2012)	C	31	PPI	Reward processing	0	1	2	0	2	1	0	2	1
(Pujara et al., 2014)	F	41	PCL-R	Reward processing	0	1	0						
(Gregory et al., 2015)	F	32	PCL-R	Reward/punishment processing	0	4	0						
(Birbaumer et al., 2005)	F	20	PCL-R	Fear conditioning	0	4	0						
(Schultz et al., 2016)	F	50	PCL-R	Fear conditioning	0	1	0						
(Veit et al., 2002)	F	15	PCL-R	Fear conditioning	0	3	0						
(Larson et al., 2013)	F	49	PCL-R	Fear conditioning, attention	0	1	0						
(Geurts et al., 2022)	F	33	PCL-R	Aversive conditioning	0	1	0						
(Fullam et al., 2009)	C	24	PPI	Deception	0	1	0	0	3	0	0	4	0
(Glenn et al., 2017)	C	16	PCL-R	Deception	0	10	0	0	20	0	0	20	0
(Abe et al., 2018)	F	43	PCL-R	Deception	1	4	0						
(Shao and Lee, 2017)	C	52	PPI-R	Deception	0	2	0						
(Anderson et al., 2017)	F	120	PCL-R	Viewing emotional images	0	4	0	0	4	0	0	4	0
(Muller et al., 2003)	F	12	PCL-R	Viewing emotional images	0	2	0						
(Shane and Groat, 2018)	F	67	PCL-R	Viewing emotional images	0	3	5						
(Kiehl et al., 2001)	F	16	PCL-R	Remembering emotional words	0	6	0						
(Sadeh et al., 2013)	C	49	NEO-FFI	Emotion word Stroop				0	1	1	0	2	0
(Volman et al., 2016)	F	34	PCL-R	Emotional control	0	1	0						
(Müller et al., 2008b)	F	22	PCL-R	Discriminating cues after emotion induction	1	1	0						
(Nummenmaa et al., 2021)	F, C	38, 100	PCL-R, LSRP	Viewing violent scenes	0	0	1	0	1	0	0	0	1
(Yoder et al., 2015b)	C	43	PPI-R	Viewing violent scenes				0	1	0	0	1	0
(Deming et al., 2018)	F	57	PCL-R	Self/other processing	0	6	0	0	6	0	0	6	0
(Overgaauw et al., 2020)	C	38	PPI-SF	Self/other processing	0	4	1						
(Nuñez et al., 2005)	C	20	PPI	Self processing				0	6	0	0	7	1
(Cope et al., 2014)	F	137	PCL-R	Viewing drug images	0	1	0	0	1	0	0	1	0
(Denomme et al., 2018)	C	105	PCL-R	Viewing drug images	0	1	1	0	1	0	0	1	0
(Rilling et al., 2007)	C	30	LSRP	Social cooperation	0	5	0						
(Overgaauw et al., 2019)	C	42	PPI-SF	Social cooperation	0	1	0						
(Osumi et al., 2012)	C	20	LSRP	Frustration	0	1	0						
(da Cunha-Bang et al., 2017)	F	44	PCL-R	Aggression	0	1	0						

(*continued on next page*)

Table 7 (*continued*)

Study	Pop.	N	Assessment	Task	Relationship Between dmPFC Activity and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Rodman et al., 2016)	F	46	PCL-R	Cognitive control	0	1	1						
(Vieira et al., 2014)	C	35	TriPM	Cognitive load	0	2	0						
(Vanova et al., 2022)	C	22	TriPM	Lexical decision-making	0	8	0						
(Freeman et al., 2015)	F	44	PCL-R	Response inhibition	0	2	0	0	2	0	0	2	0
(N. E. Anderson et al., 2018)	F	168	PCL-R	Salience processing	0	4	0	1	3	0	0	4	0
(Sommer et al., 2010)	F	28	PCL-R	Theory of mind	0	3	1						
Total					**16**	**176**	**21**	**10**	**98**	**6**	**8**	**94**	**4**
Percent					**8 %**	**83 %**	**10 %**	**9 %**	**86 %**	**5 %**	**8 %**	**89 %**	**4 %**

Note: Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: - = negative relationship, + = positive relationship, Pop. = population, F = Forensic, C = Community, ICU = Inventory of Callous-Unemotional Traits, LSRP = Levenson Self-Report Psychopathy Scale, NEO-FFI = NEO Five Factor Inventory, NEO-Tri = NEO Triarchic Scale, PCL-R = Psychopathy Checklist-Revised, PCL: SV = Psychopathy Checklist: Screening Version, PPI = Psychopathic Personality Inventory, PPI-R = Psychopathic Personality Inventory-Revised, PPI-SF = Psychopathic Personality Inventory-Short Form, SD3 = Short Dark Triad, SRP = Self-Report Psychopathy Scale, SRP-SF = Self-Report Psychopathy Scale-Short Form, TriPM = Triarchic Psychopathy Measure, YPI-SV = Youth Psychopathy Inventory-Short Version.

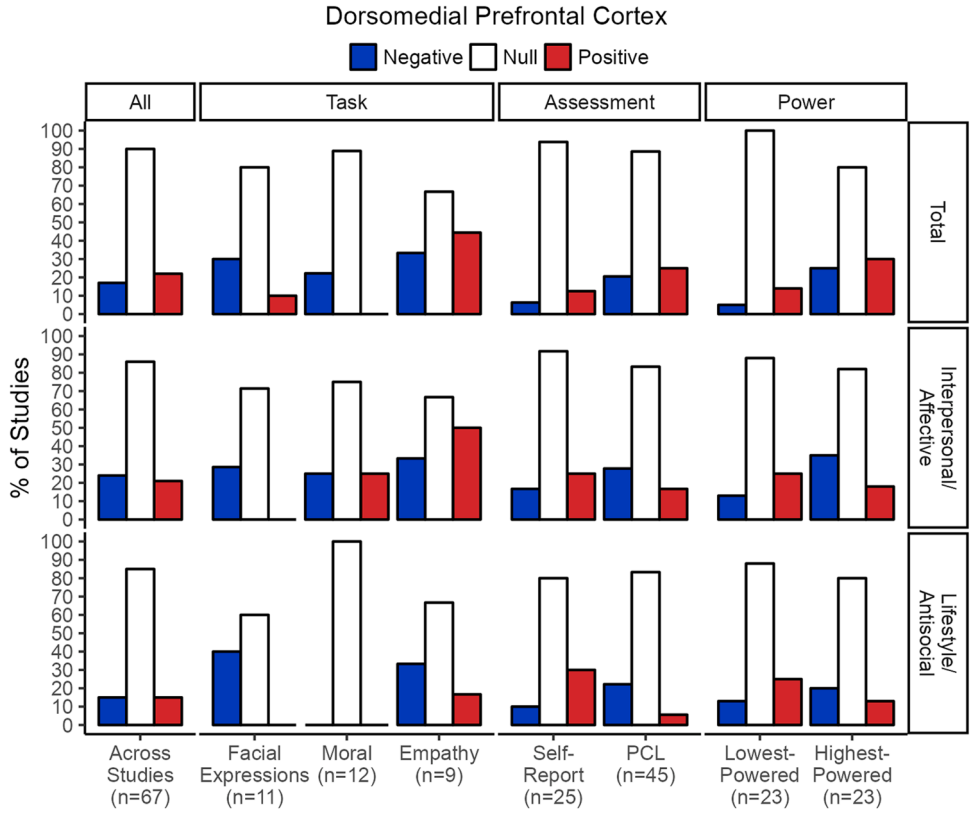

Fig. 9. Summary of studies that analyzed task-based activity in the dorsomedial prefrontal cortex in relation to psychopathy. Cumulative percentages may exceed 100 % because many studies reported a mixture of findings (e.g., null and negative). Abbreviations: n = total number of studies included in examination.

studies for each psychopathy measure; Fig. 11).

The highest-powered studies consisted of seven studies that reported 30 total tests of dmPFC gray matter structure, with a mean sample size of $N = 189.0$ (range: 57–716). Results also mirrored the results of the full review. The majority of tests (96.7 %) yielded null effects. All of the highest-powered studies observed at least one null relationship (100.0 % of studies for each psychopathy measure; Fig. 11).

4. Discussion

Prominent etiological theories of psychopathy have posited that dysfunction within the medial frontal cortex causes deficits in decision-making, emotion, and social cognition (Blair, 2005; Kiehl, 2006; Koenigs, 2012). Since their inception, these theories have been tested by more than two decades' worth of neuroimaging studies. The current study represents the most comprehensive review to date of this literature, revealing new insight into the relationship between psychopathy and the function and structure of the medial frontal cortex. Our review produced three main findings. First, null effects predominated in the literature. Second, statistically significant findings were most common among studies with larger sample sizes. Third, the peak coordinates of statistically significant clusters were widely dispersed rather than localized to one subregion, with many peak coordinates falling outside the gray matter of the medial frontal cortex. We discuss each of these

Table 8
Overlap between dorsomedial prefrontal cortex and peak coordinates associated with total psychopathy.

H	Cluster Label in Original Paper	MNI Coordinates	N	Dorsomedial Prefrontal Cortex Overlap (%)				
				Whole	Area 6m	Area 8m	Area 9m	Area 10m
Negative findings (Reduced activity)								
L	medial superior frontal gyrus	−14, 60, 14	43	41.9	0.0	0.0	0.0	41.9
R	dorsomedial prefrontal cortex	8, 58, 35	70	8.8	0.0	0.0	6.4	2.5
L	dorsomedial prefrontal cortex	−10, 40, 45	70	14.3	0.0	0.0	14.3	0.0
L	supplementary motor area	−6, 16, 54	121	**86.2**	0.0	**86.2**	0.0	0.0
R	supplementary motor area	8, 24, 46	121	**72.3**	0.0	49.9	22.4	0.0
L/R	dorsomedial prefrontal cortex	0, 58, 35	70	29.1	0.0	0.0	21.5	7.6
L	dorsomedial prefrontal cortex	−12, 45, 28	70	35.1	0.0	0.0	3.4	31.7
L/R	supplementary motor area	0, −5, 55	70	**100.0**	**100.0**	0.0	0.0	0.0
L	supplementary motor area	−10, −12, 55	70	39.3	39.3	0.0	0.0	0.0
L/R	dorsomedial prefrontal cortex	0, 50, 18	70	**92.4**	0.0	0.0	3.0	**89.4**
R	supplementary motor area	17, 12, 67	70	2.5	0.0	2.5	0.0	0.0
L	supplementary motor area	−2, −8, 58	70	**96.7**	**96.7**	0.0	0.0	0.0
R	dorsomedial prefrontal cortex	12, 50, 25	70	30.3	0.0	0.0	20.3	9.9
L	dorsomedial prefrontal cortex	−15, 45, 30	70	3.0	0.0	0.0	0.4	2.6
R	supplementary motor area	5, −18, 65	70	0.0	0.0	0.0	0.0	0.0
R	dorsomedial prefrontal cortex	8, 58, 35	70	8.8	0.0	0.0	6.4	2.5
L	dorsomedial prefrontal cortex	−10, 40, 45	70	14.3	0.0	0.0	14.3	0.0
R	supplementary motor area	15, 8, 68	70	1.2	0.0	1.2	0.0	0.0
R	Brodmann area 8	6, 27, 45	235	**91.6**	0.0	30.2	**61.5**	0.0
L	superior medial gyrus	−9, 39, 33	54	**66.8**	0.0	0.0	**59.6**	7.3
L	superior medial gyrus	−3, 30, 51	54	**86.3**	0.0	37.1	49.2	0.0
R	superior medial gyrus	6, 18, 54	54	**89.5**	0.0	**89.5**	0.0	0.0
R	pre-supplementary motor area	12, 6, 57	54	36.9	11.3	25.6	0.0	0.0
L	medial prefrontal cortex/anterior cingulate cortex	−6, 53, 31	48	**59.1**	0.0	0.0	27.0	32.1
R	medial frontal gyrus	12, 54, 25	22	24.8	0.0	0.0	6.7	18.1
R	medial frontal cortex	4, 20, 50	44	**95.2**	0.0	**95.2**	0.0	0.0
Positive findings (Increased activity)								
R	dorsomedial prefrontal cortex	6, 58, 16	70	**97.1**	0.0	0.0	0.0	**97.1**
R	supplementary motor area	8, 18, 54	70	**79.3**	0.0	**79.3**	0.0	0.0
L	dorsomedial prefrontal cortex	−10, 2, 50	70	**66.3**	**55.1**	11.2	0.0	0.0
L	dorsomedial prefrontal cortex	−4, 48, 32	155	**92.1**	0.0	0.0	**69.4**	22.7
L	dorsomedial prefrontal cortex	−2, 50, 32	155	**93.0**	0.0	0.0	**75.1**	17.9
L	dorsomedial prefrontal cortex	−2, 48, 34	155	**92.5**	0.0	0.0	**88.6**	3.9
L	dorsomedial prefrontal cortex	−5, 46, 34	105	**87.4**	0.0	0.0	**79.6**	7.8
R	Brodmann area 8	7, 53, 35	24	43.6	0.0	0.0	42.9	0.7
L	superior frontal gyrus	−3, 57, 12	54	**96.5**	0.0	0.0	0.0	**96.5**
R	superior frontal gyrus	6, 39, 30	54	**84.1**	0.0	0.0	**84.1**	0.0
R	middle frontal gyrus, superior frontal gyrus, superior medial gyrus, anterior cingulate cortex	9, 30, 45	54	**58.8**	0.0	8.0	**50.7**	0.0
L	superior medial gyrus, anterior cingulate cortex	−9, 39, 33	54	**66.8**	0.0	0.0	**59.6**	7.3
R	superior medial gyrus, anterior cingulate cortex	9, 30, 48	54	**55.6**	0.0	16.5	39.1	0.0
R	posterior medial frontal cortex	4, 32, 38	38	**99.4**	0.0	0.0	**99.4**	0.0
L	medial frontal cortex	−2, 54, 12	44	**92.0**	0.0	0.0	0.0	**92.0**
L	anterior medial prefrontal cortex	−2, 64, 22	46	**56.8**	0.0	0.0	0.0	**56.8**
L	supplementary motor area	−9, −6, 54	67	**52.1**	**52.1**	0.0	0.0	0.0
L	supplementary motor area	−6, 24, 66	67	28.3	0.0	28.3	0.0	0.0
L	supplementary motor area	−9, 15, 69	67	30.1	0.0	30.1	0.0	0.0
R	supplementary motor area	12, 9, 69	67	18.6	0.0	18.6	0.0	0.0
R	superior frontal cortex	12, 9, 66	67	23.8	0.3	23.5	0.0	0.0
L	superior frontal cortex	−15, −12, 66	67	8.8	8.8	0.0	0.0	0.0
R	superior frontal cortex, supplementary motor area, anterior cingulate cortex	9, 33, 54	67	36.0	0.0	17.2	18.8	0.0
R	superior frontal cortex, supplementary motor area, anterior cingulate cortex	12, 21, 60	67	**75.9**	0.0	**75.9**	0.0	0.0
R	superior frontal cortex, supplementary motor area, anterior cingulate cortex	12, 21, 48	67	28.7	0.0	28.7	0.0	0.0
L	medial frontal cortex	−21, 21, 6	67	0.0	0.0	0.0	0.0	0.0
R	medial frontal cortex	10, 64, 24	28	42.8	0.0	0.0	0.0	42.8

Peak coordinates that overlapped ≥ 50.0 % with the dorsomedial prefrontal cortex mask are in bold.
Abbreviations: H = hemisphere, L = left, R = right

main findings and provide recommendations for advancing the science of the neurobiology of psychopathy.

4.1. Predominantly null findings

MRI studies of psychopathy have yielded predominantly null findings in the vmPFC, ACC, and dmPFC. There was a total of 1573 tests reported across these regions, with a null proportion of 85.4 %. Addressing why null findings prevail in the field will be critical to advancing neurological research of psychopathy. The current review is not the first to observe a predominance of null relationships between psychopathy and measures of brain function or structure (see Deming et al., 2022; Griffiths and Jalava, 2017; Jalava et al., 2021, 2023). In our previous review of structural MRI studies, we found that a majority of relationships (64.1 %) between psychopathy and gray matter structure in regions across the brain were null (Jalava et al., 2021). In another

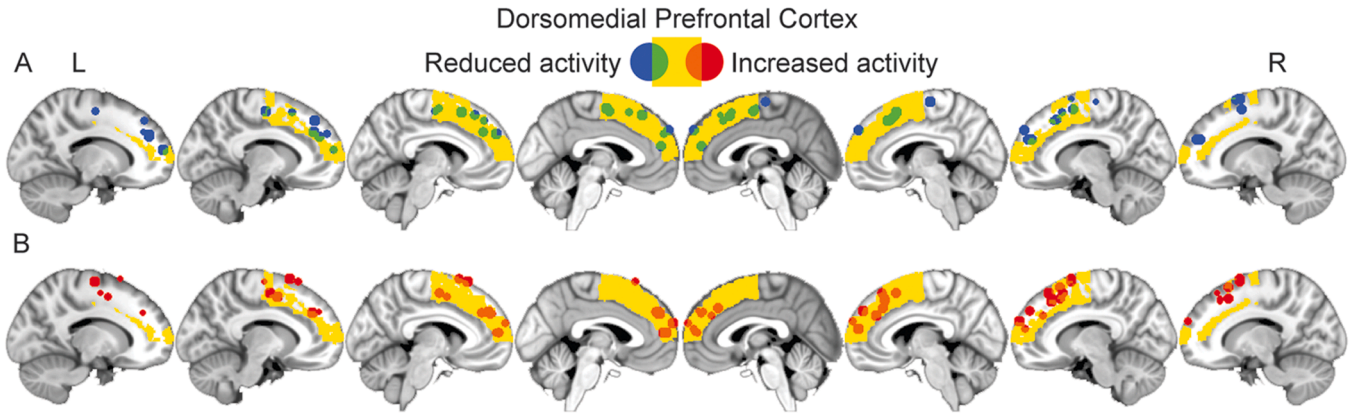

Fig. 10. Peak coordinates of dorsomedial prefrontal cortex clusters that were A) negatively related and B) positively related to total psychopathy. In each panel, sagittal slices are displayed from left to right at x = −14, −10, −6, −2, 2, 6, 10, 14.

Table 9
Studies that analyzed dmPFC structure in relation to psychopathy.

Study	Pop.	N	Assessment	Structural Measure	Relationship Between dmPFC Structure and Psychopathy Measure (# of Results by Test)								
					Total			Interpersonal/ Affective			Lifestyle/ Antisocial		
					-	Null	+	-	Null	+	-	Null	+
(Beckwith et al., 2018)	C	155	PPI	GMV	0	1	0						
(Bertsch et al., 2013)	F	39	PCL-R	GMV	0	4	0						
(Cantor et al., 2015)	F	56	LSRP	GMV	0	1	0						
(Cope et al., 2012)	F	66	PCL-R	GMV	0	2	0	0	2	0	0	2	0
(Gregory et al., 2012)	F	44	PCL-R	GMV	1	1	0						
(Hofhansel et al., 2020)	F	26	PCL-R	GMV	0	1	0	0	1	0	0	1	0
(Laakso et al., 2002)	F	57	PCL-R	GMV	1	1	0	0	1	0	0	1	0
(Leutgeb et al., 2015)	F	40	PCL-R	GMV				0	1	0	0	1	0
(Müller et al., 2008a)	F	34	PCL-R	GMV	0	2	0	0	1	0	0	1	0
(Pera-Guardiola et al., 2016)	F	39	PCL-R	GMV	0	1	0	0	2	0	0	2	0
(Tiihonen et al., 2008)	F	37	PCL-R	GMV	2	0	0						
(Vieira et al., 2015)	C	35	PPI-R, TriPM	GMV	0	2	0						
(Chester et al., 2023)	C	97	SRP-SF	GMV, CT	0	2	0	0	2	0	0	2	0
(Calzada-Reyes et al., 2021)	F	132	PCL-R	GMV, GMD, CT	0	3	0	0	2	0	0	2	0
(Ly et al., 2012)	F	52	PCL-R	CT	1	0	0						
(Yang et al., 2010)	C	53	PCL-R	CT	0	2	0						
(de de de Oliveira-Souza et al., 2008)	C	30	PCL:SV	GMC	0	2	0	0	1	0	0	1	0
(Boccardi et al., 2011)	F	51	PCL-R	GMD	2	0	0						
(Nummenmaa et al., 2021)	F, C	38, 100	PCL-R, LSRP	GMD	0	1	0	0	1	0	0	1	0
(Miskovich et al., 2018)	F	716	PCL-R	Cortical gyrification	0	1	0	0	1	0	0	1	0
Total					**7**	**27**	**0**	**0**	**15**	**0**	**0**	**15**	**0**
Percent					**21 %**	**79 %**	**0 %**	**0 %**	**100 %**	**0 %**	**0 %**	**100 %**	**0 %**

Note: Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: - = negative relationship, + = positive relationship, dmPFC = dorsomedial prefrontal cortex, Pop. = population, F = Forensic, C = Community, GMV = gray matter volume, CT = cortical thickness, GMD = gray matter density, GMC = gray matter concentration, LSRP = Levenson Self-Report Psychopathy Scale, PCL-R = Psychopathy Checklist-Revised, PCL:SV = Psychopathy Checklist: Screening Version, PPI = Psychopathic Personality Inventory, PPI-R = Psychopathic Personality Inventory-Revised, SRP-SF = Self-Report Psychopathy Scale-Short Form, TriPM = Triarchic Psychopathy Measure.

recent systematic review, we observed predominantly null relationships (≥ 76.5 %) between psychopathy and activity and volume of the amygdala (Deming et al., 2022), which had been held by several prominent etiological theories, including the *integrated emotion systems* theory (Blair, 2005) and *paralimbic hypothesis* (Kiehl, 2006), to be a primary source of neural dysfunction. The current review adds further support for the conclusion drawn from these prior studies: MRI studies of psychopathy have so far yielded little reliable evidence for altered task-based activity or gray matter structure in any of the most theoretically relevant brain regions.

There are several possible explanations for these null findings. Most importantly, psychopathy might not be reliably linked to any neural correlates across people. It is notable that there appear to be few discussions among scholars of this possibility. In fact, null findings have been vastly underreported by other theory and review papers on the neurobiology of psychopathy (Jalava et al., 2021). Alternatively, the predominance of null effects might be related to study power, experimental task, or psychopathy assessment. We review each of these explanations in turn.

4.2. *The effects of study power, task, and psychopathy assessment*

Null findings were less common, and significant findings more common, among studies that achieved higher power via larger samples, relative to studies with smaller samples. Low power resulting from small sample size is a well-documented issue in neuroimaging research that is believed to create hurdles for scientific progress (Button et al., 2013; Poldrack et al., 2017; Szucs and Ioannidis, 2020; Turner et al., 2018).

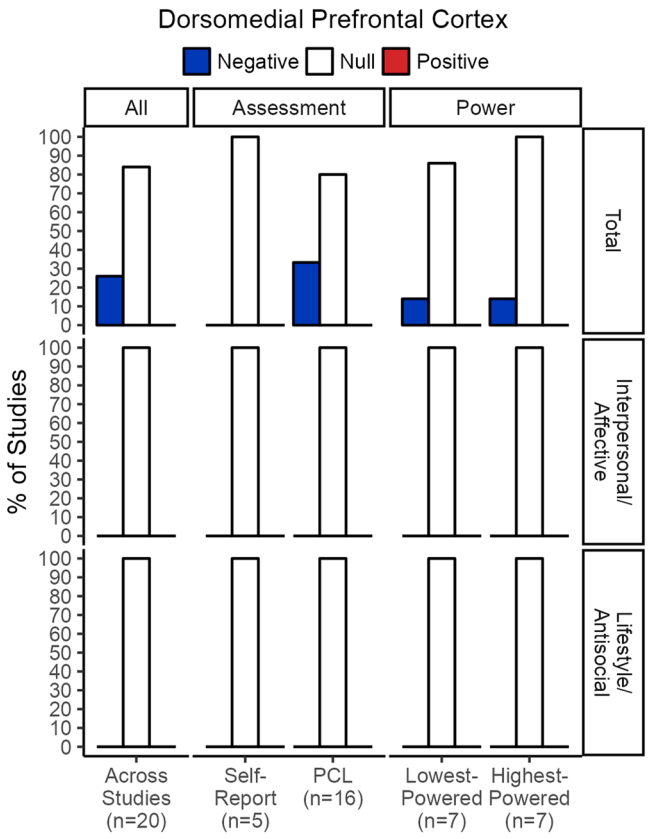

Fig. 11. Summary of studies that analyzed gray matter structure in the dorsomedial prefrontal cortex in relation to psychopathy. Cumulative percentages may exceed 100 % because many studies reported a mixture of findings (e.g., null and negative). Abbreviations: n = total number of studies included in examination.

Recruiting larger samples (ranging from over 50 to hundreds of subjects) appears to have increased studies' power to detect significant psychopathy-related effects in the medial frontal cortex. Crucially, however, null findings remained the most common result among the highest-powered studies. Perhaps more importantly, the highest-powered studies observed significantly increased activity and significantly reduced activity at similar rates. That is, the direction of statistically significant effects was inconsistent. Furthermore, most if not all of the highest-powered studies reviewed here were likely still highly underpowered. Achieving adequate power to detect relationships between personality characteristics (such as psychopathy) and brain measures (such as BOLD activity and gray matter structure) may require hundreds (Turner et al., 2018) to thousands of participants (Marek et al., 2022). The current results nonetheless suggest that analyzing larger data sets may yield a higher rate of significant findings in the medial frontal cortex.

Null findings were also relatively less common among studies that employed empathy tasks, although this finding should be interpreted with caution. Only ten studies employed empathy tasks. Of those, three studies that observed significant effects came from the same research group and likely included overlapping samples (Decety et al., 2013a, 2013b, 2015). The ten empathy studies were also characterized by heterogeneous methods (i.e., a variety of stimuli and task instructions; see Supplemental Materials). Thus, replication studies from other research groups are needed. The proportion of null findings was unrelated to the other most common tasks—prototypical facial emotion expressions tasks and moral tasks. In sum, we found preliminary evidence that altered medial frontal activity is specific to contexts in which psychopathic persons are instructed to empathize with another person (cf. Meffert et al., 2013).

We also examined whether the proportion of null findings was related to the type of psychopathy assessment used. Results were mostly null whether studies assessed psychopathy via clinician-rated measure (i.e., the Psychopathy Checklist) or via self-report. One possible explanation for the mostly null findings and heterogeneous significant findings is that heterogeneity permeates all psychopathy assessment tools, whether clinician-rated or self-report. Neuroimaging studies typically assume that high psychopathy individuals make up a homogeneous group. Yet high psychopathy as measured by the PCL likely constitutes a largely heterogeneous group of individuals. There are myriad ways to meet the conventional clinical threshold of 30 (Balsis et al., 2017), as the PCL has no necessary set of symptoms (nor any exclusion criteria; Hare, 2003). Additionally, self-report assessments construe psychopathy in different ways. Eleven separate self-report psychopathy assessments were used in the neuroimaging studies reviewed here, each assessment comprised of a unique set of items and factor structure. Neuroimaging studies may simply be reflecting considerable heterogeneity in psychopathy assessment.

4.3. *Widely dispersed peak coordinates of statistically significant findings*

Our examination of peak coordinates produced our third main finding: significant effects were widely dispersed across the frontal cortex, rather than concentrated around a single locus. This aligns with prior observations that MRI studies have related psychopathy to BOLD activity in regions scattered across the brain, including all four cortical lobes and numerous subcortical structures (Koenigs et al., 2011). On the other hand, prior meta-analyses including our own have revealed discrete regions in which psychopathy has been repeatedly associated with altered BOLD activity (Deming and Koenigs, 2020; Poeppl et al., 2018). This included area 9 m of the dmPFC. Deming and Koenigs (2020) found consistently increased activity, while Poeppl et al. (2018) found consistently *reduced* activity, within this dmPFC subregion. The current results align more closely with our own prior meta-analyses (Deming and Koenigs, 2020): many (8/27) of the peak coordinates positively related to total psychopathy fell within area 9 m of the dmPFC. Yet, the majority of dmPFC peak coordinates fell outside this subregion. Additionally, many of the medial frontal peak coordinates were situated primarily outside the medial frontal cortex, falling instead in white matter or more lateral gray matter. In sum, the current review failed to localize psychopathy-related neural dysfunction to any medial frontal subregion.

4.4. *Limitations*

The current review had several limitations. We examined only a subset of measures of medial frontal cortex function (i.e., task-based BOLD response) and structure (i.e., measures of gray matter structure) that were most relevant to neurobiological theories of psychopathy (e.g., Blair, 2005; Kiehl, 2006). We excluded other measures of function (e.g., functional connectivity) and structure (e.g., white matter integrity), which have also been linked to psychopathy (e.g., Dotterer et al., 2020; Espinoza et al., 2019; Motzkin et al., 2011). We also did not extract information about whether and how each study controlled for confounds that may have affected brain function and structure (e.g., substance use, early life adversity). Additionally, although peak coordinates indicate the voxel in which the effect of psychopathy on BOLD activity was greatest, peak coordinates do not represent the full spatial extent of significant clusters. Future studies might overcome this limitation using image-based meta-analysis (Salimi-Khorshidi et al., 2009), considered the "gold standard" for examining the topography of significant BOLD effects. Additionally, we used sample size as an index of study power, although many factors influence power in MRI studies (Marek et al., 2022; Turner et al., 2018). Lastly, we used the standard threshold of $p_{FWE} < .05$ to identify significant findings. While it is possible that

weaker effects, for example observed at $p < .001$ without family-wise error correction, may exist across studies, it was not possible for us to quantify these weaker effects as published fMRI studies do not systematically report uncorrected results.

4.5. Recommendations for advancing our understanding of the neurobiology of psychopathy

We conclude with recommendations for improving theory and the empirical study of the brain basis of psychopathy. To advance theory, we consider it necessary to account for heterogeneity in the significance, direction (i.e., positive or negative), and spatial location of brain activity/structure related to psychopathy. Perhaps the most parsimonious explanation of this heterogeneity is that there may be no consistent neural "profile" of psychopathy to be discovered. This explanation, which challenges current neurobiological theories of psychopathy, deserves consideration. Alternatively, accounting for heterogeneity in neuroimaging findings may require resolving heterogeneity in how researchers conceptualize and measure psychopathy (e.g., as a category vs. continuum, as a unitary vs. heterogeneous group; Deming and Koenigs, 2021; Koenigs et al., 2011). However, if there is no neural profile of psychopathy to be discovered, then efforts to resolve heterogeneity in how psychopathy is measured are unlikely to bear fruit. There may also be biological explanations for the mostly null and heterogeneous significant findings that may help to guide revisions to current theory in the field (e.g., degeneracy; Edelman and Gally, 2001; Sajid et al., 2020; Westlin et al., 2023).

If there is a neural "profile" of psychopathy to be discovered, then analyses of large-scale, intrinsic brain networks may have the best chance of uncovering this "profile." A number of studies have reported altered interactions between large-scale brain networks in relation to psychopathy, in particular two networks that support core cognitive functions, the default mode network and salience network (Contreras-Rodríguez et al., 2015; Decety et al., 2013a; Deming et al., 2023; Dotterer et al., 2020; Espinoza et al., 2018, 2019; Pujol et al., 2012). We recommend focusing neuroimaging analyses on interactions between large-scale brain networks rather than on task-based neural activity within individual modules (e.g., ACC).

Lastly, to improve the accuracy of anatomical labels for significant clusters, we recommend using anatomical atlases and reporting clearly how labels were derived (Poldrack et al., 2008). There is also ongoing debate about whether task-related BOLD signal can be observed in white matter, as the biological basis of white matter BOLD signal is unclear (Schilling et al., 2023). To avoid complications with interpreting significant effects of BOLD activity within white matter tracts, we recommend that future psychopathy neuroimaging studies restrict analyses to gray matter.

5. Conclusions

The evidence does not conclusively support a consistent role for medial frontal cortex in psychopathy. The extant literature is marked by predominantly null findings and considerable heterogeneity. Revising theory and adjusting methods to account for heterogeneity will be critical to advancing our understanding of neural correlates of psychopathy. Together with findings from our previous reviews (Deming et al., 2022; Griffiths and Jalava, 2017; Jalava et al., 2021; Koenigs et al., 2011), the heterogeneous findings from the current review raise concerns about using results from neuroimaging studies of psychopathy to inform decision making in legal settings (Jalava et al., 2023).

Declaration of Competing Interest

None.

Data availability

The pre-registration and complete dataset for this study can be accessed via Open Science Framework: https://osf.io/juvkm/?view_only=e0ffce18332e49c3a4b552bbbc973042

Psychopathy and medial frontal cortex: final coded dataset (Original data) (Open Science Framework)

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2024.105904.

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