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FEATURE REVIEW

Investigating the neural correlates of psychopathy: a critical review

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In recent years, an increasing number of neuroimaging studies have sought to identify the brain anomalies associated with psychopathy. The results of such studies could have significant implications for the clinical and legal management of psychopaths, as well as for neurobiological models of human social behavior. In this article, we provide a critical review of structural and functional neuroimaging studies of psychopathy. In particular, we emphasize the considerable variability in results across studies, and focus our discussion on three methodological issues that could contribute to the observed heterogeneity in study data: (1) the use of between-group analyses (psychopaths vs non-psychopaths) as well as correlational analyses (normal variation in 'psychopathic' traits), (2) discrepancies in the criteria used to classify subjects as psychopaths and (3) consideration of psychopathic subtypes. The available evidence suggests that each of these issues could have a substantial effect on the reliability of imaging data. We propose several strategies for resolving these methodological issues in future studies, with the goal of fostering further progress in the identification of the neural correlates of psychopathy.
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Human brain imaging techniques, such as magnetic resonance imaging, have become an indispensable means for investigating the neurobiological substrates of psychiatric and psychological disorders. In recent years, the use of neuroimaging in psychopathy research has become increasingly common. The potential implications of characterizing the neural correlates of psychopathy are far-reaching. Clinically, such knowledge could be used to aid in the diagnosis of the disorder and perhaps in the identification of neural targets for treatment. In the legal domain, neuroimaging data could possibly inform questions of culpability, likelihood of future offense and prospects for rehabilitation. However, structural and functional imaging studies have not yet revealed consistent neural correlates of psychopathy. The goal of this article is threefold: (1) to briefly summarize the extant neuroimaging data on psychopathy, (2) to identify a number of methodological inconsistencies that may contribute to the observed heterogeneity in the data and (3) to make constructive suggestions regarding potential strategies for remediation of methodological inconsistencies in future studies.

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Before summarizing the neuroimaging results, we first outline the scope of the studies we evaluated for this article. We specifically examined original published reports of human neuroimaging data, wherein the authors make direct conclusions about the neural correlates of psychopathy in adults (in particular, neuroimaging reports with 'psychopathy,' 'psychopaths' or 'psychopathic' in the title; see Table 1). This approach omits two important related lines of research, which we briefly mention here. One is the study of the neural correlates of antisocial traits commonly associated with, but not limited to, psychopathy. Examples include violence,[1,2] antisocial personality disorder,[3,4] aggressive/impulsive behavior[5] and pathological lying.[6] Although these traits may commonly overlap with psychopathy, none are unique to psychopathy. Accordingly, neuroimaging findings associated with these traits may not specifically inform the neural basis of psychopathy and so we omit further mention of such studies in this review. (For a recent review on neuroimaging of antisocial behavior, see Yang and Raine.[7]) The other line of research omitted here is the neuroimaging of children and adolescents with psychopathic tendencies (for example, De Brito *et al.*,[8] Jones *et al.*[9] and Marsh *et al.*[10]). Research in children and adolescents is of course critical for understanding the development of antisocial behavior. However, the comparison of imaging data from adult and child/adolescent studies can be challenging for a number of

Table 1 Neuroimaging studies of 'psychopathy'

First author	*Year*	*Title*	*Type of imaging*	*Type of analysis*	*PCL-R cutoff for* P	*Mean PCL-R for* P*'s*	P *sample size*
Birbaumer	2005	Deficient fear conditioning in *psychopathy*: a functional magnetic resonance imaging study	F	BG	15	24.9	10
Boccardi	2009	Abnormal hippocampal shape in offenders with *psychopathy*	S	BG	30	34.6	12
Buckholtz	2010	Mesolimbic dopamine reward system hypersensitivity in individuals with *psychopathic* traits	F	C/R	N/A	N/A	N/A
Craig	2009	Altered connections on the road to *psychopathy*	S	BG, C/R	25	28.4	9
Deeley	2006	Facial emotion processing in criminal *psychopathy*. Preliminary functional magnetic resonance imaging study	F	BG	25	29.3	6
Glenn	2009	The neural correlates of moral decision-making in *psychopathy*	F	C/R	N/A	N/A	N/A
Glenn	2010	Increased volume of the striatum in *psychopathic* individuals	S	BG	23	27.2	22
Gordon	2004	Functional differences among those high and low on a trait measure of *psychopathy*	F	BG	N/A	N/A	N/A
Intrator	1997	A brain imaging (single photon emission computerized tomography) study of semantic and affective processing in *psychopaths*	F	BG	25	29.9	8
Kiehl	2001	Limbic abnormalities in affective processing by criminal *psychopaths* as revealed by functional magnetic resonance imaging	F	BG	24	32.8	8
Kiehl	2004	Temporal lobe abnormalities in semantic processing by criminal *psychopaths* as revealed by functional magnetic resonance imaging	F	BG	29	32.8	8
Laakso	2001	*Psychopathy* and the posterior hippocampus	S	C/R	N/A	N/A	N/A
Muller	2003	Abnormalities in emotion processing within cortical and subcortical regions in criminal *psychopaths*: evidence from a functional magnetic resonance imaging study using pictures with emotional content	F	BG	31	36.8	6
Muller	2008	Gray matter changes in the right superior temporal gyrus in criminal *psychopaths*. Evidence from voxel-based morphometry	S	BG	29	33.4	17
Muller	2008	Disturbed prefrontal and temporal brain function during emotion and cognition interaction in criminal *psychopathy*	F	BG	28	30.5	10
Raine	2003	Corpus callosum abnormalities in *psychopathic* antisocial individuals	S	BG, C/R	23	30.3	15
Rilling	2007	Neural correlates of social cooperation and non-cooperation as a function of *psychopathy*	F	C/R	N/A	N/A	N/A
Veit	2009	Aberrant social and cerebral responding in a competitive reaction time paradigm in criminal *psychopaths*	F	C/R	N/A	N/A	N/A
Yang	2009	Localization of deformations within the amygdala in individuals with *psychopathy*	S	BG, C/R	23	28.0	27

Abbreviations: BG, between-group analysis; C/R, correlation or regression analysis; F, functional; N/A, not applicable or data not available; P, psychopathy; PCL-R, Psychopathy Checklist-Revised; S, structural.

reasons. One reason is that the diagnostic criteria for antisocial behavior in children/adolescents (such as conduct disorder) are necessarily somewhat different from the criteria for adult psychopathy, reflecting the considerable differences in life circumstances for children, adolescents and adults. A second reason is that the brain undergoes substantial structural development throughout childhood and adolescence, such that neuroimaging findings vary significantly across pre-adult age groups, even among neurologically and psychologically healthy individuals.[11] Given these important differences, we believe the child/adolescent literature warrants its own review and evaluation. (For a recent review on neuroimaging

findings related to antisocial behavior in children, see Crowe and Blair.[12])

Neuroimaging data on psychopathy: summary of results

The neuroimaging studies of psychopathy can be divided into 'structural' studies, which assess brain morphology, and 'functional' studies, which assess brain activity (Table 1). Structural neuroimaging studies associate psychopathy with a host of morphological brain abnormalities: reduced volumes of the amygdala;[13] reduced gray matter volumes in the frontal and temporal cortex, especially in the right superior temporal gyrus;[14] increased volume of the striatum;[15] increased volume of the corpus callosum;[16] reduced volume of the posterior hippocampus;[17] normal volume but abnormal shape of the hippocampus;[18] and reduced structural integrity of the uncinate fasciculus.[19] Overall these studies link psychopathy with a variety of structural abnormalities within frontal and temporal areas, involving cortical and subcortical gray matter structures as well as white-matter pathways. The identified structures have important roles in emotion and social cognition (amygdala, superior temporal cortex and uncinate fasciculus), as well as learning and memory (striatum and hippocampus). However, within this broad functional/anatomical grouping of the study results, the available structural imaging data have not yet demonstrated reliable, replicated structural abnormalities in specific brain regions.

Functional imaging studies identify brain activity associated with a particular experimental task. In psychopathy research, functional imaging studies have typically featured tasks involving social and/or emotional processing, such as fear conditioning,[20] viewing facial expressions of emotion,[21,22] moral decision-making,[23] identification of emotionally salient words,[24] recollection of emotionally salient words,[25] viewing emotionally salient scenes,[26,27] social cooperation,[28] anticipation of reward[29] and punishment administration.[30] Accordingly, many of these studies focus their analyses on emotion-related regions-of-interest, such as the amygdala.[20,22,23,25,28] However, the imaging results indicate that psychopathy is associated with abnormal activity in widespread areas of the brain, not just in those associated with emotional processing. Reduced activity has been observed in limbic and paralimbic areas, including the amygdala,[20,23,25,28] hippocampus and parahippocampal gyri,[25,26] anterior and posterior cingulate cortex,[20,25,26,28] ventral striatum[25] and insula.[20] On the other hand, reduced activity has also been observed in association areas within frontal and temporal cortices,[20,22,26–28] as well as in sensory areas such as posterior visual cortices[21,26] and parietal somatosensory cortex,[20,21] and in motor structures such as the cerebellum[21] and primary motor cortex.[21] Increased activity has been observed in frontal and temporal cortices,[24–26] nucleus accumbens,[29] as well as areas of the parietal lobe, occipital lobe, cerebellum, cingulate cortex and amygdala.[26] Taken together, these functional imaging data associate psychopathy with abnormal activity in all four lobes of the cortex (frontal, temporal, parietal and occipital), as well as several subcortical structures. As such, it is difficult to group the findings in any particular functional domain.

An intriguing observation is that, depending on the experimental context, the same brain area could be reported as either hypo- or hyper-active. For example, amygdala activity was abnormally low during fear conditioning,[20] moral decision-making,[23] social cooperation,[28] and memory for emotionally salient words,[25] but abnormally high during the viewing of certain emotionally salient scenes.[26] Similarly, ventral striatum activity was abnormally low during memory for emotionally salient words,[25] but abnormally high during reward anticipation.[29] These results suggest that neural processing abnormalities in psychopathy may be significantly context dependent. In other words, there is not yet clear evidence for a particular area being persistently hypo- or hyper-active; the functional activation data associated with psychopathy seem to depend critically on the experimenters' selection of task and stimuli.

In sum, the structural and functional abnormalities associated with psychopathy are widespread and rather variable, although regions within the frontal and temporal lobe appear to be the most commonly identified in both types of study. Given the broad array of imaging results, it is reasonable to ask whether differences in methodology could account for some of the variability in the findings. In the following sections, we highlight three methodological issues that could potentially limit the consistency and generalizability of results across the imaging studies.

Methodological issues

Two different uses of the term 'psychopathy'

One issue that could contribute to heterogeneity in the psychopathy-imaging data concerns the use of the term 'psychopathy'. In the neuroimaging literature, the term 'psychopathy' is commonly used at least two ways. In one usage, 'psychopathy' denotes the condition of being a psychopath, implying a categorical designation that corresponds to the early predominant usage of the term in the clinical literature.[31–33] In studies employing this usage the data analysis strategy typically involves between-group comparisons of neuroimaging data (that is, psychopaths vs non-psychopaths).[13–16,18–22,24–27,30,34] In the second usage, 'psychopathy' denotes the degree of psychopathy. This usage can pertain to a 'normal' sample of individuals, such as a community or university student sample, of which few, if any, would actually be diagnosed as psychopaths (for example, Gordon *et al.*,[22] Glenn *et al.*,[23] Rilling *et al.*[28] and Buckholtz *et al.*[29]). In studies employing this usage, the data analysis strategy typically involves

correlation or regression analyses between a psychopathy score and one or more neuroimaging measures.[23,28,29] (Note that the data entered into such correlational analyses may be overall psychopathy scores[23] or scores on a particular dimension or 'factor' of psychopathy, such as antisocial impulsivity[29] or the interpersonal factor.[23] Differences in the exact 'psychopathic' traits being analyzed may also contribute to heterogeneity of results regarding the neural correlates of psychopathy.) Importantly, the reported brain–behavior associations in this type of correlational analysis may depend substantially (if not entirely) on individuals within the normal range of social behavior. The implicit assumption of this correlational approach is that normal variation in certain social/affective/behavioral traits (as indexed by normal subjects' self-report scores on psychopathy questionnaires) is associated with variation in the activity of the same brain areas that are dysfunctional in severely psychopathic individuals. Although there are ample clinical and behavioral data suggesting that psychopathic traits do in fact fall along a continuum—with psychopaths representing a quantitatively greater manifestation of the traits rather than a qualitatively distinct category[35–38]—there is not yet strong evidence to support the assumption that the neurobiological data are similarly continuous.

By analogy, consider the use of neuroimaging to identify the neural correlates of depression. Studies that compare the brain activity of clinically depressed patients with psychiatrically healthy individuals have associated depression with abnormal activity in several areas of the brain, including the subgenual cingulate cortex, dorsolateral prefrontal cortex and dorsal anterior cingulate.[39–41] A separate study that correlated individual variation in the experience of negative affect with brain activity among psychiatrically healthy individuals identified an area of the ventromedial prefrontal cortex (adjacent to the subgenual cingulate), but did not identify the more dorsal frontal areas.[42] These data indicate that normal variation in a particular trait is not necessarily associated with the same brain areas that are dysfunctional in the extreme pathological manifestation of the trait. The application of this logic to psychopathy research prescribes that the identification of brain areas associated with normal variation in certain social/affective/behavioral traits should not necessarily be used as evidence for the dysfunction of these areas in severely psychopathic individuals.

As a specific example of how this issue may complicate the interpretation of psychopathy neuroimaging data, consider the findings of Kiehl *et al*.[25] and Buckholtz *et al*.[29] Comparing a group of criminal psychopaths with a group of criminal non-psychopaths, Kiehl *et al*. found reduced activity in ventral striatum among the psychopaths. Conducting a correlational analysis across a community sample of psychologically healthy individuals, Buckholtz *et al*. found that greater levels of 'psychopathic' traits (impulsive antisocial) were associated with increased activity in the ventral striatum. One possibility is that the difference in findings could be due to the different task demands in each study (memory for emotionally salient words vs reward anticipation). A second possibility is that the ventral striatum may respond differently in psychopaths than it does within the continuum of psychologically normal individuals. Buckholtz *et al*.'s data seem to predict that a group of psychopaths would exhibit increased activity in the ventral striatum (relative to non-psychopaths) during reward anticipation. The empirical confirmation of this prediction would certainly bolster the rationale for inferring the neural correlates of psychopathy through the study of psychologically normal individuals.

To conclude our discussion of this point, we offer a suggestion that researchers be mindful of the characteristics of their subject sample, and specify in their conclusions whether the neuroimaging data pertain to psychopaths, *per se*, or to normal variation in certain social/affective/behavioral traits.

Inconsistent criteria for identifying psychopaths

A second issue that may contribute to heterogeneity in psychopathy imaging data is inconsistency in the procedures for evaluating and identifying psychopaths. Most neuroimaging investigations of psychopathy rely on the Hare Psychopathy Checklist-Revised (PCL-R)[43] to define psychopathy. The PCL-R is a list of 20 psychopathic traits/behaviors that are scored from 0 to 2 based on the degree to which the subject exhibits the item, and thus total scores ranged from 0 to 40. PCL-R scores are ideally determined on the basis of a semistructured interview and review of file information such as criminal records, employment records, school records and collateral reports. However, studies involving non-incarcerated samples may lack access to detailed file information (for example, Yang *et al*.,[13] Glenn *et al*.[15] and Raine *et al*.[16]). The PCL-R manual advises cutoff scores for grouping subjects: total scores of $\geqslant 30$ indicate psychopathy, scores of $\leqslant 20$ indicate non-psychopathy and scores of 21–29 are considered intermediate.[43] (These PCL-R cutoff scores were developed with North American subject samples. A slightly lower psychopathy cutoff score (for example, PCL-R$\geqslant$28) may be appropriate for European samples.[44]) In reviewing the methods of the published imaging studies on 'psychopaths' (Table 1), we found that the recommended cutoff score of PCL-R$\geqslant$30 was followed in only two cases: one structural imaging study[18] and one functional imaging study.[26] Instead, researchers have employed a variety of minimum PCL-R total scores to define psychopathy. In fact, cutoff scores in the mid-20s (or even lower) are fairly common.[13,15,16,19–21,24,25] Because the proportion of individuals with PCL-R scores in the mid- to upper-20s is much higher than the proportion of individuals with PCL-R scores above 30, using a cutoff score in the mid 20s could potentially result in a group of 'psychopaths' among which the majority would have

PCL-R scores below 30. This supposition is borne out by the data from the imaging studies. For the groups of 'psychopaths' reported in the aforementioned imaging studies, six had mean PCL-R scores below 30.[13,15,19–21,24]

These inconsistent and relatively lenient criteria could substantially impact the variability and reproducibility of the imaging study results. A previous psychophysiological study found that subjects with intermediate PCL-R scores (21–29, mean = 25.8) exhibit significantly different patterns of emotion-modulated startle from subjects with PCL-R scores above the suggested cutoff ($\geqslant$30, mean = 33.3), but very similar patterns of emotion-modulated startle to non-psychopaths (PCL-R scores $\leqslant$20, mean = 13.4).[45] These data suggest that individuals with intermediate PCL-R scores (in the 20s) are more similar, at least in terms of affective psychophysiological responses, to non-psychopaths (PCL-R $\leqslant$20) than to psychopaths (PCL-R $\geqslant$30). If the neuroimaging data mirror these psychophysiological data, then the routine use of PCL-R cutoff scores in the 20s to define 'psychopathic' subject groups has likely resulted in seriously obscured results.

As a specific example, consider the results of two functional imaging studies in which subjects viewed pictures with negative emotional content (fearful faces)[21] or a set of negatively valenced pictures that included faces.[26] Muller *et al*. classified subjects as psychopaths if their PCL-R scores were greater than 30; Deeley *et al*. used a more liberal threshold of $\geqslant$25. The imaging results differed considerably. Deeley *et al*. found between-group differences in cerebellum, fusiform gyrus and postcentral gyrus. For each of these areas, activity was greater in the non-psychopathic group than in the psychopathic group; there were no brain areas where psychopaths exhibited greater levels of activity. By contrast, Muller *et al*. found that psychopaths had greater levels of activity in widespread areas of the brain, including the medial temporal lobe, occipital and parietal cortex, precentral gyrus, superior temporal gyrus, inferior and medial frontal gyri, anterior cingulate and amygdala. The vast differences in imaging results could be due to a number of differences in study design; however, as we describe above, the difference in psychopathic subject classification may contribute substantially to the divergent results.

Judicious subject classification is particularly germane to this field given the small sizes of psychopathic samples. Of the thirteen imaging studies that define a group of psychopaths (regardless of inclusion criteria), eight have samples of $n = 10$ psychopaths or less (Table 1). The two imaging studies that use the advised PCL-R cutoff score ($\geqslant$30) have psychopathic sample sizes of $n = 6$ and $n = 12$, respectively. Thus, at present there are insufficient data available to evaluate whether the use of more stringent PCL-R cutoff scores yields more consistent results. Given the small number of studies that actually used a PCL-R cutoff of 30 and the relatively small sample sizes within those studies, there is clearly a pressing need for imaging studies featuring larger samples of individuals with exceptionally high PCL-R scores. The recruitment of subjects with exceptionally high PCL-R scores may be costly and time-consuming, but in the long run the field of psychopathy research will benefit from more uniform standards for subject classification. In our view, a more rigorous collective effort in this regard will facilitate the integration of reliable neuroimaging results with each other, as well as with the clinical and psychological literatures on psychopathy.

Consideration of psychopathic subtypes

A third issue that may be contributing to the inconsistent imaging results in psychopathy is that psychopathy may consist of multiple distinct subtypes. The question of whether and how to subtype in psychopathy is nearly as old as the field of psychopathy research itself. Early work in this area described a theoretical distinction between 'primary' and 'secondary' psychopathy, based on the presumed etiology of the disorder as an innate vs an acquired disturbance of social-affective behavior.[33,46] More recent empirical research demonstrates that subdividing psychopaths on certain personality characteristics reveals significant behavioral and psychophysiological differences between psychopathic subgroups. Perhaps the most widely published means of subdividing psychopaths is on the basis of trait levels of anxiety and negative affectivity. Low-anxious, but not necessarily high-anxious, psychopaths have been documented to show abnormalities (relative to non-psychopaths) on a variety of laboratory measures, including tests of approach or avoidance learning,[32,47–50] delay of gratification,[51] executive function,[52] cued attention[53] and economic decision-making.[54] Taken together, these studies suggest that low-anxious psychopaths and high-anxious psychopaths have certain distinct behavioral and psychophysiological characteristics, despite similar overall levels of psychopathy. If these subgroups also have distinct neurobiological characteristics, and if the samples of psychopathic subjects in neuroimaging studies regularly contain a significant proportion of each subtype, then one might expect that the data would fail to show a consistent neurobiological defect. It seems that this has indeed been the case; as detailed above, there are few replicated neuroimaging findings in psychopathy. To date, none of the neuroimaging studies of psychopathy have employed a subtyping strategy.

The potential importance of considering subgroups within a psychopathological disorder, with respect to understanding the neuroimaging correlates of the disorder, is illustrated by studies of frontal lobe dysfunction in schizophrenia. The initial neuroimaging research on this topic generated inconsistent and ostensibly conflicting results. Several studies reported prefrontal cortex (PFC) hypo-activation among individuals with schizophrenia (for example,

Perlstein *et al*.,[55] Carter *et al*.[56] and Barch *et al*.[57]), whereas other studies reported no difference[58] or even PFC hyper-activation (for example, Manoach *et al*.[59,61] and Callicott *et al*.[60]) This apparent discrepancy has been addressed through the consideration of key differences *within* the schizophrenia patient group. For example, schizophrenia patients with significant working memory impairments typically exhibit PFC hypoactivity relative to controls, whereas patients with less impairment exhibit PFC hyperactivity.[62] Moreover, PFC hypoactivity has been specifically associated with symptoms of 'disorganization' (one of the three main symptom clusters of schizophrenia).[55] Thus, even though all patients with schizophrenia share the same diagnosis and a certain degree of overlapping symptoms, the subdivision of patients based on important differences in their neuropsychological test performance and their specific symptom profiles has proven to be a pivotal step in clarifying the neural correlates of the disorder. By analogy, the clarification of the neural correlates of psychopathy may similarly depend on the identification of one or more key variables that distinguish psychopathic subtypes.

To summarize this point, across many psychopathologies, the decision of whether and how to subtype is an issue. It is not always easy or necessary (depending on the research question) to examine disorders at this level. However, given the existing evidence that indicates significant behavioral and psychophysiological differences between certain psychopathic subgroups, it is perhaps worthwhile to consider subtyping in the neurobiological study of psychopathy. Employing this approach in future imaging studies may reduce the heterogeneity of the results and provide a more refined understanding of the disorder.

Conclusion

The elucidation of the neural correlates of psychopathy could have profound implications for the clinical and legal management of psychopaths, as well as for our basic understanding of the biological substrates underlying human social behavior. In this article, we sought to provide a critical review of structural and functional imaging studies aimed at identifying the neurobiological abnormalities associated with psychopathy. To date, the results are highly variable. Within the broad array of data one can find qualified support for theories highlighting the importance of emotion-related circuits in the brain, such as the ventromedial prefrontal cortex and amygdala[63,64] or a wider 'paralimbic' system,[65] which also includes areas involved in language and attentional orienting. Alternatively, one may view the heterogeneous collection of neuroimaging abnormalities, many of which are outside the canonical emotion circuits, as evidence for widespread, context-dependent neural deficits in information processing or integration.[66]

Given the remarkable heterogeneity of imaging results, it is perhaps premature to interpret certain findings as support for any particular theoretical viewpoint. Instead, it may be instructive to first evaluate whether differences in study methodology could account for some of the variability in the findings. To this end, we have raised a number of methodological considerations that may help explain some of the heterogeneity of data. For example, we noted that psychopathy-imaging studies have employed a variety of design and analysis strategies. Among the structural imaging studies, some have measured regional volumes whereas others have measured the integrity of white-matter pathways. Among functional imaging studies, some have used complex decision-making tasks whereas others have used simple passive viewing tasks. Among both structural and functional imaging studies, some have focused their analyses on predetermined regions-of-interest whereas others have reported effects throughout the brain. In addition, sample size (and hence statistical power) varies significantly among studies. These differences in study methodology could certainly contribute to some degree of heterogeneity in the psychopathy imaging data; indeed, these issues are relevant for interpreting neuroimaging results for any type of psychopathology. The focus of the present article is to identify issues that are especially germane to neuroimaging studies of psychopathy. We have described three such issues in this review. One issue is whether the study identifies neurobiological differences between groups (psychopaths vs non-psychopaths), or instead identifies brain areas associated with normal variation in social or affective traits among psychologically healthy individuals. The available evidence suggests that findings from these two different types of study may not be equally informative with respect to the neurobiology of psychopathy. A second issue is the consistency of criteria for classifying subjects as psychopaths—varying stringency in PCL-R cutoff scores between studies means varying levels of psychopathic behavior between study groups and, quite possibly, varying imaging findings. The use of more uniform standards for subject classification will facilitate a more straightforward comparison of results across studies. A third issue is the consideration of psychopathic subtypes. It could be that psychopaths consist of multiple subtypes (for example, low anxious vs high anxious) that have distinct neurobiological profiles. Neuroimaging data could provide key evidence to support or refute this hypothesis.

Neuroimaging research on psychopathy is a burgeoning field with immense promise but also significant methodological challenges. We are optimistic that as future imaging studies of psychopathy employ more rigorous and judicious standards for evaluating and classifying subjects, the brain anomalies characterizing psychopathy will become more clear. In turn, the more precise imaging results will illuminate the psychobiological mechanisms underlying psychopathy.

Conflict of interest

The authors declare no conict of interest.

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