Neuroimaging Studies of Psychopathy

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Contents

Abstract

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 chapter 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 (i.e., psychopaths vs. non-psychopaths) as well as correlational analyses (i.e., normal variation in "psychopathic" traits), (2) discrepancies in the

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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.

Human brain imaging techniques, such as magnetic resonance imaging (MRI), have become an indispensible 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 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 chapter 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.

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 28.1). 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 (Raine et al. 1997; Volkow et al. 1995), antisocial personality disorder (Barkataki et al. 2006; Raine et al. 2000), aggressive/impulsive behavior (Dolan et al. 2002), and pathological lying (Yang et al. 2005a). 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 2009.) The other line of research omitted here is the neuroimaging of children and adolescents with psychopathic tendencies (e.g., Dalwani et al. 2011; De Brito et al. 2009; Fairchild et al. 2013; Finger et al. 2012; Jones et al. 2009; Marsh et al. 2008). 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 reasons. One reason is that the diagnostic criteria for antisocial behavior in children/

Table 28.1 Neuroimaging studies of "psychopathy"

First author	Year	Title	Type of imaging	Type of analysis	PCL-R cutoff for P	Mean PCL-R for Ps	P sample size
Birbaumer	(2005)	Deficient fear conditioning in *psychopathy*: a functional magnetic resonance imaging study	F	BG	15	24.9	10
Bjork	(2012)	*Psychopathic* tendencies and mesolimbic recruitment by cues for instrumental and passively obtained rewards	F	C/R	n/a	n/a	n/a
Boccardi	(2011)	Cortex and amygdala morphology in *psychopathy*	S	BG	21	29.9	26
	(2010)	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
Carré	(2013)	The neural signatures of distinct *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
de Oliveira-Souza	(2008)	*Psychopathy* as a disorder of the moral brain: fronto-temporo-limbic gray matter reductions demonstrated by voxel-based morphometry	S	BG, C/R	n/a	n/a	15
Deeley	(2006)	Facial emotion processing in criminal *psychopathy*. Preliminary functional magnetic resonance imaging study	F	BG	25	29.3	6
Dolan	(2009)	*Psychopathy* and functional magnetic resonance imaging blood oxygenation level-dependent responses to emotional faces in violent patients with schizophrenia	F	BG, C/R	n/a	n/a	12
Ermer	(2012)	Aberrant paralimbic gray matter in criminal *psychopathy*	S	C/R	n/a	n/a	n/a
Glenn	(2009)	The neural correlates of moral decision-making in *psychopathy*	F	C/R	n/a	n/a	n/a
	(2010b)	No volumetric differences in the anterior cingulate of *psychopathic* individuals	S	BG, C/R	23	28.0	24
	(2010a)	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
Gregory	(2012)	The antisocial brain: *psychopathy* matters	S	BG	25	28.1	17

(continued)

Table 28.1 (continued)

First author	Year	Title	Type of imaging	Type of analysis	PCL-R cutoff for P	Mean PCL-R for Ps	P sample size
Harenski	(2009)	Neuroticism and *psychopathy* predict brain activation during moral and nonmoral emotion regulation	F	C/R	n/a	n/a	n/a
	(2010)	Aberrant neural processing of moral violations in criminal *psychopaths*	F	BG, C/R	30	31.8	16
Intrator	(1997)	A brain imaging (single photon emission computerized tomography) study of semantic and affective processing in *psychopaths*	F	BG	25	29.9	8
Juárez	(2013)	Intrinsic limbic and paralimbic networks are associated with criminal *psychopathy*	F	BG, C/R	30	32.5	17
Kiehl	(2001)	Limbic abnormalities in affective processing by criminal *psychopaths* as revealed by functional magnetic resonance imaging	F	BG	24	32.8	8
	(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
Ly	(2012)	Cortical thinning in *psychopathy*	F, S	BG	30	31.8	21
Marsh	(2012 in press)	When *psychopathy* impairs moral judgments: neural responses during judgments causing fear	F	BG	n/a	n/a	n/a
Motzkin	(2011)	Reduced prefrontal connectivity in *psychopathy*	F, S	BG	30	31.9	20
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
	(2008a)	Gray matter changes in right superior temporal gyrus in criminal *psychopaths*. Evidence from voxel-based morphometry	S	BG	28	33.4	17
	(2008b)	Disturbed prefrontal and temporal brain function during emotion and cognition interaction in criminal *psychopathy*	F	BG	28	30.5	10

Osumi	(2012)	Amygdala dysfunction attenuates frustration-induced aggression in *psychopathic* individuals in a noncriminal population	F	C/R	n/a	n/a	n/a
Pujara	(2013 in press)	Neural correlates of reward and loss sensitivity in *psychopathy*	F, S	BG, C/R	30	31.7	18
Pujol	(2012)	Breakdown in the brain network subserving moral judgment in criminal *psychopathy*	F	BG, C/R	20	27.8	22
Raine	(2003)	Corpus callosum abnormalities in *psychopathic* antisocial individuals	S	BG, C/R	23	30.3	15
	(2010)	Neurodevelopmental marker for limbic maldevelopment in antisocial personality disorder and *psychopathy*	S	BG	23	28.7	18
Rilling	(2007)	Neural correlates of social cooperation and noncooperation as a function of *psychopathy*	F	C/R	n/a	n/a	n/a
Sadeh	(2013)	Emotion disrupts neural activity during selective attention in *psychopathy*	F	C/R	n/a	n/a	n/a
Sato	(2011)	Identification of *psychopathic* individuals using pattern classification of MRI images	S	C/R	n/a	n/a	n/a
Sheng	(2010)	Default network deactivations are correlated with *psychopathic* personality traits	F	C/R	n/a	n/a	n/a
Sommer	(2010)	In *psychopathic* patients emotion attribution modulates activity in outcome-related brain areas	F	BG	28	28.6	14
Veit	(2010)	Aberrant social and cerebral responding in a competitive reaction time paradigm in criminal *psychopaths*	F	C/R	n/a	n/a	n/a
Yang	(2005b)	Volume reduction in prefrontal gray matter in unsuccessful criminal *psychopaths*	S	BG, C/R	23	28.4	29
	(2009)	Localization of deformations within the amygdala in individuals with *psychopathy*	S	BG, C/R	23	28.0	27
	(2010)	Morphological alterations in the prefrontal cortex and the amygdala in unsuccessful *psychopaths*	S	BG	23	n/a	26
	(2011)	Abnormal structural correlates of response perseveration in individuals with *psychopathy*	S	BG, C/R	23	n/a	27
	(2012)	Frontal information flow and connectivity in *psychopathy*	S	BG	n/a	n/a	55

P psychopathy, *S* structural, *F* functional, *C/R* correlation or regression analysis, *BG* between-group analysis, *n/a* not applicable or data not available

adolescents (such as conduct disorder) are necessarily somewhat different than 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 preadult age groups, even among neurologically and psychologically healthy individuals (Giedd et al. 2009). 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 2008.)

28.1 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 28.1). Structural neuroimaging studies associate psychopathy with a host of morphological brain abnormalities: reduced volumes of the amygdala, (Boccardi et al. 2011; Ermer et al. 2012; Yang et al. 2009, 2010); reduced volume of the basolateral nucleus of the amygdala and increased volumes of the central and lateral nuclei of the amygdala (Boccardi et al. 2011); reduced gray matter volumes in frontal cortex, especially the orbitofrontal cortex, the frontopolar cortex, the anterior rostral prefrontal cortex, and right inferior frontal gyrus (Boccardi et al. 2011; de Oliveira-Souza et al. 2008; Ermer et al. 2012; Gregory et al. 2012; Ly et al. 2012; Muller et al. 2008a; Yang et al. 2005b, 2010, 2011); reduced volume of the dorsal anterior cingulate cortex and bilateral precentral gyri (Ly et al. 2012); reduced volumes in temporal cortex, especially right superior temporal gyrus, anterior temporal cortices, superior temporal sulcus, and bilateral temporal pole (de Oliveira-Souza et al. 2008; Ermer et al. 2012; Gregory et al. 2012; Ly et al. 2012; Muller et al. 2008a; Yang et al. 2011); reduced volume of midline cortical structures (Boccardi et al. 2011); reduced volume of the posterior cingulate cortex (Ermer et al. 2012); increased volume of the striatum (Glenn et al. 2010a); increased volume of the left nucleus accumbens (Pujara et al. 2013); increased volume of the corpus callosum (Raine et al. 2003); reduced volume of posterior hippocampus (Laakso et al. 2001); normal volume but abnormal shape of the hippocampus (Boccardi et al. 2010); reduced volume in parahippocampal regions (Ermer et al. 2012); reduced volume of the insula (de Oliveira-Souza et al. 2008; Gregory et al. 2012; Ly et al. 2012); presence of cavum septum pellucidum (Raine et al. 2010); and reduced structural integrity of the uncinate fasciculus (Craig et al. 2009; Motzkin et al. 2011). 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 play important roles in emotion and social cognition (amygdala, superior temporal cortex, uncinate fasciculus) as well as learning and memory (striatum, hippocampus). But within this broad functional/anatomical grouping of the study results, the available structural imaging data

have not yet demonstrated reliable, replicated structural abnormalities in many of the identified brain regions.

Functional imaging studies measure brain activity, either at "rest" or during a particular cognitive task. In psychopathy research, functional imaging studies have typically featured tasks involving social and/or emotional processing, such as fear conditioning (Birbaumer et al. 2005), viewing facial expressions of emotion (Carré et al. 2013; Deeley et al. 2006; Gordon et al. 2004), emotion attribution (Sommer et al. 2010), moral decision-making (Glenn et al. 2009; Harenski et al. 2009, 2010; Pujol et al. 2012), identification of emotionally salient words (Intrator et al. 1997), memory for emotionally salient words (Kiehl et al. 2001), selective attention and emotional processing during an emotion-word Stroop task (Sadeh et al. 2013), viewing emotionally salient scenes (Muller et al. 2003, 2008b), social cooperation (Rilling et al. 2007), anticipation and/or receipt of reward (Bjork et al. 2012; Buckholtz et al. 2010; Carré et al. 2013; Pujara et al. 2013), and punishment administration (Veit et al. 2010). Accordingly, many of these studies focus their analyses on emotion-related regions of interest, such as the amygdala (Birbaumer et al. 2005; Carré et al. 2013; Glenn et al. 2009; Gordon et al. 2004; Kiehl et al. 2001; Rilling et al. 2007). However, the imaging results indicate that psychopathy is associated with abnormal activity in widespread areas of the brain, not just those associated with emotional processing. Reduced activity has been observed in limbic and paralimbic areas, including amygdala (Birbaumer et al. 2005; Carré et al. 2013; Glenn et al. 2009; Kiehl et al. 2001; Rilling et al. 2007), hippocampus and parahippocampal gyri (Kiehl et al. 2001; Muller et al. 2003), anterior and posterior cingulate cortex (Birbaumer et al. 2005; Kiehl et al. 2001; Muller et al. 2003; Rilling et al. 2007), ventral striatum (Kiehl et al. 2001), and insula (Birbaumer et al. 2005). On the other hand, reduced activity has also been observed in association areas within frontal and temporal cortices (Birbaumer et al. 2005; Gordon et al. 2004; Muller et al. 2003, 2008b; Rilling et al. 2007) as well as sensory areas, such as posterior visual cortices (Deeley et al. 2006; Muller et al. 2003) and parietal somatosensory cortex (Birbaumer et al. 2005; Deeley et al. 2006), and motor structures such as cerebellum (Deeley et al. 2006) and primary motor cortex (Deeley et al. 2006). Increased activity has been observed in frontal and temporal cortices (Intrator et al. 1997; Kiehl et al. 2001; Muller et al. 2003), nucleus accumbens (Bjork et al. 2012; Buckholtz et al. 2010), as well as areas of parietal lobe, occipital lobe, cerebellum, cingulate cortex, and amygdala (Muller et al. 2003). Functional imaging studies may also assess the correlated activity, or "functional connectivity," between various brain regions at rest or during a task. Psychopathy was associated with connectivity among brain regions known as the "default mode network," which includes the medial prefrontal cortex, posterior cingulate, and the inferior parietal lobule; frontoparietal connectivity; and a visual/posterior cingulate connectivity during an auditory "oddball" task (Juárez et al. 2013). Further, amygdala dysfunction in psychopaths during a task of moral decision-making was associated with reduced functional connectivity between the amygdala and the striatum (Osumi et al. 2012). At rest, psychopaths exhibit a reduction in functional connectivity between the left insula and dorsal ACC, the vmPFC and the amygdala, the vmPFC and medial

parietal cortex, and the posterior cingulate cortex and anterior frontal cortical areas (Ly et al. 2012; Motzkin et al. 2011; Pujol et al. 2012). Taken together, these functional imaging data associate psychopathy with abnormal activity in limbic, subcortical, and cortical 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 hyperactive. For example, amygdala activity was abnormally low during fear conditioning (Birbaumer et al. 2005), moral decision-making (Glenn et al. 2009), social cooperation (Rilling et al. 2007), viewing facial expressions of fear (Carré et al. 2013; Dolan and Fullam 2009), and memory for emotionally salient words (Kiehl et al. 2001) but abnormally high during the viewing of certain emotionally salient scenes (Muller et al. 2003) and facial expressions of anger (Carré et al. 2013). Similarly, ventral striatum activity was abnormally low during memory for emotionally salient words (Kiehl et al. 2001) but abnormally high during reward anticipation (Bjork et al. 2012; Buckholtz et al. 2010). 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 hyperactive; 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 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.

28.2 Methodological Issues

28.2.1 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 (Cleckley 1941; Karpman 1946; Lykken 1957). In studies employing this usage, the data analysis strategy typically involves between-group comparisons of neuroimaging data (i.e., psychopaths vs. non-psychopaths; see Table 28.1). 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. In studies employing this usage, the data analysis strategy typically involves correlation or

regression analyses between a psychopathy score[1] and one or more neuroimaging measures (see Table 28.1). 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 (Edens et al. 2006; Marcus et al. 2004; Walters et al. 2008; Walters et al. 2007)—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 subgenual cingulate cortex, dorsolateral prefrontal cortex, and dorsal anterior cingulate (Greicius et al. 2007; Johnstone et al. 2007; Mayberg et al. 2005). A separate study that correlated individual variation in the experience of negative affect with brain activity among psychiatrically healthy individuals identified an area of ventromedial prefrontal cortex (adjacent to subgenual cingulate) but did not identify the more dorsal frontal areas (Zald et al. 2002). 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 findings on activity in ventral striatum, a critical brain area in processing reward and positive emotion. 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 ventral striatum in the anticipation of reward. Another study found a similar association between

[1] Note that the data entered into such correlational analyses may be overall psychopathy scores (Glenn et al. 2009) or scores on a particular dimension or "factor" of psychopathy, such as antisocial impulsivity (Buckholtz et al. 2010) or the interpersonal factor (Glenn et al. 2009). Differences in the exact "psychopathic" traits being analyzed may also contribute to heterogeneity of results regarding the neural correlates of psychopathy.

"psychopathic" traits and ventral striatum activity in response to the anticipation of reward (Bjork et al. 2012). 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. The Buckholtz et al.. data seem to predict that a group of psychopaths would exhibit increased activity in the ventral striatum (relative to non-psychopaths) during reward anticipation. However, a recent study comparing a group of criminal psychopaths to non-psychopaths on a task involving the passive receipt of reward found that psychopaths and non-psychopaths did *not* differ in ventral striatal response to monetary reward (Pujara et al. 2013). Instead, PCL-R score was positively correlated with ventral striatal response to reward only in the psychopathic group but not the non-psychopathic group. This finding clearly does not support the rationale for inferring 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.

28.2.2 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) (Hare 2003) 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 range from 0 to 40. PCL-R scores are ideally determined on the basis of a semi-structured 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 (e.g., Glenn et al. 2010a; Raine et al. 2003; Yang et al. 2009). The PCL-R manual advises cutoff scores for grouping subjects: total scores of 30 or greater indicate psychopathy, scores of 20 or less indicate non-psychopathy, and scores of 21–29 are considered intermediate[2] (Hare 2003). In reviewing the methods of the published imaging studies on "psychopaths" (see Table 28.1), we found that this recommendation was followed in only a few studies of psychopathy (Boccardi et al. 2011; Harenski et al. 2010; Juárez et al. 2013; Motzkin et al. 2011; Muller et al. 2003; Pujara et al. 2013). Instead, researchers have routinely 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

[2] These PCL-R cutoff scores were developed with North American subject samples. A slightly lower psychopathy cutoff score (e.g., 28) may be appropriate for European samples (44).

(Birbaumer et al. 2005; Craig et al. 2009; Deeley et al. 2006; Glenn et al. 2010a; Gregory et al. 2012; Intrator et al. 1997; Kiehl et al. 2001; Ly et al. 2012; Raine et al. 2003; Yang et al. 2009, 2010). 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, 12 had mean PCL-R scores below 30 (see Table 28.1).

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 (≥30, mean = 33.3) but very similar patterns of emotion-modulated startle to non-psychopaths (PCL-R scores ≤20, mean = 13.4) (Patrick et al. 1993). 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 ≤20) than to psychopaths (PCL-R ≥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 (Deeley et al. 2006) or a set of negatively valenced pictures that included faces (Muller et al. 2003). 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 25 or greater. 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 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 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 28 imaging studies that define a group of psychopaths (regardless of inclusion criteria), eight have samples of $n = 10$ psychopaths or less (Table 28.1). The seven imaging studies that use the advised PCL-R cutoff score (30 or greater) have psychopathic sample sizes ranging from $n = 6$ to $n = 21$, 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.

28.2.3 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 versus an acquired disturbance of social/affective behavior (Karpman 1946, 1948). 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 (Arnett et al. 1993, 1997; Lykken 1957; Newman et al. 1990; Schmauk 1970), delay of gratification (Newman et al. 1992), executive function (Smith et al. 1992), cued attention (Zeier et al. 2009), and economic decision-making (Koenigs et al. 2010). 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, only one neuroimaging study of psychopathy has employed this subtyping strategy (Motzkin et al. 2011).

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 PFC hypoactivation among individuals with schizophrenia (e.g., Barch et al. 2001; Carter et al. 1998; Perlstein et al. 2001), whereas other studies reported no difference (Honey et al. 2002) or even PFC

hyperactivation (e.g., Callicott et al. 2000; Manoach et al. 1999, 2000). 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 (Manoach 2003). Moreover, PFC hypoactivity has been specifically associated with symptoms of "disorganization" (one of the three main symptom clusters of schizophrenia) (Perlstein et al. 2001). 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 (Blair 2007, 2008) or a wider "paralimbic" system[3] (Kiehl 2006). 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 (Newman et al. 2010).

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

[3] In addition to proposing dysfunction in areas preferentially involved in affective processing, Kiehl's "paralimbic hypothesis" also proposes dysfunction in spatially distributed areas involved in language and attentional orienting.

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 chapter 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 (e.g., 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.

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