Neuroscience and Biobehavioral Reviews 142 (2022) 104875

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

How reliable are amygdala findings in psychopathy? A systematic review of MRI studies

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ARTICLE INFO

Keywords:
Psychopathy
Callous-unemotional
Amygdala
Neuroimaging
MRI

ABSTRACT

The amygdala is a key component in predominant neural circuitry models of psychopathy. Yet, after two decades of neuroimaging research on psychopathy, the reproducibility of amygdala findings is questionable. We systematically reviewed MRI studies (81 of adults, 53 of juveniles) to determine the consistency of amygdala findings across studies, as well as within specific types of experimental tasks, community versus forensic populations, and the lowest- versus highest-powered studies. Three primary findings emerged. First, the majority of studies found null relationships between psychopathy and amygdala structure and function, even in the context of theoretically relevant tasks. Second, findings of reduced amygdala activity were more common in studies with low compared to high statistical power. Third, the majority of peak coordinates of reduced amygdala activity did not fall primarily within the anatomical bounds of the amygdala. Collectively, these findings demonstrate significant gaps in the empirical support for the theorized role of the amygdala in psychopathy and indicate the need for novel research perspectives and approaches in this field.

1. Introduction

The amygdala has been a central focus of the neuroscientific study of psychopathy for decades (Patrick, 1994; Birbaumer et al., 2005; Blair, 2005; Kiehl, 2006; Yang et al., 2009; Osumi et al., 2012; Marsh and Cardinale, 2014; Umbach et al., 2015). Yet, despite the accumulation of neuroimaging studies of psychopathy in the last twenty years, there has not yet been a comprehensive review and evaluation of the evidence for (or against) this hypothesized neural correlate of psychopathy. The present systematic review intends to clarify the consistency of amygdala findings in neuroimaging studies of psychopathy.

The amygdala has often been called the “fear center” of the brain; that is, the key node of what many researchers consider to be a circuit devoted to the subjective experience of and behaviors associated with fear (Maren, 2001; Perusini and Fanselow, 2015; critically reviewed in LeDoux, 2020). The “fear center” view has drawn on convergent evidence from animal studies, studies of humans with amygdala damage, and neuroimaging studies of healthy humans. Animal studies have documented a central role of the amygdala in pairing threatening cues with non-threatening cues (classically referred to as "fear conditioning"; Maren, 2001; Fendt and Fanselow, 1999). Patients with bilateral amygdala damage have shown a generally impoverished experience of the emotion category of fear (Feinstein et al., 2011), deficits in fear conditioning (Bechara et al., 1995), and impaired processing of prototypical facial expressions of fear (Adolphs et al., 1994, 1995, 1999, 2005). Human neuroimaging studies have also implicated the amygdala in a number of related functions, including orienting to threat cues (Davis and Whalen, 2001), processing prototypical facial expressions of fear (Jiang and He, 2006), and feelings of autonomic arousal (Wilson-Mendenhall et al., 2013; Touroutoglou et al., 2014).

The “fear center” view of the amygdala has informed etiological theories of psychopathy to varying degrees. An early influential theory (Lykken’s low fear hypothesis) proposed that psychopathy is rooted in “fearlessness,” a diminished capacity for experiencing fear and for associating actions with, for example, the threat of punishment (Lykken, 1957, 1995). Patrick attributed this “fearlessness” to the amygdala, proposing that amygdala dysfunction may be related to the lack of startle response to aversive and threatening cues displayed by people with psychopathy (Patrick, 1994). Blair argued that reduced amygdala activity may be related to impairments associating aversive cues with non-aversive cues, particularly associating others’ facial muscle movements with distress (e.g., fear and sadness; Blair, 2003, 2005). A later

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https://doi.org/10.1016/j.neubiorev.2022.104875
Received 12 July 2022; Received in revised form 31 August 2022; Accepted 13 September 2022
Available online 15 September 2022

theory specified that the basolateral subnucleus of the amygdala, but not the central nucleus, may be underactive, resulting in impaired shifting of attention to cues that predict reward or threat (Moul et al., 2012). These theories and others posit that reduced amygdala activity and volume is accompanied by dysfunction in other (primarily limbic) cortical and subcortical regions (Blair, 2005; Kiehl, 2006; Moul et al., 2012). Notably, a large body of research investigating amygdala function, both in healthy people and individuals with psychopathy, has amassed since these theories were formulated.

Crucially, recent evidence has challenged the idea of the amygdala as the brain's "fear center." The amygdala may not be necessary for the subjective experience of fear (LeDoux, 2020); a patient with bilateral amygdala damage has reported feeling afraid in certain contexts (Feinstein et al., 2013; Barrett and Satpute, 2019). Meta-analyses further suggest that the human amygdala may not be involved in pairing threatening with non-threatening cues (Fullana et al., 2016, 2018; Visser et al., 2021), in contrast to findings from animal studies. There is even mounting evidence refuting the notion that a circuit devoted to the emotion category of fear, or any other discrete emotion category, for that matter, exists in the human brain (Touroutoglou et al., 2015; Barrett, 2017; Barrett et al., 2007). Instead, the amygdala may be better understood as part of a broader, domain-general brain network whose primary function is to sense and control the viscera (Kleckner et al., 2017) and whose emergent functions include representing affect (i.e., feelings characterized by valence and arousal; Pessoa and Adolphs, 2010; Lindquist et al., 2012, 2016). This revised understanding of amygdala function in healthy brains suggests the need to reevaluate the amygdala's role in representing discrete emotion categories and in associating threatening (or aversive) cues with non-threatening (or non-aversive) cues in psychopathy.

At this time, a clear picture of the amygdala's role in these and other processes in psychopathy is lacking, as indicated by several pieces of evidence. First, MRI studies using a variety of experimental tasks have yielded mixed results. Some studies have indeed found a negative relationship with psychopathy (i.e., reduced amygdala activity or volume; Yang et al., 2009; Yoder et al., 2015a; Sethi et al., 2018), although other studies have observed a positive (i.e., increased amygdala activity or volume; Schiffer et al., 2011; da Cunha-Bang et al., 2017; Denomme et al., 2018) or null relationship with psychopathy (Cope et al., 2012; Caldwell et al., 2015; Zijlmans et al., 2018). Even studies using tasks in the same domain have yielded opposing findings. For example, Birbaumer et al. (2005) associated psychopathy with *reduced* amygdala activity in response to an aversive cue paired with a non-aversive cue (i.e., the acquisition phase of a "fear conditioning" task), whereas Schultz et al., (2016) found *increased* amygdala activity. Second, two coordinate-based meta-analyses of fMRI studies of psychopathy yielded opposing findings: Poeppl et al. (2018) found aggregated evidence of *reduced* activity of the right amygdala, whereas a more recent study using multilevel kernel density analysis found aggregated evidence of *increased* activity of the right amygdala across a variety of tasks (Deming and Koenigs, 2020). However, these two meta-analyses both necessarily excluded dozens of studies that used region-of-interest (ROI) analyses of the amygdala, and thus do not represent the whole of the neuroimaging literature on psychopathy. Third, a recent analysis concluded that published reviews of amygdala structural abnormalities in psychopathy have underreported null findings (Jalava et al., 2021). Fourth, a case study found that bilateral amygdala damage does not result in the expression of the core social-affective deficits of psychopathy (Lilienfeld et al., 2016). Taken together, these findings indicate the need to comprehensively evaluate the literature on the relationship between psychopathy and amygdala structure and function.

The current study aimed to achieve this through a systematic literature review of MRI studies of psychopathy in adult and juvenile samples. The review included any study that had the potential to observe a relationship between the amygdala and psychopathy, by examining the amygdala either as part of the whole brain or as a region of interest (ROI). Thus, our first goal was to characterize the consistency of the relationship between psychopathy and amygdala function (during experimental tasks) and structure across studies. We subsequently examined the consistency of the relationship between psychopathy and amygdala function in specific experimental tasks, such as face processing tasks. Similarly, we examined the consistency of findings in specific populations (i.e., forensic or community; adult or juvenile) and for different sample sizes. Finally, we examined the spatial distribution of reported amygdala findings through an analysis of peak coordinates, to characterize the degree of anatomical overlap within the amygdala. We conducted these examinations in relation to total scores on conventional psychopathy measures, as well as in relation to scores for specific clusters of traits, including interpersonal/affective (e.g., shallow affect, callousness, deceitfulness) and lifestyle/antisocial traits (e.g., impulsivity, irresponsibility, varied criminal behavior).

2. Methods

2.1. Study selection and coding

A forward literature search was conducted in PubMed, PsycINFO, and Google Scholar, and a backward literature search was conducted in the reference sections of relevant papers. The following terms were used in the forward literature search for studies of adults: psychopathy, psychopathic, magnetic resonance imaging, MRI, functional magnetic resonance imaging, fMRI, and neuroimaging. The following terms were used in the forward literature search for studies of juveniles: callous unemotional, magnetic resonance imaging, MRI, functional magnetic resonance imaging, fMRI, and neuroimaging. Studies published prior to June 30, 2022 were included based on the following criteria: 1) analyzed gray matter structure or task-based BOLD response of the amygdala (using either a whole-brain or ROI approach), 2) tested the main effect of a measure of psychopathy, 3) reported group statistics rather than single-case results, 4) recruited participants $\geq$ 18 years of age (for studies of adults) or $<$ 18 years of age (for studies of juveniles), 5) published in a peer-reviewed academic journal, and 6) written in English.

Studies of adults and juveniles were reviewed separately. For studies of adults, we sought to characterize the relationship between the amygdala and three measures of psychopathy: total psychopathy, interpersonal/affective traits, and lifestyle/antisocial traits. Measures of total psychopathy assessed the full set of psychopathy traits for that measure (i.e., all subsets of traits). If studies further subdivided the interpersonal/affective traits or lifestyle/antisocial traits, we included the findings in our review of the two overarching sets of traits. For example, findings related to the antisocial traits (e.g., Cope et al., 2014) were included in our review of the lifestyle/antisocial traits. Similarly, we reviewed studies of juveniles with the aim of characterizing three measures of psychopathic traits: total psychopathy, callous-unemotional traits, and externalizing traits (e.g., conduct disorder, disruptive behavior disorder). Measures of total psychopathy included assessments of the full set of traits for that measure, as well as group analyses of juveniles with conduct disorder and high callous-unemotional traits. Studies that further subdivided callous-unemotional traits or externalizing traits were handled in the same manner as in the studies of adults.

First, we ensured that relevant statistical contrasts were coded such that "positive" within-subjects activation indicated greater activity associated with the cognitive function of interest (e.g., Faces $>$ Shapes). Next, results of each relevant between-subjects test were extracted from each study and coded as negative (i.e., reduced activity or volume in relation to psychopathy), positive (i.e., increased activity or volume), or null (i.e., non-significant). Results that did not survive family-wise error correction (i.e., for voxel-wise analyses) were coded as null. Many functional studies reported results from multiple contrasts, for example Faces $>$ Shapes and Angry Faces $>$ Neutral Faces. Similarly, several structural studies reported results from multiple measures of gray matter

structure, for example gray matter volume (GMV) and gray matter concentration (GMC). We extracted results from all reported functional contrasts and structural measures. Thus, it was possible for studies to report a mixture of findings (e.g., negative and null) if they analyzed multiple functional contrasts or structural measures. Next, to examine consistency of findings across studies, we summed the number of studies that reported at least one negative, positive, or null result. Finally, we calculated the percentage of studies that reported at least one negative, positive, or null result by dividing the sums by the total number of studies that analyzed the measure of interest (e.g., total psychopathy in relation to amygdala activity). Because each study could report a mixture of findings, it was possible for the calculated percentages to sum to more than 100%. We display percentages in figures and report exact counts and percentages in Supplemental Materials.

2.2. Examination of peak coordinates of significant amygdala findings

To characterize the spatial consistency of psychopathy-amygdala relationships, we extracted the peak coordinates of amygdala clusters that were significantly related to psychopathy. Fewer than five studies of amygdala structure in adult or juvenile samples reported peak coordinates. We therefore only examined peak coordinates reported by studies of amygdala activity. Accuracy of the extracted peak coordinates was verified by a second coder. Peak coordinates were coded as negative or positive as described above. Coordinates reported in Talairach template space were converted to Montreal Neurological Institute (MNI) space in the MNI-Talairach Tool of the BioImage Suite (Lacadie et al., 2008). We then created a sphere with 4 mm radius around each coordinate. This radius length corresponds to a recommended smoothing kernel for preserving regional specificity and sensitivity in amygdala analyses (Morawetz et al., 2008). Next, we mapped the spheres onto the amygdala mask from the Brainnetome Atlas in AFNI 21.0 (Fan et al., 2016; Cox, 1996). The atlas subdivides the amygdala into medial and lateral portions, the latter of which approximates the basolateral subnucleus. Coordinates were mapped onto the medial, lateral, and full amygdala masks. Lastly, we calculated the percent of voxels in each peak coordinate sphere that overlapped with the amygdala mask (3dABoverlap in AFNI). Overlap $\geq$ 50% indicated the peak coordinate was within the bounds of the amygdala mask, whereas no (0.0%) or partial (0.1–49.9%) overlap indicated the peak coordinate was outside the bounds of the amygdala mask.

2.3. Examination of experimental tasks

We labeled experimental tasks to reflect the stimuli and task demands within each study and to capture similarities between studies. We reviewed the consistency of findings separately for experimental tasks that were employed by at least five studies. The most common tasks were coded as follows. Experimental paradigms that involved viewing facial configurations typically associated with discrete emotion states (e.g., fear, sadness) were coded as "prototypical facial emotion expressions." Paradigms that required participants to make judgments about moral dilemmas or rate the moral severity of scenarios were coded as "moral judgment." Tasks that presented participants with moral dilemmas or images but did not require a judgment were coded as "viewing moral images," and were reviewed in combination with moral judgment tasks. Paradigms that required participants to identify another person's emotion based on information other than (or in addition to) facial configuration were coded as "empathy." Finally, paradigms that involved the anticipation or receipt of a reward were coded as "reward processing." Several reward processing tasks also included a reward loss condition, which was coded as "punishment processing."

2.4. Examination of population

Neuroimaging studies of psychopathy have commonly recruited samples from one of two populations: facilities such as prisons, jails, detention centers, and forensic hospitals, which we call "forensic samples," or the general public, which we call "community samples." Individuals presenting with high levels of psychopathy are more prevalent in forensic samples than community samples (Hare, 2003). It is possible that the neural correlates of severe psychopathy differ from the neural correlates of normal variation in social, affective, and behavioral traits, the latter being the focus of studies of community samples (Koenigs et al., 2011). We sought to determine whether the amygdala is a consistent neural correlate of high psychopathy. Thus, in the literature on adults we separately examined studies of forensic samples and studies of community samples. Samples of subjects with a criminal history were coded as forensic. In the literature on juveniles, studies have commonly recruited samples from a third population, which we call "clinical samples:" those with a diagnosis of externalizing disorders, such as conduct disorder or oppositional defiant disorder, but without a criminal history. Because these studies of clinical samples recruited participants with elevated levels of psychopathic (i.e., externalizing) traits, in the literature on juveniles we examined studies of clinical samples together with studies of forensic samples. We will refer to these collectively as "enriched samples." In sum, we examined the consistency of amygdala findings in forensic vs. community samples in studies of adults and in enriched (i.e., forensic plus clinical) vs. community samples in studies of juveniles.

2.5. Examination of study power

The adverse effect of small sample sizes on study power and reproducibility has been widely recognized in the field of neuroimaging (Button et al., 2013; Poldrack et al., 2017; Szucs and Ioannidis, 2020; Marek et al., 2022; Cremers et al., 2017). Therefore, we examined the effect of study power on the consistency of amygdala findings, using sample size as an approximation of power. We separately examined the top one-third and bottom one-third of studies based on sample size. We selected this method because of the complexity of selecting a single objective cutoff for sufficiently high-powered studies and because an objective cutoff would likely exclude the majority of studies (Marek et al., 2022; Cremers et al., 2017). We elaborate on issues with post-hoc estimation of study power in the Discussion.

3. Results

In all, 81 studies of psychopathy in adult samples, including 62 functional and 21 structural studies, and 53 studies of callous-unemotional traits in juveniles, including 40 functional and 13 structural studies, were included in the review. Fig. 1 displays the number of studies excluded at each stage of the literature search.

3.1. Functional studies of adults

3.1.1. Results across all studies

Table 1 displays the 62 studies of adults that analyzed relationships between amygdala activity and a measure of psychopathy. These studies are summarized in Fig. 2. Collapsed across experimental tasks, over three-quarters of studies found a null relationship between amygdala activity and the three measures of psychopathy. Negative relationships with total psychopathy (35.3% of studies) or interpersonal/affective traits (28.9%) were more common than positive relationships with these measures (17.6% and 2.6%, respectively). Studies of lifestyle/antisocial traits were roughly equally likely to find a negative relationship (15.2%) as a positive relationship (18.2%).

3.1.2. Peak coordinates of results across all studies

We extracted a total of 41 peak coordinates from 26 studies of adults. Five of the 15 peak coordinates negatively related to total psychopathy overlapped 0.0% with the amygdala, six overlapped 0.1–49.9%, and

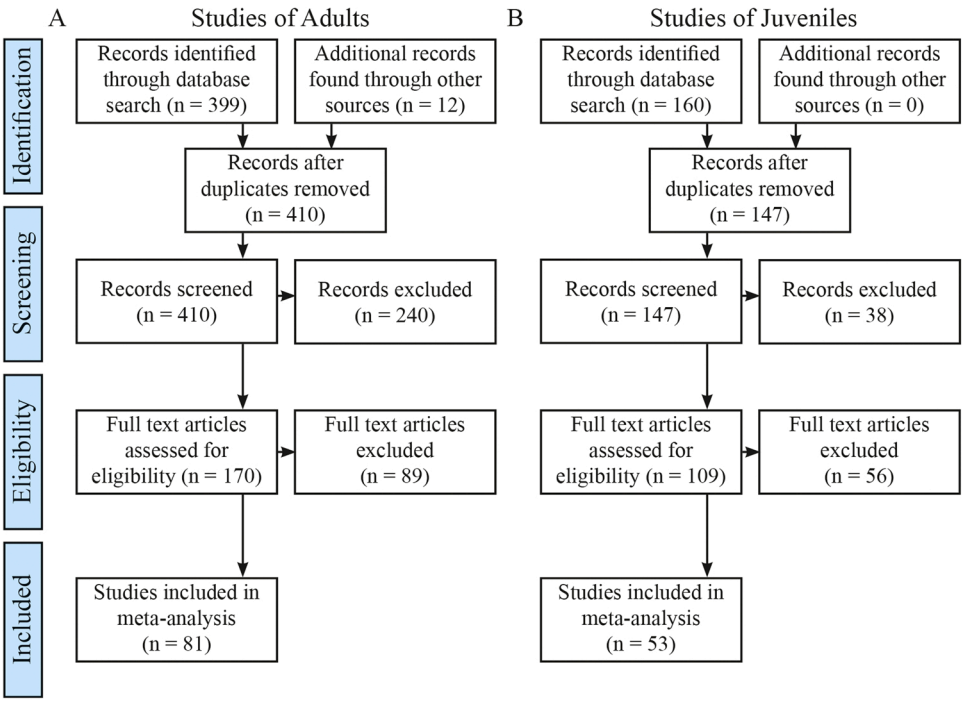

Fig. 1. Flowchart of the literature search process for studies of A) adults and B) juveniles. After a full-text review, studies were excluded for the following reasons: not analyzing a main effect of psychopathy or callous-unemotional traits in relation to amygdala gray matter structure or task-based BOLD activity (65 studies of adults, 37 studies of juveniles), summarizing prior literature rather than reporting new findings (22 studies of adults, 14 studies of juveniles), recruiting a mixed sample of adults and juveniles (1 study of adults, 1 study of juveniles), reporting single-case results (1 study of adults), not published in a peer-reviewed academic journal (3 studies of juveniles), not written in English (1 study of juveniles).

four overlapped ≥ 50.0% with the amygdala (Table 2, Fig. 3). Additionally, these peak coordinates did not overlap differentially with medial or lateral amygdala (Table 2). By contrast, the nine peak coordinates positively related to total psychopathy more consistently overlapped with the amygdala. Five of these coordinates overlapped ≥ 50.0% and four overlapped 0.1–49.9%. Again, these peak coordinates did not overlap differentially with medial or lateral amygdala (Table 2). Similar patterns were observed for peak coordinates related to interpersonal/affective traits and lifestyle/antisocial traits, which are summarized in Supplemental Materials.

3.1.3. Results within tasks

The most common experimental tasks involved viewing prototypical facial emotion expressions, making moral judgments or viewing moral images, and empathizing with another person (Table 1). Overall, the results within tasks paralleled the results across tasks; studies most commonly found a null relationship with the three measures of psychopathy (≥ 71.4% of studies for each psychopathy measure for each task category; Fig. 2). The next most common finding for each task category was a negative relationship. Half of studies (50.0%) found a negative relationship between total psychopathy and amygdala activity during moral judgment tasks. Within prototypical facial emotion expression tasks, half of studies (50.0%) found a negative relationship with interpersonal/affective traits. Positive relationships were the least common finding, with one exception: three studies (37.5%) found a positive relationship between lifestyle/antisocial traits and amygdala activity during prototypical facial emotion expression tasks. Results of specific contrasts within these tasks are presented in Supplemental Materials.

3.1.4. Results within community and forensic populations

The results from 22 studies of community samples mirrored the results of the full review across studies, with the exception that higher proportions observed negative relationships with total psychopathy and interpersonal/affective traits (Fig. 2). The most common finding was a null relationship with the three measures of psychopathy (≥ 61.5% of studies for each measure). Nearly half of studies of community samples found a negative relationship with total psychopathy (46.2%) and interpersonal/affective traits (47.1%). Similar to the full review, few studies observed a positive relationship with the three psychopathy measures (≤ 30.8% of studies for each measure).

The results from 41 studies of forensic samples mirrored the results of the full review across studies, with the exception that the relationship between amygdala activity and the interpersonal/affective traits was more consistently null (Fig. 2). The most common finding was a null relationship with the three measures of psychopathy (≥ 82.1% of studies for each measure). Similar to the full review, roughly one-third of studies (30.8%) found a negative relationship with total psychopathy. However, fewer studies found a negative relationship with interpersonal/affective (13.6%) or lifestyle/antisocial traits (14.3%). One-fifth of studies (20.5%) found a positive relationship with total psychopathy, while fewer studies found a positive relationship with interpersonal/affective (4.5%) or lifestyle/antisocial traits (9.5%).

3.1.5. Results of lowest- and highest-powered studies

The studies included in the full review across studies of adults had a mean sample size of $N = 62.9$ (range: 10–245). By contrast, the 21 lowest-powered functional studies of adults had a mean sample size of $N = 24.3$ (range: 10–34). The lowest-powered studies also most commonly found a null relationship with the three psychopathy measures (≥ 69.2% of studies for each measure; Fig. 2). However, these lowest-powered studies reported negative relationships with total psychopathy (61.1%) and interpersonal/lifestyle traits (69.2%) at rates much higher than those observed in the full review or in the highest-powered studies.

The 21 highest-powered functional studies of adults had a mean sample size of $N = 122.0$ (range: 57–245). The results of the highest-powered studies largely mirrored the results of the full review across studies, with the exception that relationships were even more consistently null (≥ 84.2% of studies for each measure; Fig. 2).

3.2. Functional studies of juveniles

3.2.1. Results across all studies

Table 3 displays the 40 studies of juveniles that analyzed relationships between amygdala activity and a measure of psychopathy. These

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

Study	Pop.	N	Assessment	Task	Relationship Between Amygdala Activity and Psychopathy Measure		
					Total	Interpersonal/ Affective	Lifestyle/ Antisocial
(Contreras-Rodríguez et al., 2014)	F	44	PCL-R	Prototypical facial expressions	Null	Null	Null
(Decety et al., 2014)	F	70	PCL-R	Prototypical facial expressions	Null	Null	Null
(Pardini and Phillips, 2010)	F	42	SRP	Prototypical facial expressions		Null	Null
(da Cunha-Bang et al., 2019)	F	47	PCL-R	Prototypical facial expressions		Null	
(Deeley et al., 2006)	F	15	PCL-R	Prototypical facial expressions	Null		
(Han et al., 2012)	C	34	PPI-R	Prototypical facial expressions		–, Null	
(Szabó et al., 2017)	C	41	ICU	Prototypical facial expressions		Null	
(Vieira et al., 2016)	C	23	PPI-R	Prototypical facial expressions		–, Null	
(Carré et al., 2013)	C	200	SRP-SF	Prototypical facial expressions		–, Null	+, Null
(Dolan and Fullam, 2009)	F	24	PCL:SV	Prototypical facial expressions	–, +, Null	–, Null	+, Null
(Gordon et al., 2004)	C	20	PPI	Prototypical facial expressions	Null	–	+
(Bobes et al., 2013)	C	60	PCL-R, LSRP	Prototypical facial expressions		Null	–, Null
(Seara-Cardoso et al., 2016a)	C	30	SRP-SF	Prototypical facial expressions	–	–	Null
(Sethi et al., 2018)	C	232	SRP-SF	Prototypical facial expressions	–		
(Decety et al., 2013a)	F	80	PCL-R	Prototypical facial expressions, empathy	Null		
(Mier et al., 2014)	F	29	PCL-R	Prototypical facial expressions, empathy	–, Null		
(Deming et al., 2020)	F	94	PCL-R	Empathy	Null	Null	Null
(Decety et al., 2015)	F	155	PCL-R	Empathy	+, Null	Null	
(Meffert et al., 2013)	F	44	PCL-R	Empathy	–, Null		
(Yoder et al., 2021)	F	107	PCL-R	Empathy	Null	Null	Null
(Decety et al., 2013b)	F	121	PCL-R, PPI-R	Empathy, imagining self-pain	–, Null	–	Null
(Harenski et al., 2010)	F	32	PCL-R	Moral judgment	–, Null	–, Null	Null
(Marsh and Cardinale, 2014)	C	33	PPI-R	Moral judgment	–, Null	–	Null
(Yoder et al., 2015a)	F	88	PCL-R	Moral judgment	–, Null	Null	Null
(Zijlmans et al., 2018)	F	122	YPI-SV	Moral judgment	Null	Null	Null
(Seara-Cardoso et al., 2016b)	C	32	SRP-SF	Moral judgment		Null	+, Null
(Harenski et al., 2014a)	F	164	PCL-R	Moral judgment	–	Null	–
(Fede et al., 2016)	F	245	PCL-R	Moral judgment	Null	Null	Null
(Glenn et al., 2009)	C	17	PCL-R	Moral judgment	–	–	–
(Caldwell et al., 2015)	F	87	PCL-R	Viewing moral images	Null	Null	Null
(Pujol et al., 2012)	F	44	PCL-R	Viewing moral dilemmas, Stroop	Null		
(Harenski et al., 2009)	C	10	PPI	Viewing moral images, emotion regulation	Null	–, Null	
(Birbaumer et al., 2005)	F	20	PCL-R	Fear conditioning	–		
(Schultz et al., 2016)	F	66	PCL-R	Fear conditioning	+		
(Larson et al., 2013)	F	49	PCL-R	Fear conditioning, attention	–, Null		
(Schneider et al., 2000)	F	24	PCL-R	Aversive conditioning	–, +, Null		
(Geurts et al., 2016)	F	34	PCL-R	Reward processing	Null		Null
(Bjork et al., 2011)	C	31	PPI	Reward processing	Null	Null	Null
(Pujara et al., 2014)	F	41	PCL-R	Reward processing	Null		
(Gregory et al., 2015)	F	50	PCL-R	Reward/punishment processing	Null		
(Nummenmaa et al., 2021) †	F; C	38; 100	PCL-R; LSRP	Viewing violent scenes	Null	Null	Null
(Yoder et al., 2015b)	C	43	PPI-R	Viewing violent scenes		Null	Null
(Seara-Cardoso et al., 2015)	C	53	SRP-SF	Viewing violent images		Null	Null
(Overgaauw et al., 2020)	C	49	PPI-SF	Social cooperation	Null		
(Rilling et al., 2007)	C	30	LSRP	Social cooperation	–	Null	Null
(Denomme et al., 2018)	F	47	PCL-R	Viewing drug images	+	+	Null
(Cope et al., 2014)	F	137	PCL-R	Viewing drug images	Null	Null	–, Null
(Anderson et al., 2017)	F	120	PCL-R	Viewing emotional images	Null	Null	–, +
(Müller et al., 2003)	F	18	PCL-R	Viewing emotional images	+, Null		
(Kiehl et al., 2001)	F	24	PCL-R	Remembering emotional words	–		
(Sadeh et al., 2013)	C	49	NEO-FFI	Emotion-word Stroop		Null	+, Null
(Shane and Groat, 2018)	F	38	PCL-R	Emotion regulation	–, +		
(Osumi et al., 2012)	C	20	LSRP	Frustration	–, Null		
(da Cunha-Bang et al., 2017)	F	44	PCL-R	Aggression	+		
(Rodman et al., 2016)	F	49	PCL-R	Cognitive control	Null		
(Abe et al., 2018)	F	67	PCL-R	Dishonest decision making	Null		
(Vieira et al., 2014a)	C	36	TriPM	Economic decision-making	Null		
(Sommer et al., 2010)	F	28	PCL-R	Theory of Mind	Null		
(Deming et al., 2018)	F	57	PCL-R	Self/other judgment	Null	Null	Null
(Overgaauw et al., 2019)	C	42	PPI-SF	Social conformity	+		
(Anderson et al., 2018)	F	168	PCL-R	Salience processing	Null	Null	Null
(Kiehl et al., 2004)	F	16	PCL-R	Reading concrete and abstract words	Null	Null	Null

Note: Multiple relationships are listed for studies that analyzed multiple experimental contrasts or comparisons and yielded mixed results. 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 (Frick, 2004), LSRP = Levenson Self-Report Psychopathy Scale (Levenson et al., 1995), NEO-FFI = NEO Five Factor Inventory (Costa and Macrae, 1992), PCL-R = Psychopathy Checklist-Revised (Hare, 2003), PCL:SV = Psychopathy Checklist: Screening Version (Hart et al., 1995), PPI = Psychopathic Personality Inventory (Lilienfeld and Andrews, 1996), PPI-SF = Psychopathic Personality Inventory-Short Form (Tonnaer et al., 2013), PPI-R = Psychopathic Personality Inventory-Revised (Lilienfeld and Widows, 2005), SRP = Self-Report Psychopathy Scale (Paulhus et al., 2015), SRP-SF = Self-Report Psychopathy Scale-Short Form (Paulhus et al., 2015),

TriPM = Triarchic Psychopathy Measure (Patrick, 2010), YPI-SV = Youth Psychopathy Inventory-Short Version (Van Baardewijk et al., 2010).
†One study separately analyzed a forensic sample and a community sample. Characteristics of each sample are separated by a semi-colon.

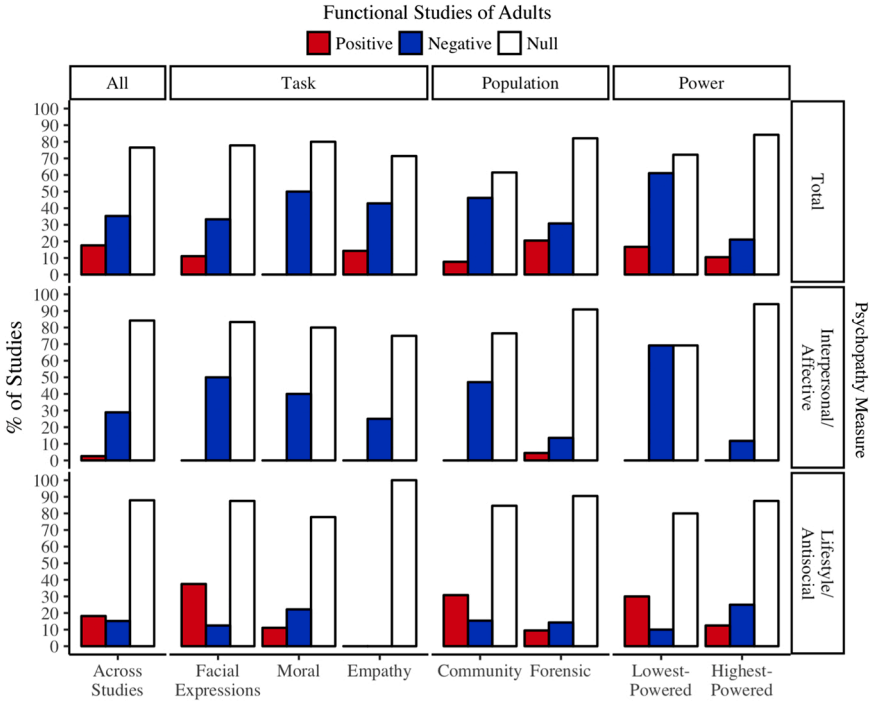

Fig. 2. Summary of studies that analyzed task-based activity in the amygdala in relation to psychopathy in adults.

studies are summarized in Fig. 4. Collapsed across experimental tasks, most studies of juveniles found a null relationship between amygdala activity and the three psychopathy measures (≥ 68.2% of studies for each measure). Half of studies found a negative relationship with total psychopathy (54.5%) and callous-unemotional traits (48.3%), and nearly half (41.9%) found a negative relationship with externalizing behavior. Few studies found a positive relationship with total psychopathy (9.1%) or callous/unemotional traits (3.4%), while one third of studies (32.3%) found a positive relationship between amygdala activity and externalizing behavior.

3.2.2. Peak coordinates of results across all studies

We extracted a total of 56 peak coordinates from 24 studies of juveniles. Similar to the literature on adults, five of the 16 peak coordinates negatively related to total psychopathy overlapped 0.0% with the amygdala, six overlapped 0.1–49.9%, and five overlapped ≥ 50.0% (Table 4, Fig. 5). Peak coordinates negatively related to total psychopathy overlapped more frequently with medial than lateral amygdala (see Table 4). Only one peak coordinate was positively related to total psychopathy in juveniles (see Table 4). Similar patterns were observed for peak coordinates related to callous-unemotional traits and externalizing traits, which are summarized in Supplemental Materials.

3.2.3. Results within tasks

Among studies of juveniles, the most common experimental tasks involved viewing prototypical facial emotion expressions and reward/punishment processing (Table 3). Results within tasks largely reflected the results across tasks (Fig. 4). The most common finding was a null relationship between amygdala activity and the three psychopathy measures (≥ 70.0% of studies for each psychopathy measure for each task category). Within prototypical facial emotion expression tasks, half of studies found a negative relationship with total psychopathy (50.0%) and externalizing traits (50.0%), and a majority of studies (70.0%) found a negative relationship with callous-unemotional traits. Within reward/punishment processing tasks (specifically in punishment conditions), few studies (28.6%) found a negative relationship with callous-unemotional traits. Fewer studies found a positive relationship, although half of prototypical facial emotion expression studies (50.0%) found a positive relationship with externalizing traits. Results of specific contrasts within these tasks are presented in Supplemental Materials.

3.2.4. Results within community and enriched populations

The results of 14 studies of community samples largely paralleled the results of the full review across studies of juveniles, although studies of community samples were less likely to observe a negative relationship with total psychopathy and more likely to observe a positive relationship with externalizing traits (Fig. 4). The most common finding was a null relationship with the three psychopathy measures (≥ 66.7% of studies for each measure). A majority of studies observed a negative relationship with callous-unemotional traits (57.1%) and externalizing traits (62.5%), and a majority (62.5%) also observed a positive relationship with externalizing traits.

Compared to the results of the full review, the results from 26 studies of forensic and clinical samples showed a more consistent negative relationship with total psychopathy (Fig. 4). An equal number of studies (69.2%) found a negative relationship and a null relationship with total psychopathy. Studies most commonly found a null relationship between amygdala activity and callous-unemotional traits (81.8%) and externalizing traits (87.0%). Similar to the full review, nearly half of studies (45.5%) found a negative relationship with callous-unemotional traits and one-third (34.8%) found a negative relationship with externalizing traits. The least common finding was a positive relationship with the three psychopathy measures (≤ 21.7% of studies for each measure).

3.2.5. Results of lowest- and highest-powered studies

The studies included in the full review across studies of juveniles had a mean sample size of $N = 146.1$ (range: 22–1688). By contrast, the 14 lowest-powered functional studies of juveniles had a mean sample size

Table 2
Overlap between amygdala and peak coordinates associated with total psychopathy in studies of adults.

No.†	Cluster Label in Original Paper	MNI Coordinates x, y, z	Amygdala Overlap		
			Whole	Medial	Lateral
Negative Findings (Reduced Activity)					
1	amygdala/ parahippocampal gyrus	15, 12, -25	0.0%	0.0%	0.0%
2	amygdala/ hippocampus	-19, 9, -31	0.0%	0.0%	0.0%
3	amygdala	-18, 6, -24	0.0%	0.0%	0.0%
4	parahippocampal gyrus/amygdala	15, 3, -18	31.1%	31.1%	0.0%
5	amygdala	24, 2, -11	0.0%	0.0%	0.0%
6	amygdala	28, 2, -16	9.6%	0.0%	9.6%
7	amygdala	26, 2, -18	37.5%	15.9%	21.5%
8	amygdala	33, 0, -18	8.4%	0.0%	8.4%
9	amygdala	33, 0, -21	21.1%	0.0%	21.1%
10	amygdala	25, 0, -21	**90.8%**	**56.6%**	34.3%
11	amygdala	-22, -2, -14	**51.8%**	24.7%	27.1%
12	amygdala	-21, -3, -15	**80.5%**	**53.4%**	25.1%
13	amygdala	20, -5, -15	**98.8%**	**87.6%**	11.2%
14	amygdala	-21, -10, -14	31.5%	25.9%	5.6%
15	amygdala/ hippocampus	36, -9, -28	0.0%	0.0%	0.0%
Positive Findings (Increased Activity)					
1	amygdala	30, 2, -20	15.5%	0.0%	15.5%
2	amygdala	-26, 0, -26	**64.1%**	28.7%	35.5%
3	amygdala	-22, 0, -18	**74.3%**	**68.9%**	4.4%
4	amygdala	-30, -3, -24	**66.1%**	0.0%	**66.1%**
5	amygdala	-21, -4, -22	43.0%	43.0%	0.0%
6	amygdala	20, -4, -16	**99.2%**	**92.0%**	7.2%
7	amygdala	28, -4, -21	**83.7%**	14.3%	**69.3%**
8	amygdala	-24, -6, -21	25.1%	17.1%	8.0%
9	amygdala/corpus amygdaloideum	19, -7, -19	40.6%	40.6%	0.0%

Note: † Cluster number corresponds to the numbers assigned in Fig. 2.
Peak coordinates that overlapped ≥ 50.0% with the amygdala mask are in bold.

of $N = 35.1$ (range: 22–48). The results of the lowest-powered studies largely paralleled the results of the full review, with the exception that the lowest-powered studies were more likely to observe negative relationships with callous-unemotional traits (72.7%) and externalizing traits (66.7%; Fig. 4). These studies commonly found negative relationships with each of the three psychopathy measures (≥ 50.0% of studies for each measure). Yet, the most common finding for each psychopathy measure was a null relationship (≥ 62.5% of studies for each measure). Few of the lowest-powered studies observed positive relationships with the three psychopathy measures (≤ 33.3% of studies for each measure).

The 14 highest-powered functional studies of juveniles had a mean sample size of $N = 332.6$ (range: 74–1688). Overall, the results of the highest-powered studies mirrored the results of the full review (Fig. 4), with the following exceptions: studies of callous-unemotional traits and externalizing traits were less likely to find a negative relationship, and studies of externalizing traits were slightly more likely to find a positive relationship. The majority of studies found a null relationship with the three psychopathy measures (≥ 66.7% of studies for each measure). Half of studies (50.0%) found a negative relationship with total psychopathy, while few found a negative relationship with callous-unemotional (20.0%) or externalizing (15.4%). Nearly half of studies (46.2%) found a positive relationship with externalizing traits.

3.3. Structural studies of adults

3.3.1. Results across all studies

Table 5 displays the 21 studies of adults that analyzed the structure of the amygdala in relation to a measure of psychopathy. Fig. 6 summarizes these studies. Almost every study (20/21) analyzed GMV in the amygdala, while three studies analyzed GMC and one study analyzed surface area of the amygdala. Most studies found a null relationship between amygdala structure and the three psychopathy measures (≥ 77.8% of studies for each measure). The next most common finding was a negative relationship (≤ 31.6% of studies for each measure), followed by a positive relationship (≤ 11.1% of studies for each measure).

3.3.2. Results within community and forensic populations

Although only five studies examined amygdala structure in community samples, the results of these studies roughly mirrored the results of the full review across studies (Fig. 6). These studies most commonly found a null relationship with total psychopathy (75.0%). Of the two studies that examined relationships with interpersonal/affective traits, one (50.0%) found a null relationship and one (50.0%) found a negative relationship. We reviewed only one study that analyzed the relationship between lifestyle-antisocial traits and amygdala structure in a community sample.

Results from the 17 studies of forensic samples paralleled the results from the full review (which included only four additional studies; Fig. 6). The most common finding for each psychopathy measure was a null relationship (≥ 81.3% of studies for each measure). Similar to the full review, fewer studies found a negative relationship (≤ 31.3% of studies for each measure) or positive relationship (12.5% of studies for each measure).

3.3.3. Results of lowest- and highest-powered studies

The structural studies of adults included in the full review had a mean sample size of $N = 60.7$ (range: 27–254). By contrast, the seven lowest-powered structural studies of adults had a mean sample size of $N = 33.6$ (range: 27–38). In contrast to the results of the full review and the highest-powered studies, the results of the lowest-powered studies were uniformly null (Fig. 6). All of the lowest-powered studies found a null relationship. One study (14.3%) found a negative relationship with total psychopathy, while no studies found a negative relationship with interpersonal/affective or lifestyle/antisocial traits. None of the lowest-powered studies found a positive relationship with any of the psychopathy measures.

The seven highest-powered structural studies of adults had a mean sample size of $N = 106.2$ (range: 59–254). The results of the highest-powered studies largely mirrored the results of the full review (Fig. 6). The most common finding for each psychopathy measure was a null relationship (≥ 66.7% of studies for each measure). Few studies found a negative relationship (≤ 33.3% of studies for each measure), and none of the highest-powered studies found a positive relationship with any of the psychopathy measures.

3.4. Structural studies of juveniles

3.4.1. Results across all studies

Table 6 displays the 13 studies of juveniles that analyzed the structure of the amygdala in relation to a measure of psychopathy. Fig. 7 summarizes these studies. Every included study analyzed GMV in the amygdala, while two studies additionally analyzed GMC, one analyzed gray matter density, and one analyzed GMV asymmetry of the amygdala. The most common finding for total psychopathy (100.0%) and callous-unemotional traits (88.9%) was a null relationship. In contrast, the most common finding for externalizing traits (66.7%) was a negative relationship. Over half of studies (55.6%) found a negative relationship with callous-unemotional traits, while only one study (20.0%) found a negative relationship with total psychopathy. Few studies found a positive relationship between amygdala structure and the three psychopathy measures among juveniles (≤ 16.7% of studies for each measure).

3.4.2. Results within community and enriched populations

Only five studies examined amygdala structure in community samples of juveniles. The results of these few studies were uniformly null (Fig. 7). We reviewed no studies that examined amygdala structure in

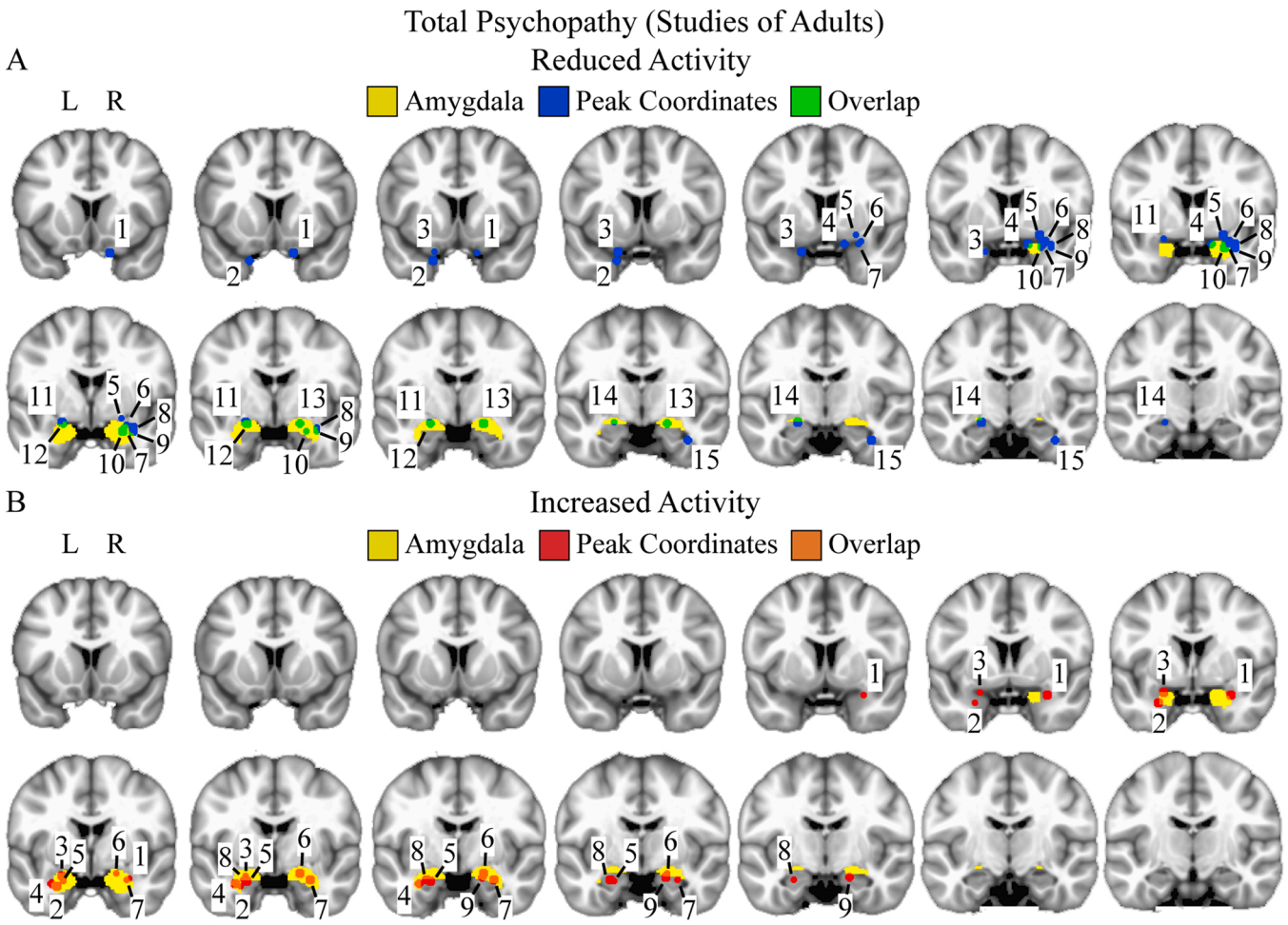

Fig. 3. Peak coordinates that were A) negatively related and B) positively related to total psychopathy in studies of adults. In each panel, each peak coordinate is labeled numerically, starting with the most anterior coordinate. Every other coronal slice is displayed from y = 13 (upper left, most anterior) to y = −13 (lower right, most posterior).

relation to externalizing traits in a community sample of juveniles.

Compared to the results of the full review, negative relationships with callous-unemotional traits made up a higher proportion of findings from the eight studies of forensic and clinical samples (Fig. 7). An equal number of studies found a negative relationship (83.3%) and a null relationship (83.3%) with callous unemotional-traits, while more studies found a negative relationship (66.7%) than a null relationship (50.0%) with externalizing traits. Three studies (100.0%) found a null relationship and one study (33.3%) found a negative relationship with total psychopathy. Few studies found a positive relationship with the three psychopathy measures (≤ 16.7% of studies for each measure).

3.4.3. Results of lowest- and highest-powered studies

The structural studies of juveniles included in the full review had a mean sample size of $N = 261.8$ (range: 29–2000). By contrast, the five lowest-powered structural studies of juveniles had a mean sample size of $N = 59.8$ (range: 29–89). The results of the lowest-powered studies were mostly null (Fig. 7). For total psychopathy (100.0%) and callous-unemotional traits (100.0%), the most common finding was a null relationship. Two studies (100.0%) found a negative relationship with externalizing traits, while no studies found a negative relationship with total psychopathy or callous-unemotional traits. None of the lowest-powered studies found a positive relationship with any of the psychopathy measures.

The five highest-powered structural studies of juveniles had a mean sample size of $N = 547.8$ (range: 138–2000). Only one of the highest-powered studies analyzed total psychopathy or externalizing traits; we will not summarize these findings here. For callous-unemotional traits, the most common finding was a null relationship (75.0%), followed by a negative relationship (50.0%; Fig. 7). No studies found a positive relationship between amygdala structure and callous-unemotional traits.

4. Discussion

Etiological theories have highlighted the amygdala as a key neural correlate of psychopathy. These theories propose that reduced amygdala activity and volume is related to attenuated experience of fear, disrupted pairing of threatening and non-threatening cues, and impaired shifting of attention to cues that predict reward or threat. The current systematic review has established the most comprehensive picture to date of evidence for amygdala dysfunction in psychopathy. Three primary findings arose from the review. First, studies of adults and juveniles most commonly found a null relationship between psychopathy and amygdala gray matter volume or task-based activity. Second, lower statistical power (based on sample size) appeared to increase the likelihood of observing significant relationships between psychopathy and the amygdala. Third, many reported peak coordinates did not fall within the amygdala, nor did peak coordinates cluster consistently in the medial or lateral amygdala. Here we discuss these primary findings and offer recommendations for the field of psychopathy.

Null relationships between psychopathy and amygdala structure and function predominated in the literature. This finding challenges

Table 3
Studies that analyzed task-based activity in the amygdala in relation to psychopathy in juveniles.

Study	Pop.	N	Assessment	Task	Relationship Between Amygdala Activity and Psychopathy Measure		
					Total	Callous-Unemotional	Externalizing
(Aggensteiner et al., 2020)	Cl	166	ICU, K-SADS, CBCL	Prototypical facial expressions	–, Null		+, Null
(Dotterer et al., 2017)	Co	232	SRD, APSD	Prototypical facial expressions		Null	+, Null
(Fairchild et al., 2014)	Cl	22	K-SADS, YPI, ICU	Prototypical facial expressions	Null	Null	–, Null
(Hyde et al., 2016)	Co	167	SRD, SCID, APSD	Prototypical facial expressions		–, Null	–, Null
(Ibrahim et al., 2019)	Cl	57	CBCL, ICU	Prototypical facial expressions		–	+
(Jones et al., 2009)	Co	30	SDQ, APSD	Prototypical facial expressions	–		
(Lozier et al., 2014)	Co	46	SDQ, CBCL, ICU	Prototypical facial expressions	Null	–, Null	+, Null
(Marsh et al., 2008)	Co	36	APSD, PCL-YV, YPI	Prototypical facial expressions	Null		
(Rhoads et al., 2020)	Cl	30	ICU	Prototypical facial expressions		–, Null	
(Sebastian et al., 2014)	Co	34	CASI-4R, ICU	Prototypical facial expressions	+, Null		–, +
(Sebastian et al., 2021)	Co	52	CASI-4R, ICU	Prototypical facial expressions	Null		–
(Viding et al., 2012)	Co	46	CASI-4R, ICU	Prototypical facial expressions	–	–	
(von Polier et al., 2020)	Cl	29	K-SADS, APSD	Prototypical facial expressions		–, Null	–, Null
(White et al., 2012)	Cl	32	K-SADS, APSD	Prototypical facial expressions	–	–, Null	Null
(Klapwijk et al., 2016a)	F	56	K-SADS, ICU	Prototypical facial expressions	–, Null	Null	
(Hawes et al., 2020)	Co	1161	K-SADS, CBCL, SDQ	Reward processing	+, Null		+, Null
(Veroude et al., 2016)	Cl	328	ICU	Reward processing		Null	
(Byrd et al., 2018)	Cl	64	APSD, CBCL	Reward/punishment processing	–, Null	–, Null	–, Null
(Cohn et al., 2015)	F	127	DISC-IV, YPI	Reward/punishment processing		Null	+, Null
(Huang et al., 2019)	Co	29	CBCL, ICU	Reward/punishment processing		Null	–, Null
(Schwenck et al., 2017)	Cl	43	FBB-SSV, ICU	Reward/punishment processing		–, Null	Null
(White et al., 2016a)	Cl	72	CBCL, ICU	Reward/punishment processing		Null	Null
(Zhang et al., 2021a)	Cl	178	ICU	Reward/punishment processing		Null	Null
(Byrd et al., 2021)	Cl	1688	SDQ, CBCL	Punishment processing	Null		Null
(Cohn et al., 2013)	Cl	74	DISC-IV, YPI	Fear conditioning		+, Null	+, Null
(Fanti et al., 2020)	F	136	ICU	Fear conditioning	–	Null	Null
(White et al., 2018)	Cl	58	K-SADS, ICU	Threat processing		Null	–
(Zhang et al., 2021b)	Cl	105	SDQ, ICU	Threat processing		Null	Null
(Gao et al., 2019)	Co	35	ICU	Empathy, Theory of Mind		Null	
(Sebastian and McCrory, 2012)	Co	31	CASI-4R, ICU	Empathy, Theory of Mind		–	–, +, Null
(O'Nions et al., 2014)	Co	48	CASI-4R, ICU	Theory of Mind	Null		
(Cardinale et al., 2018)	Cl	48	SDQ, CBCL, ICU	Reading emotional statements	–, Null	–	–, Null
(Klapwijk et al., 2016b)	F	65	K-SADS, ICU	Reading emotional statements	Null		Null
(Hwang et al., 2016)	Cl	63	K-SADS, ICU	Viewing emotional images	–, Null	Null	Null
(Thornton et al., 2017)	Cl	49	K-SADS, ICU	Viewing faces, emotional images		–, Null	–, Null
(Fehlbaum et al., 2018)	Cl	78	YPI, DSM-5	Viewing numbers, emotional images	Null		+, Null
(Harenski et al., 2014b)	F	111	K-SADS, PCL-YV, ICU	Moral judgment	–	–	–, Null
(Yoder et al., 2016)	Cl	106	DISC-IV, ICU	Viewing violent scenes		Null	Null
(White et al., 2016b)	56	ICU	Economic decision-making	–			
(Vincent et al., 2018)	F	54	PCL-YV	Viewing drug images	–	–	–

Note: Multiple relationships are listed for studies that analyzed multiple experimental contrasts or comparisons and yielded mixed results. Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: – = negative relationship, + = positive relationship, Pop. = population, F = forensic, Cl = clinical, Co = Community, APSD = Antisocial Process Screening Device (Frick and Hare, 2001), CASI-4R = Child and Adolescent Symptom Inventory-4R (Gadow and Sprafkin, 2009), CBCL = Child-Behavior Checklist (Achenbach, 2009), DISC-IV = Diagnostic Interview Schedule for Children Version IV (Shaffer et al., 2000), DSM-5 = Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013), FBB-SSV = Fremdbeurteilungsbogen für Störungen des Sozialverhaltens (Döpfner et al., 2009), ICU = Inventory of Callous-Unemotional Traits (Frick, 2004), K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia (Kaufman et al., 1997), PCL-YV = Psychopathy Checklist-Youth Version (Forth and Kosson, 2003), SCID = Structured Clinical Interview for DSM (First et al., 2012), SDQ = Strengths and Difficulties Questionnaire (Goodman, 1997), SRD = Self-Report of Delinquency Questionnaire (Elliott et al., 1985), YPI = Youth Psychopathic Traits Inventory (Andershed et al., 2007).

theoretical frameworks that position the amygdala at the center of a neural circuit that is disrupted in psychopathy. Null relationships between psychopathy and the amygdala are rarely, if ever, cited in the reviews and discussions of published papers. In fact, the tendency to disproportionately cite significant findings and ignore null findings is highly prevalent in the neuroimaging literature on psychopathy (Jalava et al., 2021). It will be necessary to grapple with this preponderance of null findings in order to advance the field's understanding of the neurobiology of psychopathy. We have begun by considering the following explanations for the null findings: 1) anomalous amygdala activity is context-dependent, 2) methodological limitations have routinely impeded the ability to detect amygdala anomalies and/or resulted in spurious findings, and 3) opposing relationships with interpersonal/affective traits and lifestyle/antisocial traits have suppressed observable relationships with total psychopathy. We consider each of these explanations in turn.

Anomalous amygdala activity in psychopathy may be context-dependent, as several authors have previously suggested (Hoppenbrouwers et al., 2016; Larson et al., 2013; Glass and Newman, 2006). This hypothesis is consistent with the view that amygdala activity is context-dependent even in healthy brains. This view proposes that the amygdala may switch its affective mode (positive or negative, appetitive or avoidant) across situations (Berridge, 2019). This is not a unique characteristic of the amygdala. Across the brain, varying populations of neurons can represent instances of the same category (e.g., fear) in different contexts (known as "degeneracy;" Barrett, 2017; Edelman and Gally, 2001). The studies we reviewed varied in the context induced by the experimental task. Thus, we examined consistency within experimental tasks to assess the context specificity of amygdala anomalies in psychopathy. We failed to identify a context (i.e., experimental task) in which amygdala anomalies were reliably observed. In other words, null findings predominated in studies of prototypical facial expressions tasks, moral tasks, empathy tasks, and reward/punishment tasks. Within these tasks, we also failed to identify experimental contrasts in which

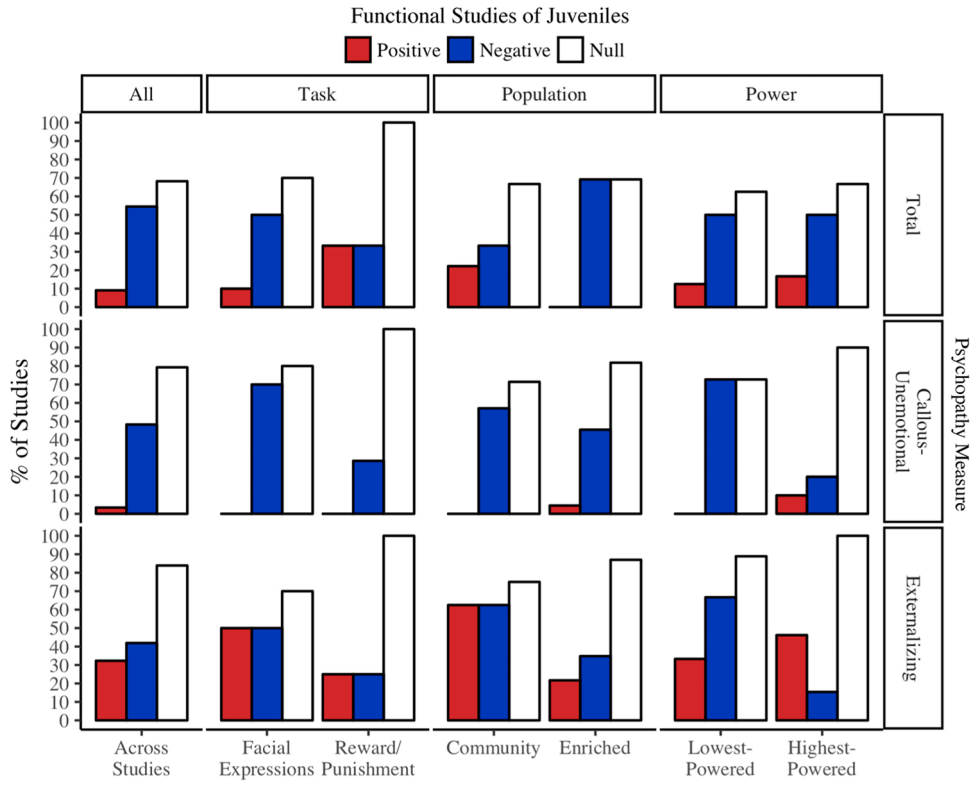

Fig. 4. Summary of studies that analyzed task-based activity in the amygdala in relation to psychopathy in juveniles.

Table 4
Overlap between amygdala and peak coordinates associated with total psychopathy in studies of juveniles.

No.†	Cluster Label in Original Paper	MNI Coordinates x, y, z	Amygdala Overlap		
			Whole	Medial	Lateral
Negative Findings (Reduced Activity)					
1	amygdala	31, 7, -33	0.0%	0.0%	0.0%
2	amygdala	-13, 2, -30	0.8%	0.8%	0.0%
3	amygdala	-10, 2, -29	0.0%	0.0%	0.0%
4	amygdala	27, 2, -29	36.7%	6.0%	30.7%
5	amygdala	-12, -1, -16	22.7%	22.7%	0.0%
6	amygdala	20, -2, -22	**80.9%**	**80.9%**	0.0%
7	amygdala	-18, -3, -21	**67.7%**	**67.7%**	0.0%
8	amygdala	-15, -3, -21	**67.3%**	**67.3%**	0.0%
9	amygdala	21, -4, -35	0.0%	0.0%	0.0%
10	amygdala	-22, -6, -19	42.6%	40.2%	2.4%
11	amygdala	-27, -6, -12	30.7%	0.0%	30.7%
12	amygdala	-16, -5, -20	48.2%	48.2%	0.0%
13	amygdala	24, -6, -12	**65.7%**	28.3%	37.4%
14	amygdala	-20, -8, -14	**55.4%**	44.2%	11.2%
15	amygdala	-26, -8, -30	0.0%	0.0%	0.0%
16	amygdala/lentiform nucleus	-19, -13, -3	0.0%	0.0%	0.0%
Positive Findings (Increased Activity)					
1	amygdala	20, -10, -14	46.6%	46.6%	0.0%

Note: † Cluster number corresponds to the numbers assigned in Fig. 3.
Peak coordinates that overlapped ≥ 50.0% with the amygdala mask are in bold.

amygdala anomalies were reliably observed (Supplemental Materials). Although studies of moral tasks and empathy tasks were slightly more likely to find reduced amygdala activity compared to the full review across a variety of tasks, null findings were still more prevalent than positive or negative findings. Notably, prototypical facial expressions tasks and empathy tasks have been specified by prior theories as the contexts in which reduced amygdala activity is likely to be observed (Blair, 2005; Kiehl, 2006). In sum, if anomalous amygdala activity in psychopathy is context-dependent, the tasks commonly employed in the existing literature have not captured this context specificity.

Methodological limitations may have contributed to null or spurious findings. We examined the influence of two methodological characteristics: power and population. Low power due to small sample size is a well-known limitation of MRI studies that inflates observed effect sizes and contributes to low reproducibility of results (Button et al., 2013; Poldrack et al., 2017; Szucs and Ioannidis, 2020; Turner et al., 2018). The highest-powered and lowest-powered studies observed relatively similar rates of null relationships. However, the lowest-powered studies were more likely to observe reduced amygdala activity in relation to total psychopathy and interpersonal/affective traits in adults and callous-unemotional traits in juveniles. In particular, the lowest-powered studies of juveniles were over three times more likely than the highest-powered studies to observe reduced amygdala activity in relation to callous-unemotional traits. The lowest-powered studies were conversely *less* likely to observe significant relationships with measures of amygdala structure. Critically, even the studies we reviewed with the largest sample sizes were likely highly underpowered. MRI studies associating brain structure or function with a clinical phenotype such as psychopathy may require thousands of subjects to achieve sufficient power (Marek et al., 2022). We reviewed only three studies with sample sizes greater than 1000. This underscores the power limitations of the existing fMRI studies of psychopathy, which have likely hindered replications and inflated observed effect sizes.

Alternatively, studies of the community population with generally low levels of psychopathy may have yielded more null findings. This appeared to be the case in the literature on juveniles but not the literature on adults. In the literature on juveniles, studies of community samples were less likely to find reduced amygdala activity in relation to total psychopathy than studies of enriched (i.e., forensic and clinical) samples. Conversely, in the literature on adults, studies of community samples were *more* likely than studies of forensic samples to find reduced amygdala activity. In conclusion, methodological limitations seem to have contributed at least in part to the consistent null findings across studies, as well as to observations of reduced amygdala activity.

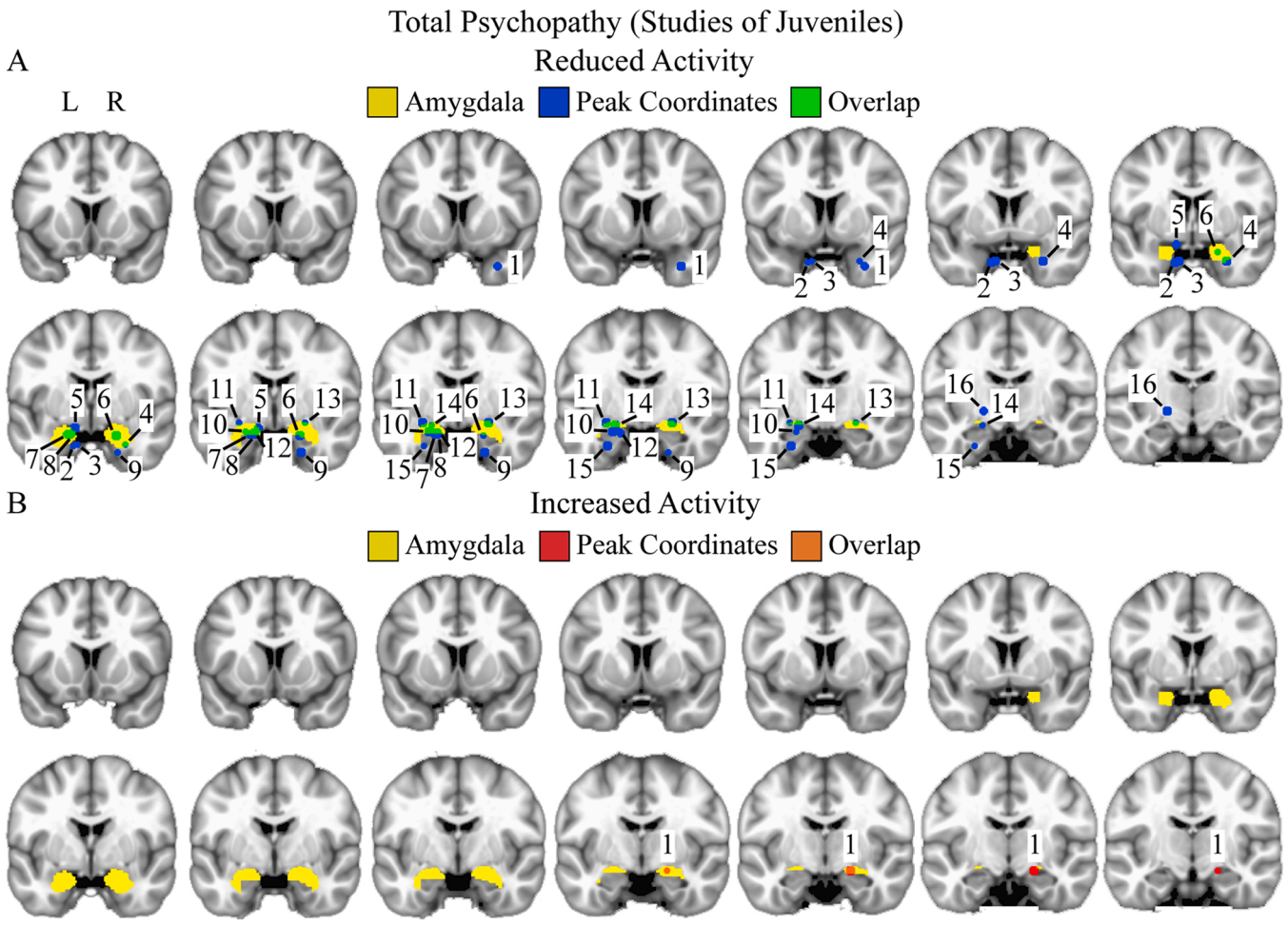

Fig. 5. Peak coordinates that were A) negatively related and B) positively related to total psychopathy in studies of juveniles. In each panel, each peak coordinate is labeled numerically, starting with the most anterior coordinate. Every other coronal slice is displayed from y = 13 (upper left, most anterior) to y = −13 (lower right, most posterior).

Opposing relationships with interpersonal/affective traits and lifestyle/antisocial traits may have suppressed observable relationships with total psychopathy. Such suppressor effects have previously been observed in a study of amygdala activity among juveniles (Lozier et al., 2014). In this previous study, the relationship between total psychopathy and amygdala activity was null. In contrast, the two constituent clusters of psychopathic traits showed opposing relationships: callous-unemotional traits were negatively related and externalizing traits were positively related with amygdala activity in the same experimental contrast. How common was this pattern of findings across studies? Of the 134 total studies we reviewed, only two found a null relationship with total psychopathy and opposing relationships with the constituent clusters of traits in the same experimental contrast (Lozier et al., 2014; Gordon et al., 2004). Three additional studies found opposing relationships with the constituent clusters of traits in the same experimental contrast but did not analyze total psychopathy, providing partial evidence for suppressor effects (Carré et al., 2013; Sebastian and McCrory, 2012; Cohn et al., 2016). Therefore, by and large, suppressor effects did not appear to drive the null relationships between total psychopathy and amygdala structure or function.

The third primary finding of the systematic review was that many peak coordinates of amygdala anomalies related to psychopathy did not fall within the anatomical bounds of the amygdala. Each of these significant clusters was labeled "amygdala" in the original paper. Imprecise labeling has possibly inflated the impression that the amygdala is a reliable neural correlate of psychopathy. As an illustration, consider the 15 peak coordinates negatively related to total psychopathy in adults. Five of these peak coordinates (from four studies) overlapped 0.0% with the amygdala (Table 2). These four studies have left a substantial mark in the literature, having together been cited over 2300 times. Two of these studies provided comprehensive labels of significant clusters (e.g., "amygdala/hippocampus"), while the other two studies simply labeled these clusters "amygdala." The problem associated with the latter is clear: papers citing these original studies may accept the "amygdala" label at face value. The problem associated with the former is that papers citing these original studies are likely to simply label these clusters "amygdala," rather than reporting the comprehensive label. A quick review of papers citing these four original studies provides several examples of this (Siever, 2008; Kirsch et al., 2005; Coccaro et al., 2007; Vieira and Marsh, 2014; Pardini et al., 2014). In addition, six of the peak coordinates (from six studies) negatively related to total psychopathy in adults overlapped 0.1–49.9% with the amygdala, suggesting the most significant relationship between BOLD activity and psychopathy was observed in structures adjacent to the amygdala in these cases. Taken together, and in conjunction with the null findings discussed above, the peak coordinates suggest that the amygdala is a less reliable neural correlate of psychopathy than previously described.

Of course, the significant clusters associated with these peak coordinates may have contained voxels within the amygdala. It is difficult to assess this possibility without access to the original data sets. If this is the case, how should we interpret findings of reduced activity in a few (e.g., 1–2) voxels in the amygdala? The preprocessing step of spatial

Table 5
Studies that analyzed structure of the amygdala in relation to psychopathy in adults.

Study	Pop.	N	Assessment	Structural Measure	Relationship Between Amygdala Structure and Psychopathy Measure		
					Total	Interpersonal/ Affective	Lifestyle/ Antisocial
(Bell et al., 2022)	F	34	PCL-R	GMV	Null	Null	Null
(Bertsch et al., 2013)	F	39	PCL-R	GMV	Null		
(Boccardi et al., 2011)	F	51	PCL-R	GMV	–, +		
(Calzada-Reyes et al., 2021)	F	132	PCL-R	GMV	Null	Null	Null
(Cope et al., 2012)	F	66	PCL-R	GMV	Null	Null	Null
(Gregory et al., 2012)	F	66	PCL-R	GMV	Null		
(Hofhansel et al., 2020)	F	27	PCL-R	GMV	Null	Null	Null
(Kolla et al., 2014)	F	37	PCL-R	GMV	Null		
(Leutgeb et al., 2015)	F	40	PCL-R	GMV		Null	Null
(Müller et al., 2008)	F	34	PCL-R	GMV	Null		
(Nummenmaa et al., 2021) †	F; C	38; 100	PCL-R; LSRP	GMV	Null		
(Pardini et al., 2014)	F	56	SRP	GMV	–, Null	–, Null	–
(Schiffer et al., 2011)	F	51	PCL:SV	GMV	+	+, Null	+, Null
(Tiihonen et al., 2008)	F	51	PCL-R	GMV	Null		
(Vieira et al., 2014b)	C	35	PPI-R, TriPM	GMV	Null		
(Yang et al., 2009)	C	59	PCL-R	GMV	–	–	–
(Yang et al., 2010)	F	53	PCL-R	GMV	–, Null		
(Kolla et al., 2017)	F	38	PCL-R	GMV, SA	–, Null		
(De Oliveira-Souza et al., 2008)	C	30	PCL:SV	GMV, GMC	Null		
(Ermer et al., 2012)	F	254	PCL-R	GMV, GMC	–	Null	Null
(Bobes et al., 2013)	C	60	PCL-R, LSRP	GMC		Null	

Note: Multiple relationships are listed for studies that analyzed multiple experimental contrasts or comparisons and yielded mixed results. Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: – = negative relationship, + = positive relationship, Pop. = population, F = forensic, C = community, GMV = gray matter volume, GMC = gray matter concentration, SA = surface area, LSRP = Levenson Self-Report Psychopathy Scale (Levenson et al., 1995), PCL-R = Psychopathy Checklist-Revised (Hare, 2003), PCL: SV = Psychopathy Checklist: Screening Version (Hart et al., 1995), PPI-R = Psychopathic Personality Inventory-Revised (Lilienfeld and Widows, 2005), SRP = Self-Report Psychopathy Scale (Paulhus et al., 2015)(1, TriPM = Triarchic Psychopathy Measure (Patrick, 2010).
†One study separately analyzed a forensic sample and a community sample. Characteristics of each sample are separated by a semi-colon.

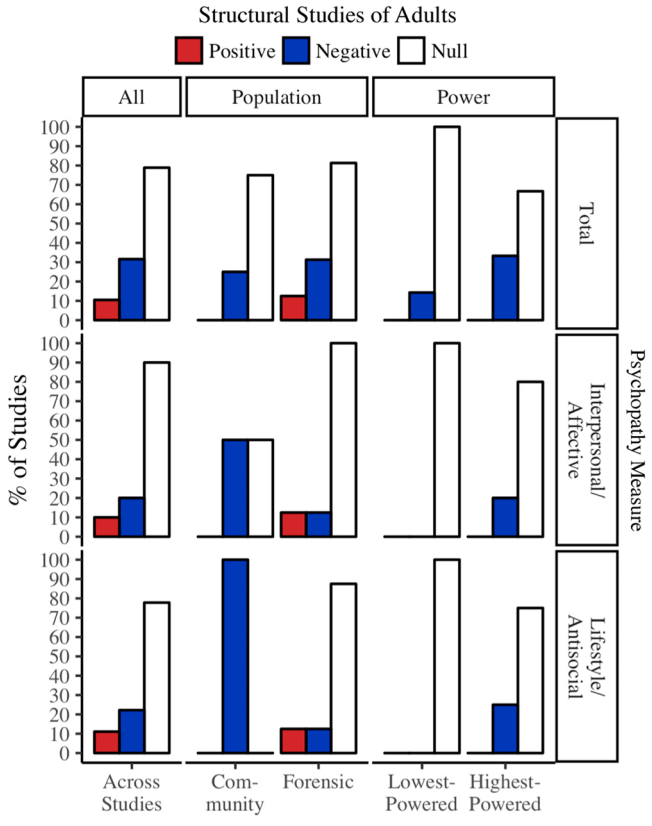

Fig. 6. Summary of studies that analyzed gray matter structure in the amygdala in relation to psychopathy in adults. Note that we reviewed only one study that examined the relationship between lifestyle/antisocial traits and amygdala structure in a community sample.

smoothing is known to affect the significance of a given voxel by including the values of neighboring voxels (Morawetz et al., 2008). Notably, although a small smoothing kernel (e.g., 4 mm) is recommended for analyzing subcortical structures (Morawetz et al., 2008; Poldrack et al., 2017), we reviewed a number of studies that used a large smoothing kernel (e.g., ≥ 8 mm; Birbaumer et al., 2005; Kiehl et al., 2001; Seara-Cardoso et al., 2016a). Spatial smoothing with large kernels may have led to the inclusion of a few amygdala voxels in a cluster with peak coordinates outside the amygdala. Furthermore, peak coordinates did not cluster consistently in the medial or lateral amygdala. This result does not support the hypothesis that psychopathy is related to dysfunction in the basoleateral but not central subnucleus of the amygdala (Moul et al., 2012).

We note the following limitations of this systematic review. Studies were selected for analyzing psychopathy in relation to amygdala structure or function. Other papers have systematically reviewed broader neural correlates of psychopathy and antisocial behavior in adults (Murray et al., 2018; Johanson et al., 2020) and conduct disorder in juveniles (Noordermeer et al., 2016). Functional connectivity of the amygdala was also outside the scope of this review. Additionally, our examination of the spatial consistency of amygdala findings was limited to peak coordinates (from 50 out of 102 task-based fMRI studies), which do not capture the full spatial extent of the significant clusters observed in the original studies. Future studies might use image-based meta--analysis to test effect sizes at each voxel in the amygdala (Salimi--Khorshidi et al., 2009). This method, the "gold standard" for neuroimaging meta-analyses, would provide a clearer picture of the spatial consistency of anomalous amygdala activity in psychopathy. However, we note that peak coordinates serve as the unit of analysis for other common neuroimaging meta-analyses (Wager et al., 2007; Eickhoff et al., 2016). Furthermore, we used sample size as an approximation of study power, although many factors influence power in fMRI studies, including the spatial scope of analyses (whole-brain vs. ROI) and number of trials (Marek et al., 2022; Turner and Miller, 2013). Our systematic review was also unable to consider the standard deviations of

Table 6
Studies that analyzed structure of the amygdala in relation to psychopathy in juveniles.

Study	Pop.	N	Assessment	Structural Measure	Relationship Between Amygdala Structure and Psychopathy Measure		
					Total	Callous-Unemotional	Externalizing
(Bolhuis et al., 2019)	Co	2000	ICU	GMV		Null	
(Caldwell et al., 2019)	F	269	ICU	GMV		–, Null	
(Cardinale et al., 2019)	Cl	93	CBCL, ICU	GMV		–, Null	–, Null
(Fairchild et al., 2013)	F	84	K-SADS, YPI	GMV	Null	Null	–, Null
(Gao et al., 2020)	Cl	138	SDQ, APSD	GMV		–	Null
(Ibrahim et al., 2021)	Cl	138	CBCL, ICU	GMV		–, +, Null	–
(Raschle et al., 2018)	Co	189	ICU	GMV		Null	
(Sebastian et al., 2016)	Co	89	CASI-4R, ICU	GMV	Null		
(Wallace et al., 2014)	F	49	K-SADS, ICU	GMV	Null		–
(Lam et al., 2021)	Co	29	ICU	GMV Asymmetry		Null	
(Cohn et al., 2016)	F	134	DISC-IV, YPI	GMV, GMC		–, Null	+
(De Brito et al., 2009)	Co	48	SDQ, APSD	GMV, GMC	Null		
(Steele et al., 2017)	F	143	PCL-YV	GMV, GMD	–, Null		

Note: Multiple relationships are listed for studies that analyzed multiple experimental contrasts or comparisons and yielded mixed results. Blank cells indicate that the study did not test the relevant relationship.
Abbreviations: – = negative relationship, + = positive relationship, Pop. = population, F = forensic,Co = community, Cl = clinical, GMV = gray matter volume, GMC = gray matter concentration, GMD = gray matter density, APSD = Antisocial Process Screening Device (Frick and Hare, 2001), CASI-4R = Child and Adolescent Symptom Inventory-4R (Gadow and Sprafkin, 2009), CBCL = Child-Behavior Checklist (Achenbach, 2009), DISC-IV = Diagnostic Interview Schedule for Children Version IV (Shaffer et al., 2000), ICU = Inventory of Callous-Unemotional Traits (Frick, 2004), K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia (Kaufman et al., 1997), PCL-YV = Psychopathy Checklist-Youth Version (Forth and Kosson, 2003), SDQ = Structured Clinical Interview for DSM (First et al., 2012), YPI = Youth Psychopathic Traits Inventory (Andershed et al., 2007).

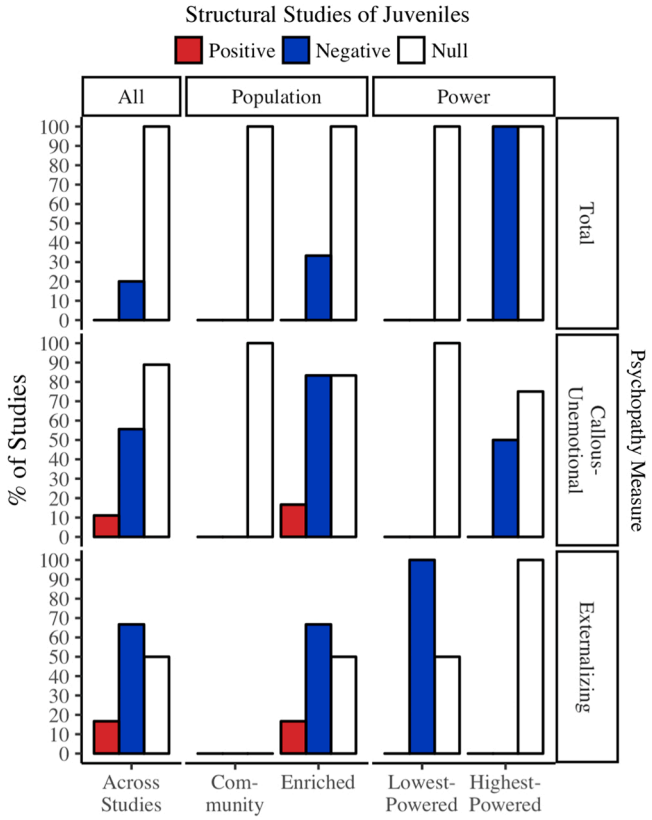

Fig. 7. Summary of studies that analyzed gray matter structure in the amygdala in relation to psychopathy in juveniles. Note that only one of the highest-powered studies analyzed the relationship with total psychopathy and with externalizing traits. We reviewed no studies that examined the relationship between amygdala structure and externalizing traits in a community sample.

the results. Finally, although the current review might have been bolstered by estimating the Bayesian probabilities of detecting a relationship between the amygdala and psychopathy, fMRI studies rarely report the effect sizes of null (i.e., non-significant) findings, preventing the calculation of comprehensive Bayesian statistics. Thus, the null findings should be interpreted as a failure of the original studies to reject the null hypothesis using frequentist statistical testing.

We conclude with recommendations for the field of psychopathy neuroimaging. First, we recommend rigorous labeling of significant clusters of voxels. This step is crucial for advancing neurobiological theory. We recommend using anatomical atlases to comprehensively label the brain structures in which significant voxels were observed. Published guidelines recommend reporting how the label was identified (Poldrack et al., 2008). Relatedly, we recommend testing the robustness of significant clusters that only contain a few voxels within the amygdala. Do the voxels within the amygdala remain significant when, for example, a smaller smoothing kernel (i.e., 4 mm) is used? Second, we recommend optimizing study power by analyzing data sets of hundreds (Turner et al., 2018) if not thousands (Marek et al., 2022) of participants. The latter would require the use of a large, open source data set such as the ABCD study (https://nda.nih.gov/abcd). Third, we reiterate the recommendations made by Koenigs et al. (2011) for addressing heterogeneous methods in the psychopathy neuroimaging literature, which may contribute to inconsistent findings across studies. That is, researchers should consider the potential effects of study population and psychopathy assessment when designing studies and interpreting data. Future studies might also examine possible amygdala differences between psychopathic subtypes. Finally, and perhaps most importantly, we recommend expanding the focus of neuroimaging studies of psychopathy from examinations of neural modules, such as the amygdala, toward examinations of neural networks that support core cognitive functions. A number of studies have already done so, with promising results (Philippi et al., 2015; Pujol et al., 2012; Contreras-Rodríguez et al., 2015; Dotterer et al., 2020; Espinoza et al., 2018, 2019; Tillem et al., 2019; Dotterer, 2018; Lindner et al., 2018).

5. Conclusions

The extant neuroimaging data does not conclusively support a relationship between the amygdala and psychopathy. A deeper understanding of amygdala function in healthy human brains coupled with more rigorous and sophisticated neuroimaging methodology may yield greater insight on the neurobiology of psychopathy in the future.

Acknowledgments

The authors declare no competing financial interests.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2022.104875.

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