

Functional Neuroscience of Psychopathic Personality in Adults

Ana Seara-Cardoso¹ and Essi Viding^{1,2}

¹Division of Psychology and Language Sciences, University College London

²Institute of Cognitive Neuroscience, University College London

Abstract

Psychopathy is a personality disorder that involves a constellation of traits including callous-unemotionality, manipulativeness, and impulsiveness. Here we review recent advances in the research of functional neural correlates of psychopathic personality traits in adults. We first provide a concise overview of functional neuroimaging findings in clinical samples diagnosed with the PCL-R. We then review studies with community samples that have focused on how individual differences in psychopathic traits (variously measured) relate to individual differences in brain function. Where appropriate, we draw parallels between the findings from these studies and those with clinical samples. Extant data suggest that individuals with high levels of psychopathic traits show lower activity in affect-processing brain areas to emotional/salient stimuli, and that attenuated activity may be dependent on the precise content of the task. They also seem to show higher activity in regions typically associated with reward processing and cognitive control in tasks involving moral processing, decision making, and reward. Furthermore, affective-interpersonal and lifestyle-antisocial facets of psychopathy appear to be associated with different patterns of atypical neural activity. Neuroimaging findings from community samples typically mirror those observed in clinical samples, and largely support the notion that psychopathy is a dimensional construct.

Psychopathy is a multidimensional personality disorder, which is primarily diagnosed in criminal justice settings. The current definitions of criminal psychopathy comprise elements of affective and interpersonal dysfunction, as well as of parasitic and irresponsible lifestyle and antisocial behavior (e.g., Blair, Mitchell, & Blair, 2005; Hare, 2003; Hare & Neumann, 2008). The origins of the current description of the psychopathy syndrome can be traced back to the work of Cleckley and his book *The Mask of Sanity* (1941). Some of the key features of psychopathy recorded by Cleckley (1941) included absence of nervousness, interpersonal charm, lack of shame, impoverished affect, and poorly motivated antisocial behavior. From the criteria delineated by Cleckley, and from his own clinical impressions, Robert Hare developed the Psychopathy Checklist (PCL; 1980), and later the PCL-Revised (PCL-R; 1991, 2003), a formalized, empirically validated tool for the assessment of psychopathy in incarcerated adults. The PCL-R identifies the most severe, versatile, and persistent offenders characterized by pronounced lack of empathy and guilt, reduced attachment to other people, shallow affect, conning and manipulative conduct, superficial charm, grandiosity, parasitic use of others, inability to hold down a job, criminal versatility, and early onset of antisocial conduct. Extensive factor-analytic work on criminal samples in several different countries indicates that psychopathy symptoms load on four separable, but interrelated facets: affective, interpersonal, lifestyle, and antisocial (Hare, 2003; Hare & Neumann, 2008).

The affective facet includes characteristics such as lack of remorse or guilt, shallow affect, callousness, and lack of empathy, whereas the interpersonal facet includes characteristics such as superficial charm, grandiose sense of self-worth, pathological deception, and manipulation of others. Lifestyle characteristics include need for stimulation, parasitic lifestyle, lack of realistic long-term goals, impulsivity, and irresponsibility; the antisocial behavior facet records poor behavioral controls, early behavior problems, juvenile delinquency, and criminal versatility. These facets can be modeled in terms of the traditional two-factor dimensions of psychopathy—Factor 1 consisting of affective-interpersonal traits and Factor 2 consisting of antisocial-lifestyle characteristics. All facets share variance and load onto a superordinate psychopathy factor (Hare & Neumann, 2008).

Given the extreme profile of the criminals with psychopathy, there has been considerable interest in the neural correlates of the disorder. This article focuses on reviewing functional magnetic resonance imaging (fMRI) findings of psychopathic

Correspondence concerning this article should be addressed to Ana Seara-Cardoso, Division of Psychology and Language Sciences, University College London, 26 Bedford Way, London WC1H 0AP. Email: ana.cardoso.09@ucl.ac.uk.

This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.

personality in adulthood.¹ fMRI is a commonly used neuroimaging technique for measuring brain activity in response to experimental task demands. It is noninvasive and has excellent spatial and fairly good temporal resolution. Broadly speaking, fMRI detects changes in blood oxygenation and flow that occur in response to neural activity. Brain areas that display a change in neural activity, for example, when participants view emotional instead of neutral faces, will be associated with a change in the relative concentration of oxygenated and deoxygenated blood. These alterations in the local magnetic properties of the blood (hemoglobin in blood is diamagnetic when oxygenated but paramagnetic when deoxygenated) result in differences in brain oxygenation level dependent (BOLD) signal intensity that can be measured to generate estimates of neural activity related to a particular psychological process. In this article, we will first review fMRI findings on samples of individuals who qualify for the diagnosis of criminal psychopathy as assessed using the PCL-R. We will then discuss fMRI research on community samples that have reported associations with dimensional measures of psychopathy. Finally, we will very briefly discuss fMRI research on those aspects of a general model of personality that have been associated with psychopathy. In order to most readily compare the findings from incarcerated and healthy volunteer samples, we have restricted this review to adult samples. We would, however, like to highlight that a number of fMRI studies of children and youth with high levels of psychopathic/callous-unemotional traits have been conducted to date and that the findings from these studies are similar to those reported in adult samples (see, e.g., Blair, 2013; Viding & McCrory, 2012, for recent reviews).

fMRI FINDINGS IN HIGH-RISK SAMPLES DIAGNOSED WITH THE PCL-R

At the time of writing this article, our PubMed and Scopus search brought up 19 fMRI studies that included participants assessed with the PCL-R. Search terms were (“magnetic resonance” or “MR” or “MRI” or “fMRI” or “neuroimaging”) and (“psychopathy”). Exclusion criteria were (a) studies that did not include a formal assessment with the PCL-R, (b) studies of children and youth, (c) studies with psychiatric populations, and (d) studies reporting structural, resting-state, or exclusively connectivity MRI analyses. These studies varied considerably in their sample sizes (ranging from 12 to 121 participants) and paradigms used (ranging from tasks assessing basic processing of salient/affective stimuli to more complex tasks indexing neural correlates of moral and economic decision making; see Table 1). The selection of the fMRI paradigms reflects attempts to understand the neural underpinnings of the unemotional, unempathetic, amoral, and impulsive behavior that characterizes psychopathy in both naturalistic and experimental settings.

Basic Emotional Stimuli

One of the first fMRI studies on psychopathy was performed by Kiehl et al. in 2001. This study contrasted eight criminal psychopaths, eight criminal nonpsychopaths, and eight noncriminal controls when they performed an affective recognition memory task where they had to indicate whether a list of words contained words from a previously memorized list. These lists were composed of all neutral or all negatively valenced words. Compared with criminal nonpsychopaths and noncriminal controls, individuals with psychopathy presented significantly less differential activation between affective and neutral conditions in the amygdala/hippocampal formation, parahippocampal gyrus, ventral striatum, and the anterior and posterior cingulate gyrus, as well as increased activity in the bilateral frontotemporal cortex. Although the size of the psychopathic group was small ($n = 8$), the sample was well characterized, all individuals in the psychopathic group had high PCL-R scores (above 28), and groups were carefully matched. Furthermore, the only difference between the task conditions pertained to the affective content of the stimuli, as the affective and nonaffective words were matched on length, imagery, concreteness, and frequency. This study therefore enabled the authors to conclude that the neural differences associated with psychopathy indexed disturbed affective processing.

Two studies to date have focused on classical conditioning to investigate possible neurobiological correlates that underlie impaired emotional learning in psychopathy. One of these studies used an unpleasant olfactory stimulus (Schneider et al., 2000) and the other painful pressure (Birbaumer et al., 2005) as unconditioned stimuli, and both used neutral male faces as neutral and conditioned stimuli. Schneider et al. (2000) reported that in contrast to healthy controls, individuals with psychopathy failed to activate the amygdala in response to the unconditioned stimulus (unpleasant odor) before this was paired with the faces. Instead, they showed decreased activity in this region. Healthy controls and individuals with psychopathy also exhibited opposite patterns during the acquisition of conditioning phase. While individuals with psychopathy showed increased amygdala and dorsolateral prefrontal cortex activation in response to the conditioned stimuli (neutral faces paired with an unpleasant odor), healthy controls showed decreased activity in these regions. In contrast, Birbaumer et al. (2005) reported no group differences in response to the unconditioned stimulus (pain) when this was presented on its own, and significantly less activation in several cortical and subcortical regions in the psychopathic group in relation to healthy controls during the acquisition phase in response to the conditioned stimulus. These included the amygdala, anterior insula, anterior cingulate, and orbitofrontal cortex. In sum, these studies show that deficits in emotional learning, which are hypothesized to be fundamental to the disorder (Blair, 2008; Blair et al., 2005), seem to be accompanied by atypical response in cortical and subcortical regions, in particular in the

Table 1 fMRI Studies in High-Risk Samples Diagnosed With the Psychopathy Checklist–Revised (PCL-R)

Study	Sample	Tasks	Compared to Controls, High PP Presented:
Kiehl et al., 2001	8 high PP (PCL-R > 28); 8 low PP offenders (PCL-R < 23); 8 controls	Affective recognition memory task	Less AMY/hippocampal formation, parahippocampal gyrus, VS, ACC, and PCC response to affective words More bilateral frontotemporal cortex response to affective words condition
Schneider et al., 2000	12 high PP offenders (PCL-R > 24); 12 controls	Emotional learning	Decreased AMY response to unconditioned stimulus (unpleasant odor) Increased AMY and dlPFC response to conditioned stimuli (neutral faces paired with unpleasant odor)
Birbaumer et al., 2005	10 offenders (PCL-R > 15; FI > 10.5); 10 controls	Emotional learning	Less AMY, AI, ACC, and OFC response to conditioned stimulus (neutral faces paired with painful pressure)
Müller et al., 2003	6 high PP offenders (PCL-R > 30); 6 controls	Mood induction	More fusiform, temporal, precentral, cerebellum, and IFG response to positive stimuli Less occipital, MFG, and MTG response to positive stimuli More response in occipital, MTG, precentral gyrus, STG, IFG and MFG, ACC and AMY to negative stimuli Less activation in ACC, MTG, fusiform, and parahippocampal gyrus in response to negative stimuli
Müller et al., 2008	10 high PP offenders (PCL-R > 28); 12 controls	Attentional paradigm after mood induction	High PP group presented a significant Task × Emotion interaction in right insula Controls presented a significant interaction Task × Emotion in MFG, IFG, SMG, and precuneus
Deeley et al., 2006	10 high PP offenders (PCL-R > 25); 9 controls	Implicit facial emotion	Less fusiform gyrus and extrastriate cortex response to emotional faces
Decety et al., 2014	27 high PP (PCL-R > 30); 25 medium PP (PCL-R: 21–29); 28 low PP (PCL-R < 20) offenders	Passive viewing of dynamic facial expressions	Less IFG, OFC, inferior occipital gyrus, fusiform gyrus, and STS response to emotional facial expressions More AI response to negative facial expressions
Mier et al., 2014	11 high PP offenders (PCL-R > 25); 18 controls	Facial gender, emotion, and ToM recognition	Less fusiform gyrus response to faces Less STS, IFG, and AMY in response to increasing demands of the task (gender < emotion < ToM)
Decety, Skelly, et al., 2013	27 high PP (PCL-R > 30); 28 medium PP (PCL-R: 21–29); 25 low PP (PCL-R < 20) offenders	Empathy for pain	Less vmPFC, IOFC, and periaqueductal gray response to body parts in pain More AI, dACC, DS, IFG, mPFC, pSTS, and SMG response to body parts in pain Less IFG, midCC, pSTS, SMG, dACC, dmPFC, and putamen response to pain facial expressions More AI, poscentral gyrus, IPL, and precentral gyrus response to pain facial expressions
Decety, Chen, et al., 2013	37 high PP (PCL-R > 30); 44 med PP (PCL-R: 21–29); 40 low PP (PCL-R < 20) offenders	Perspective taking/Empathy for pain	More AI, ACC, and IFG response to body parts in pain when instructed to imagine self Less AI, AMY, OFC, and vmPFC response to body parts in pain when instructed to imagine other
Meffert et al., 2013	18 high PP offenders (PCL-R > 28); 26 controls	Empathy for pain	Less AI, IFG, dACC, and AMY response to pain stimuli Group differences reduced when instructed to “empathize”
Sommer et al., 2010	14 high PP (PCL-R > 28); 14 low PP (PCL-R < 15) offenders	Affective ToM/Emotion attribution	More OFC, MFG, and left TPJ response during emotion attribution
Glenn et al., 2009a,b	17 participants from community (PCL-R: 7.2–32)	Moral dilemmas	Negative correlation between PCL-R scores and left AMY response to highly emotional moral dilemmas Negative correlation between interpersonal PCL-R facet scores and mPFC, PCC, and angular gyrus Positive correlations between lifestyle and antisocial PCL-R facets scores and dlPFC response to highly emotional moral dilemmas
Pujol et al., 2012	22 high PP offenders (PCL-R > 20); 22 controls	Moral dilemmas	Less mPFC, PCC, and hippocampus response to highly emotional moral dilemmas
Harenski et al., 2010	16 high PP (PCL-R > 30); 16 low PP (PCL-R < 18) offenders	Moral judgment of visual stimuli	Less vmPFC and anterior temporal cortex response to moral violations Weaker association between AMY response and severity of moral violation ratings Stronger association between posterior temporal response and severity of moral violation ratings
Prehn et al., 2013	11 high PP (PCL-R > 23); 12 borderline and antisocial offenders; 13 controls	Economic decision making	Less ACC response to high uncertainty trials Less IFG response when making low-risk options during high uncertainty More IFG response when making high-risk options during high uncertainty
Pujara et al., 2013	18 high PP (PCL-R > 30); 23 low PP (PCL-R < 20) offenders	Passive monetary reward	No significant differences between groups in VS response to reward High PP group presented positive association between VS response to reward and PCL-R scores; the low PP group did not

Note. ACC = anterior cingulate cortex; AI = anterior insula; AMY = amygdala; dACC = dorsal anterior cingulate cortex; dlPFC = dorsolateral prefrontal cortex; dmPFC = dorsomedial prefrontal cortex; DS = dorsal striatum; IFG = inferior frontal cortex; IPL = inferior parietal lobe; IOFC = lateral orbitofrontal cortex; MFG = middle frontal gyrus; midCC = midcingulate cortex; MTG = middle temporal gyrus; PP = psychopathy; OFC = orbitofrontal cortex; PCC = posterior cingulate cortex; pSTS = posterior superior temporal sulcus; SMG = supramarginal gyrus; STG = superior temporal gyrus; STS = superior temporal sulcus; ToM = theory of mind; TPJ = temporo-parietal junction; vmPFC = ventromedial prefrontal cortex; VS = ventral striatum.

amygdala (albeit in opposite directions). It should be noted that both of these studies have relatively small groups of individuals with psychopathy ($N < 12$), with some individuals not scoring above the PCL-R cut-off of 30.

Two studies by Müller and colleagues investigated the impact of mood induction on neural activity. The first of these employed a simple mood induction task that involved showing the participants positive, negative, and neutral photos from the International Affective Picture System and measuring neural activity against rest (Müller et al., 2003). The second study involved a similar mood induction task prior to performing a simple attentional paradigm (Müller et al., 2008). The authors report reduced BOLD response in psychopaths in several brain areas (e.g., anterior cingulate, amygdala, and hippocampal gyrus) following the mood induction, but the findings from these studies are difficult to interpret for the following reasons. The sample sizes in both studies were very small (high PCL-R $N < 10$), and the statistical analyses were such that it is difficult to interpret the specific cognitive/affective processes that the differential neural activity related to (e.g., in Müller et al. 2003, all stimuli were contrasted against rest, and therefore the precise cognitive process driving any group differences in neural activation was not clear).

These studies suggest that individuals with psychopathy have atypical brain activity during processing of basic emotional stimuli, during emotional learning, and when performing simple cognitive tasks following emotion induction. An extended set of cortical and subcortical brain regions has been implicated, with the most commonly reported functional atypicalities, albeit sometimes in opposing directions, across studies found in the anterior insula, amygdala, and portions of the prefrontal cortex. Differences in functional activity in this set of brain regions in individuals with psychopathy in comparison to control groups also seem to extend to more complex forms of social cognition, such as empathic processing, affective theory of mind, decision making, or moral judgment, which will be reviewed next.

Emotional Facial Expressions

Facial expressions constitute important cues to others' emotional states. They can be readily perceived and have important communicatory functions, conveying information about the observed person to the observer (Blair, 2003). Facial expressions can thus trigger robust emotional reactions in humans, such as feelings of threat, personal distress, and empathy. Three studies to date have investigated the neural basis of facial emotion processing in psychopathy.

Deeley et al. (2006) presented individuals with psychopathy (PCL-R range: 25–34, $n = 6$) and healthy controls ($n = 9$) facial expressions of happiness and fear, as well as neutral faces. The participants' task was to name the gender of the face; the emotion processing was therefore an implicit aspect of the task, and participants were not actively identifying the emotions from the faces. Deeley et al.

(2006) found that these groups presented significant differences in BOLD response while implicitly processing emotional facial expressions. Compared with controls, individuals with psychopathy showed decreased activity in the fusiform gyrus and in the extrastriate cortex to both types of emotional facial expressions. Previous studies have shown that activity in these regions in response to emotional faces is boosted by feedback modulation from the amygdala (Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004), and the authors speculated that the decreased activation of these regions in response to emotional faces is a reflection of amygdala dysfunction. However, possibly due to a small sample ($N = 15$; high PCL-R $n = 9$), significant differences in amygdala response in the two groups were not detected.

More recently, Decety, Skelly, Yoder, and Kiehl (2014) investigated neural processing of dynamic facial expressions of fear, sadness, happiness, and pain in a large group of incarcerated males ($N = 80$) with varying levels of PCL-R scores. Decety et al. (2014) found that while passively observing dynamic emotional facial expressions (contrasted against dynamically scrambled baseline), inmates with the highest levels of psychopathy presented significantly lower hemodynamic response than inmates with low and medium levels of psychopathy in a wide set of regions, including facial cortical processing areas such as the fusiform gyrus, and regions typically involved in affective processing like the inferior frontal gyrus and orbitofrontal cortex. In contrast to what might be expected, given the putative role of the anterior insula in affective processing, and in particular in sensory integration (Critchley, Wiens, Rotshtein, Öhman, & Dolan, 2004) and interoceptive awareness (Craig, 2009; Critchley & Harrison, 2013), Decety et al. (2014) found that the group with the highest levels of psychopathy presented increased activity in this region in response to negative emotional facial expressions.

Finally, Mier et al. (2014) compared a group of 11 incarcerated males with psychopathy (PCL-R range: 25–29) and a group of 18 controls from the general population using a more complex task of facial processing. In this task, participants had to recognize the gender of neutral faces (neutral condition), recognize the emotion portrayed by emotional facial expressions (emotional recognition condition) or recognize the intentions of the people posing the emotional expressions (theory of mind condition). In line with findings from Deeley et al. (2006), Mier et al. (2014) found that, contrary to controls, the psychopathy group did not present an increase in BOLD response in the fusiform gyrus when processing faces. The authors also found that, contrary to controls, the group with psychopathy did not present increased response in the superior temporal sulcus, inferior frontal gyrus, and amygdala, with increasing demands of the task (i.e., when contrasting BOLD response during theory of mind > emotion recognition > neutral). Interestingly, there were no significant differences across the groups in their behavioral responses to the

task. The authors speculate that it is possible that although individuals with psychopathy might be able to correctly identify emotional and mental states, they do not show the typical accompanying neural response in brain areas associated with affective processing.

Empathy and Related Constructs

Callous and unempathic behavior is the hallmark of individuals with psychopathy. It has been proposed that the absence of a robust empathic response (i.e., the experience of a corresponding affective state that is elicited by the observation or imagination of another person's affective state; e.g., Eisenberg, 2000) to other people's distress explains why these individuals find it easier to aggress against their fellow human beings (Blair, 2013; Blair et al., 2005). Three recent studies have addressed the neural correlates of empathic processing in adults with psychopathy while they viewed other people in painful and nonpainful situations.

Recent meta-analyses of empathy for pain studies (Fan, Duncan, de Greck, & Northoff, 2011; Lamm, Decety, & Singer, 2011) indicate that the observation of others' experiences of pain elicits robust activation in the anterior insula, inferior frontal gyrus, and dorsal anterior cingulate. Decety, Skelly, and Kiehl (2013) found that when observing facial expressions of pain, incarcerated men with high levels of psychopathy (compared with incarcerated men with low levels of psychopathy) exhibited reduced activity in the inferior frontal gyrus and dorsal anterior cingulate and *greater* activity in the anterior insula. When they observed body parts in painful situations, they showed *greater* responses in all these regions. Because this study did not include specific instructions for imagining self versus another person in pain, it is not possible to know precisely what the participants were thinking in relation to the pain stimuli in that study. In a follow-up study, Decety, Chen, Harenski, and Kiehl (2013) reported that manipulating the instruction given to participants before the observation of body parts in painful situations has an impact in the patterns of activation observed. When inmates with high levels of psychopathic traits were instructed to imagine themselves in the pictures, they showed increased activity in the anterior insula, dorsal anterior cingulate, and inferior frontal gyrus in relation to those with low levels of these traits. In contrast, when instructed to imagine another person in the pictures, they showed *reduced* response and connectivity in the anterior insula and orbitofrontal cortex as well as in the amygdala. The response in the amygdala and anterior insula was also negatively associated with the Factor 1 dimension of psychopathy. It is thus possible that individuals with high levels of psychopathy show a stronger response in affective brain regions when imagining themselves in pain, while at the same time a weaker response in the same brain regions when imagining others in pain, which may contribute to their callous behavior toward others despite the suffering they may cause.

Consistent with Decety, Chen, et al. (2013), Meffert, Gazzola, Den Boer, Bartels, and Keysers (2013) reported that when observing videos depicting hands in emotional interactions (e.g., a hand being caressed or hit by another hand), male offenders with psychopathy ($n = 18$; PCL-R > 28) had *lower* activation relative to non-offenders ($n = 26$) in a similar set of brain regions, including the anterior insula, inferior frontal gyrus, dorsal anterior cingulate, and amygdala. Interestingly, Meffert et al. (2013) also reported that when participants were explicitly instructed to "empathize" with the actors in the video, group differences in activation related to psychopathy were reduced. This suggests that top-down instruction can modulate neural responses to affective stimuli in individuals with psychopathy, although it is still unclear whether their subjective experience of empathy is altered or comes to resemble empathic response as experienced by healthy controls.

Psychopathy has also been associated with atypical brain function during affective theory of mind (i.e., the ability to attribute affective states to others) tasks. Sommer et al. (2010) compared two groups of criminal patients in a high-security hospital with different levels of psychopathy (high PCL-R > 28 , $n = 14$; low PCL-R < 15 , $n = 14$) while performing a task where they had to guess the emotional state of the protagonist of a cartoon story. Interestingly, as in Mier et al. (2014), the two groups did not differ behaviorally but presented distinct brain activation during the performance of the task. During the emotion attribution condition, the control group presented increased activity in the superior temporal sulcus, supramarginal gyrus, and frontal gyrus. The psychopath group did not show this increased activation, although the difference between groups was not statistically significant. Importantly, psychopathic patients, compared to nonpsychopathic patients, exhibited significantly more activation in the orbitofrontal cortex, medial frontal cortex, and left temporo-parietal junction during emotion attribution. The distinct pattern of neural responses in each group was interpreted by the authors as a possible indicator of different computational strategies for the inference of others' mental states. Increased response in the orbitofrontal cortex and left temporo-parietal junction, which have been previously associated with processing the value of an outcome and with mentalizing efforts (Amodio & Frith, 2006; Rushworth, Buckley, Behrens, Walton, & Bannerman, 2007; Rushworth, Kolling, Sallet, & Mars, 2012), may reflect additional efforts in computing the emotion attribution due to an inability to automatically simulate the emotional state of the cartoon character.

Existing evidence thus suggests atypical engagement of brain areas typically associated with emotional resonance and understanding in individuals with high levels of psychopathic traits. Affective impairments may partly explain the amoral behavior exhibited by individuals with psychopathy. A number of fMRI tasks have investigated moral processing in psychopathy and will be reviewed next.

Moral Processing

Psychopathy has been associated with atypical neural activity in response to moral judgment tasks, especially in the ventromedial prefrontal cortex, dorsolateral prefrontal cortex, and amygdala (Glenn, Raine, & Schug, 2009a; Glenn, Raine, Schug, Young, & Hauser, 2009b; Harenski, Harenski, Shane, & Kiehl, 2010; Pujol et al., 2012). Glenn et al. (2009a,b) used a well-known set of classic moral dilemmas in a community sample of 17 individuals with a wide range of PCL-R scores (range: 7.4–32) to inspect the associations between psychopathy and moral judgment. This set included dilemmas involving either extreme emotional content (e.g., killing a baby) or less emotional content (e.g., keeping someone else's money) as well as nonmoral dilemmas (e.g., taking the bus). Glenn et al. (2009a,b) found that, although there was no association with behavioral responses to dilemmas, psychopathy scores were negatively associated with activity in the amygdala, medial prefrontal cortex, posterior cingulate, and angular gyrus when processing the more extreme emotional dilemmas in comparison to the less extreme emotional dilemmas. Reduced activity in the amygdala was associated with all facets of psychopathy, whereas reduced activity in the other regions was associated with the interpersonal facet only. Furthermore, Glenn et al. (2009a,b) found that psychopathy scores, more specifically the lifestyle and the antisocial facets, were positively associated with increased activity in the dorsolateral prefrontal cortex when processing more versus less extreme emotional dilemmas. These results seem to suggest less involvement of regions typically involved in evaluative and affective processing and increased recruitment of regions typically associated with cognitive control during moral judgment of extreme dilemmas. Pujol et al. (2012), using the same type of dilemmas, reported similar findings in a group of incarcerated men with high levels of psychopathy (PCL-R > 20) compared with healthy controls.

Harenski et al. (2010) showed incarcerated men with different levels of psychopathic traits (high PCL-R > 30, $n = 16$; low PCL-R < 18, $n = 16$) three types of visual stimuli depicting either a moral violation, an emotionally arousing scene without moral content, or a neutral scene. Participants were asked to determine whether the picture represented a moral violation and to rate its severity. The two groups did not differ in relation to their moral judgments of the scenes. Compared with low PCL-R incarcerated men, high PCL-R individuals showed significantly lower activity in the anterior temporal cortex and ventromedial prefrontal cortex when viewing moral stimuli (contrasted to emotional nonmoral or neutral stimuli). In addition, contrary to incarcerated controls, psychopathic individuals did not show a positive modulation of the severity of moral content in the amygdala. That is, contrary to incarcerated controls, for individuals with psychopathy, amygdala response to stimuli depicting moral violations did not increase as a function of their perceived moral wrongness. In contrast, they showed a negative modulation of the posterior temporal

cortex as a function of moral wrongness ratings that was not present in the nonpsychopathic group. These results suggest that moral judgment ability may be spared in individuals with psychopathy but that they may use different strategies, or different brain regions, to compute their judgments. In particular, the differential activation of the ventromedial prefrontal cortex, important for evaluation, and the differential modulation in the amygdala and posterior temporal cortex, regions typically associated with emotional processing and mentalizing, respectively, may reflect their lack of care and insensitivity to others' well-being.

Decision Making

Individuals with psychopathy also seem to present atypical neural function during financial decision making and monetary reward. Prehn et al. (2013) compared offenders with high levels of psychopathy (PCL-R range: 23–29, $n = 11$) with a group of offenders with antisocial and borderline personality disorder ($n = 12$) and a control group of healthy individuals ($n = 13$) performing a decision-making task involving high- and low-risk economic decisions. The level of uncertainty in relation to high-risk options varied along the task. There were no behavioral differences between the groups with regard to risk-seeking behavior. The three groups were equally likely to choose the low-risk option under high uncertainty. However, compared with healthy controls, individuals with high levels of psychopathy did not show increased activity in the rostral anterior cingulate cortex before trials with high uncertainty (as contrasted against the low uncertainty condition). Individuals with high psychopathy, compared with healthy controls, also showed significantly less activity in the right inferior frontal gyrus before choosing the low-risk options under high uncertainty. However, their response in the inferior frontal gyrus was higher when choosing the high-risk option. These findings indicate that individuals with psychopathy present atypical brain function during decision making, which was interpreted by the authors as reflecting diminished emotional arousal in anticipation of possible punishment and diminished ability to emotionally represent uncertainty (which are thought to be underpinned by the rostral anterior cingulate), as well as atypical emotional and behavioral regulation (which are thought to be underpinned by the inferior frontal gyrus).

Reward

Pujara, Motzkin, Newman, Kiehl, and Koenigs (2013) have compared incarcerated men with high levels of psychopathy (high PCL-R > 32; $n = 18$) with incarcerated men with low levels of psychopathy (low PCL-R < 20; $n = 23$) while they completed a passive monetary reward and loss task. They inspected group differences in neural response to reward in the ventral striatum, a region considered to be crucial in the brain reward circuitry, but they observed no group differences.

However, when they inspected the correlations between levels of psychopathy and neural response in this region, they did observe a significant positive association in the high-psychopathy group albeit not in the low-psychopathy group.

Summary

Studies on high-risk samples using PCL-R have reported atypical function in an extensive set of brain regions (from cortical prefrontal and temporal regions to subcortical regions such as the amygdala) in response to a wide range of tasks probing different cognitive processes (from tasks involving simple viewing of emotional stimuli to tasks requiring complex moral judgment and economic decision making). Although the direction of the findings is not entirely consistent across studies, overall, these studies seem to point to reduced response in regions typically associated with affective processing and increased activity in regions typically associated with cognitive control during processing of emotional and salient stimuli.

It should be noted that some of the extant studies have only included a comparison group of individuals with antisocial behavior with low levels of psychopathy. The lack of a healthy adult comparison group precludes the establishment of a neurotypical baseline, and therefore it is difficult to know whether a significant group difference equates departure from healthy functioning and for which group. Other extant studies have included a comparison group of healthy adults only, making it difficult to disambiguate whether the differential activations found between groups are driven by high levels of psychopathy per se or are due to other characteristics present in individuals with extreme antisocial behavior. Finally, equivalent paradigms have not been routinely replicated across different laboratories, making it difficult to assess the robustness of the findings.

fMRI FINDINGS IN COMMUNITY SAMPLES WITH RATINGS OF PSYCHOPATHIC TRAITS

There is good evidence that the structure of psychopathic personality is dimensional in nature, whether it is measured by the PCL-R or by self-report measures normally used in nonforensic contexts (see Hare & Neumann, 2008, for a review). This dimensional perspective of psychopathy has led to a growing number of studies with community samples inspecting the behavioral and neurophysiological correlates of psychopathic traits in the general population (Lilienfeld & Fowler, 2006). Findings from these studies seem to mirror those observed in clinical/forensic samples and suggest that there are continuities in the mechanisms underlying psychopathy between community and forensic participants. High levels of psychopathic traits in the general population are associated

with reduced startle potentiation (Benning, Patrick, & Iacono, 2005; Justus & Finn, 2007), reduced autonomic responses to aversive images (Benning et al., 2005; Osumi, Shimazaki, Imai, Sugiura, & Ohira, 2007), reduced affective responses to others' emotions (Ali, Amorim, & Chamorro-Premuzic, 2009; Seara-Cardoso, Dolberg, Neumann, Roiser, & Viding, 2013; Seara-Cardoso, Neumann, Roiser, McCrory, & Viding, 2012), atypical moral processing (Cima, Tonnaer, & Hauser, 2010; Seara-Cardoso et al., 2012, 2013), and poor decision making during gambling tasks (Mahmut, Homewood, & Stevenson, 2008).

Our PubMed and Scopus search brought up 12 fMRI studies that included participants from the general population assessed with a self-report measure of psychopathic traits. Search terms were (“magnetic resonance” or “MR” or “MRI” or “fMRI” or “neuroimaging”) and (“psychopathy” or “psychopathic traits”). Exclusion criteria were (a) studies that did not include assessment with psychopathy measures, (b) studies of children and youth, (c) studies with psychiatric populations, and (d) studies reporting structural, resting-state or exclusively connectivity MRI analyses. Like the studies of clinical/forensic samples, studies with community samples also varied in their sample size (ranging from 10 to 200) as well as the paradigms used. In this review, we concentrate on describing those studies ($n = 7$) that utilized paradigms that can be reasonably and readily compared with paradigms used in the clinical/forensic samples. We will also briefly summarize findings from studies that, although not readily comparable with clinical/forensic findings, still give us important cues to the neural correlates of variation in psychopathic traits ($n = 5$).

Emotional Facial Expressions

In the first fMRI study of psychopathic traits in the general population, Gordon, Baird, and End (2004) reported findings from 20 male college students who performed a facial recognition task in the scanner. To measure psychopathic traits in this sample, Gordon et al. (2004) used the Psychopathy Personality Inventory (PPI; Lilienfeld & Andrews, 1996). The PPI yields eight subscales that are thought to measure lower-order factors of psychopathy: Social Potency, Stress Immunity, Fearlessness, Carefree Nonplanfulness, Blame Externalization, Machiavellian Egocentricity, Impulsive Nonconformity, and Coldheartedness. Although not originally designed to do so, the PPI conforms to a model with three higher-order factors—one factor indexing fearless dominance (consisting of the Social Potency, Stress Immunity, and Fearlessness scales), a second indexing impulsive antisociality (consisting of the Carefree Nonplanfulness, Blame Externalization, Machiavellian Egocentricity, and Impulsive Nonconformity scales), and a third factor indexing coldheartedness (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003). It is important to keep in mind that the PPI was not originally designed to mirror the

structure of the PCL-R in the general population, and, although the PPI does share some variance with the features measured by the PCL-R, its factors may not directly map the factors from the PCL-R (Malterer, Lilienfeld, Neumann, & Newman, 2010). Inside the scanner, participants were shown a target picture in the beginning of each block (a male or a female with an emotional expression of sadness, fear, anger, or joy) and had to press a button every time a subsequent picture depicted the same emotion (emotion condition) or the same identity (identity condition). These conditions were contrasted against rest, and differences between high and low psychopathic trait groups were inspected. A median split of the participants' scores on the first factor of the PPI was used to establish group assignment. When performing the emotion condition, the group with high levels of psychopathic traits presented significantly less activation in the inferior frontal gyrus, medial prefrontal cortex, and amygdala and greater activation in the dorsolateral prefrontal cortex and primary visual cortex than the group with low levels of psychopathic traits. In contrast, there were no significant differences between the groups in the identity recognition condition. These findings are in line with those from forensic populations and suggest diminished input from regions typically associated with affect processing during the evaluation of emotional facial expressions. Interestingly, Gordon et al. (2004) also found that, when dividing groups on the basis of scores of the second factor of the PPI, the group with high scores presented increased amygdala response during emotional recognition in comparison to the low group. Although the sample of this study was relatively small, and the reliance on median split for group assignment is less than ideal, this study presented important preliminary evidence of the similarities of neural correlates of psychopathic traits between forensic and nonforensic samples.

In a much larger sample of 200 healthy volunteers from the community, Carré, Hyde, Neumann, Viding, and Hariri (2013) have also shown that amygdala reactivity to fearful faces was negatively associated with interpersonal traits of psychopathy as measured by the Self-Report Psychopathy questionnaire (SRP; Paulhus, Neumann, & Hare, in press). The SRP, developed by Hare and colleagues as an extension of the PCL-R to measure psychopathic traits in the general population, mirrors the latent structure of the PCL-R. Like the PCL-R, the SRP assesses four facets of psychopathic traits—interpersonal, affective, lifestyle, and antisocial—that can also be modeled in terms of the traditional two-factor dimensions. Interestingly, and in line with the findings of Gordon et al. (2004), Carré et al. (2013) also found that amygdala reactivity to angry faces was positively correlated with lifestyle traits of psychopathy. These findings suggest that different facets of psychopathy might present differential (and potentially stimulus-dependent) associations with amygdala reactivity.

Pardini and Phillips (2010) used a gender recognition task to inspect neural responses to sad, fearful, angry, happy, and neutral faces in a cohort of 20 chronically violent men, compared with 22 nonviolent men. Compared with nonviolent

men, violent men exhibited lower amygdala response to happy faces (as compared with neutral faces) and increased dorsomedial prefrontal cortex response to fearful faces (as compared with neutral faces). No associations between psychopathic traits, as measured by the SRP, and amygdala response to any of the emotions were found within the group of violent men. However, as the authors point out, the lack of significant findings could have been due to limited power to detect significant effects as a consequence of the limited number of trials per emotion type as well as of the small sample size of the group of violent men.

Han, Alders, Greening, Neufeld, and Mitchell (2012) employed an emotion recognition task that isolated different parts of the face to inspect differences in fear processing in 32 individuals with high *versus* individuals with low scores of the Coldheartedness scale of the PPI-Revised (PPI-R; Lilienfeld & Widows, 2005). Inside the scanner, participants identified the emotion portrayed in “whole faces,” in “eyes-only” (cropped from the whole face so that only the eye region could be seen), or in faces with “eyes-occluded” (in which the eye region was occluded from the whole face, leaving the rest of the face visible). When the “eyes-occluded” condition (i.e., the condition where critical information relating to fear was removed) was contrasted with the “eyes-only” condition (i.e., the condition containing the critical information regarding fear), the group with high levels of coldheartedness presented significantly less activity in the amygdala than the group with low levels of the trait. Lower activity in the amygdala was also reported for this group for happy “eyes-only” versus “eyes-occluded” conditions (similarly presenting a contrast with the most critical part of the emotional expression, the mouth in this case, missing vs. isolated). These findings are, however, complicated to interpret. The stimuli are very unusual and do not represent something that is typically encountered. It is therefore difficult to ascertain that amygdala response in the reported contrasts pertains to critical aspects of the stimuli that index fear (eyes), as opposed to the stimuli being very salient because they are unusual. Because the conditions that were contrasted against one another both had unusual elements (showing one part of the face only vs. occluding a salient feature of the face), it is also unclear what cognitive process is being isolated in the analysis.

Moral Processing

Only one published study to date has investigated the association between psychopathic traits and moral processing in the general population. Harenski, Sang, and Hamann (2009), using the same stimuli reported in the previous section in Harenski et al. (2010), found that psychopathic traits measured by the PPI, in a small sample of 10 females, were negatively correlated with medial prefrontal cortex activity in response to moral stimuli when contrasted to unpleasant stimuli without moral content. Additionally, amygdala response to moral

stimuli in comparison to a baseline task (a simple cognitive task where participants had to determine whether numbers presented were odd or even) was negatively correlated with scores of coldheartedness. Although these results are in line with the findings that the same authors report for their forensic sample, it should be noted that the small sample used in this study may not be ideal for correlational analyses. It is also not entirely clear whether the reduced amygdala activity reported for those with higher levels of coldheartedness relates to moral processing specifically or whether it is driven by differences between stimuli type (aversive social stimuli vs. numbers) or even between computational demands in the two tasks.

Reward

Three studies to date have inspected the neural correlates of reward in relation to psychopathic traits in the general population (Bjork, Chen, & Hommer, 2012; Buckholtz et al., 2010; Carré et al., 2013). Buckholtz et al. (2010) reported findings from a sample of 20 males who performed a monetary incentive delay task inside the scanner. They observed that during monetary reward anticipation (contrasted to anticipation in no-reward trials), psychopathic traits were positively associated with activity in the ventral striatum. Interestingly, this association was specific to impulsive antisocial features of psychopathic traits (as measured by the second factor of the PPI-R). Bjork et al. (2012) replicated these findings and extended them by showing that activity in the medial prefrontal cortex during passive reward anticipation was also positively correlated with levels of total psychopathic traits (but note that sample size for the analyses varied from 5 to 17 participants).

Carré et al. (2013), with a large sample of 200 participants, found that psychopathic traits, measured by the SRP, were also associated with ventral striatum response during reward (positive feedback) in a card guessing game. Interestingly, they found divergent associations between the unique variance (i.e., variance left in each facet after the variance shared with all other facets is removed) of the lifestyle and of the antisocial facets and ventral striatum response to reward. While the unique variance in lifestyle traits presented a negative association with ventral striatum response to reward, the unique variance in antisocial traits presented a positive association, suggesting that hyperreactivity to reward in the ventral striatum may be specifically associated with antisocial traits and not with other characteristics of the psychopathic personality. It should be noted, however, that the task used by Carré et al. (2013) relied on a block design that prevented the assessment of different phases of reward processing and, therefore, the assessment of the extent to which associations of psychopathic traits with ventral striatum response were related to the anticipation of receiving a reward or to indeed receiving a reward.

Other Forms of Social Interaction

Five additional studies investigated the neural correlates of psychopathic traits in the general population, but these studies used paradigms that have not been employed in forensic/clinical samples. They focused on assessing cooperation and noncooperation (Rilling et al., 2007), response to unfair offers (Osumi et al., 2012; Vieira et al., 2013), delivery of punishment to others (Molenberghs et al., 2014), and deception (Fullam, McKie, & Dolan, 2009). Individuals with high levels of psychopathic traits, compared to individuals with low levels of these traits, presented less amygdala reactivity when their cooperation efforts were not reciprocated (Rilling et al., 2007). Psychopathic traits were also negatively associated with amygdala reactivity to unfair offers in one study (Osumi et al., 2012). Vieira et al. (2013) reported that individuals with higher psychopathy scores appeared less sensitive to the fairness of the offers they received. Although there was no straightforward association between psychopathic traits and brain activity, associations between behavioral performance and neural activity in the rostral anterior cingulate differed for high versus low psychopathic trait groups, which might indicate differential coding of unfairness in the two groups. Finally, individual differences in psychopathic traits were negatively correlated with neural response in the ventromedial prefrontal cortex and insular cortex in a task during which participants told lies (Fullam et al., 2009), as well as in a task where participants delivered punishment to others (Molenberghs et al., 2014). This interesting overall pattern of lower reactivity in brain regions typically involved in basic affect processing and affect regulation could be interpreted as reflecting low aversiveness to negative, unpleasant, social interactions in individuals with high levels of psychopathic traits.

Summary

Findings from fMRI research largely support the notion that psychopathy is a dimensional construct, with findings from community samples typically mirroring those observed in clinical/forensic samples. Overall, fMRI research on the neural correlates of psychopathic traits in the general population indicate that variance in these traits is negatively associated with neural response in brain areas typically associated with affect processing (e.g., amygdala, inferior frontal gyrus) during tasks that involve affective stimuli, from basic emotion recognition to more complex and sophisticated social interactions.

Research on the general population has also shown that variance in psychopathic traits appears to be positively associated with activity in brain regions typically engaged during reward processing (e.g., ventral striatum) when performing tasks involving reward. This is a different pattern of findings to that seen in a reward processing study in a forensic sample (Pujara et al., 2013; see previous section), where no group differences in ventral striatum response to reward were

reported for those with high versus low psychopathic traits. Curiously, a positive correlation between ventral striatum response and levels of psychopathic traits was observed within the group of offenders with high psychopathic traits. Further studies in larger forensic samples are required to investigate how psychopathic traits within this population relate to reward processing.

Of interest, findings from the general population also seem to indicate that different facets of psychopathy might present differential (and potentially stimulus-dependent) associations with amygdala reactivity to affective stimuli. This pattern of differential, and at times opposing, relationships of the two dimensions of psychopathy with criterion variables of affect processing is consistent with the suggestion that these two dimensions tap into somewhat separable constructs that may exert suppressor effects on each other (e.g., Hicks & Patrick, 2006; Uzieblo, Verschuere, van den Bussche, & Crombez, 2010). Behavioral studies have shown that after partialing out the shared variance between the two dimensions, the affective-interpersonal dimension is negatively associated with emotion-related variables such as fearfulness, emotional reactivity, and empathic concern, whereas the lifestyle-antisocial dimension is positively associated with these variables (e.g., Hicks & Patrick, 2006; Seara-Cardoso et al., 2012; Uzieblo et al., 2010). These relationships are typically stronger than equivalent bivariate analyses (i.e., when shared variance between the two dimensions is not removed). At the neural level, this pattern has been reported in children with variable levels of callous-unemotional traits (akin to psychopathic traits in adults) in response to emotional stimuli (Lockwood et al., 2013; Lozier, Cardinale, VanMeter, & Marsh, 2014; Sebastian et al., 2012) and typical adults (Carré et al., 2013), with the affective-interpersonal features of psychopathic personality being negatively associated with activity in emotion-processing areas of the brain (e.g., amygdala and insula) and lifestyle-antisocial features showing the opposite association. This suggests that, although affective-interpersonal and lifestyle-antisocial features can co-occur, the unique aspects of each dimension (i.e., those not shared with the other dimension) may reflect distinct underlying vulnerabilities, one corresponding to low emotional reactivity and the other to increased emotional reactivity and a basic weakness in inhibitory control systems (Patrick, Hicks, Nichol, & Krueger, 2007). It should be noted, however, that these effects have not yet been reported in adult forensic samples. Furthermore, although the use of partial correlations is a powerful and informative technique to identify associations between different variables, it also poses some difficulties in the interpretation of results (Lynam, Hoyle, & Newman, 2006). The most important one is the difficulty in knowing exactly what construct is left once the variance of another correlated construct is removed (Lynam et al., 2006). The replication of these findings using a group comparison approach, with groups defined by high and low levels on the two dimensions, would provide important further validation of these results.

DOES fMRI RESEARCH INTO “NORMAL” PERSONALITY CORROBORATE THE FINDINGS FROM CLINICAL AND COMMUNITY STUDIES OF PSYCHOPATHY?

In the final section of this article, we will briefly consider whether fMRI research into variation of traits from a general model of personality corroborates the findings from clinical and community studies of psychopathy. The Five-Factor Model (FFM) categorizes personality traits in five broad domains: Extraversion, Neuroticism, Agreeableness, Conscientiousness, and Openness/Intellect (e.g., Costa & McCrae, 1985). Lynam and colleagues (Miller & Lynam, 2003; Miller, Lynam, Widiger, & Leukefeld, 2001; Widiger & Lynam, 1998) have made substantial contributions to investigating psychopathy from the perspective of the FFM of personality. They propose that psychopathy corresponds to a constellation of traits from a general model of personality functioning, and thus can be understood as an extreme variant of common dimensions of personality. Factor 1 psychopathic traits map onto low Agreeableness (e.g., low altruism, compliance, modesty) and low Neuroticism (e.g., low anxiety, depression, vulnerability to stress), whereas Factor 2 psychopathic traits map onto low Agreeableness, low Conscientiousness (e.g., low dutifulness, self-discipline, deliberation) and high Neuroticism (e.g., high angry hostility, impulsiveness).

Only two fMRI studies to date have explicitly focused on psychopathy dimensions as derived from the FFM (Hyde, Byrd, Votruba-Drzal, Hariri, & Manuck, 2014; Sadeh et al., 2013). Sadeh et al. (2013) computed the fearless-dominance and impulsive-antisociality scales of the PPI from the NEO Five-Factor Inventory (NEO-FFI; Costa & McCrae, 1992) as described in Ross, Benning, Patrick, Thompson, and Thurston (2009). This study had 49 participants (30 females) and employed an emotion-word Stroop task where the participants named the ink color of positive, negative, or neutral words. The neural responses to positive (positive–neutral contrast) and negative (negative–neutral contrast) were extracted and related to fearless-dominance or impulsive-antisociality scores. Fearless-dominance was associated with behavioral interference (higher error rate) to positive words, with activation in the right superior frontal gyrus mediating the relationship between this personality dimension and errors to positive words. The impulsive-antisocial dimension was associated with response interference (higher reaction times) to both positive and negative words, with activity in the temporal cortex (extending to the orbitofrontal cortex and inferior frontal gyrus) and in the medial temporal gyrus mediating the association with this personality dimension and higher reaction times to positive words.

Similarly, Hyde et al. (2014) derived psychopathy and anti-social personality disorder scores from the NEO Personality

Inventory Revised (NEO PI-R; Costa & McCrae, 1995) in a sample of 103 participants (53 females) to inspect amygdala reactivity to fearful and angry faces with the same task as Carré et al. (2013). The authors derived a total psychopathy score and an antisocial personality disorder score using the method described in Lynam and Widiger (2001), as well as factor scores based on the PCL-R as described in Derefinko and Lynam (2006). Interestingly, they found a suppressor effect of psychopathy and antisocial personality disorder scores on amygdala reactivity to angry faces. Total psychopathy score presented a negative association with amygdala reactivity to angry faces, but only after shared variance with scores of antisocial personality disorder was accounted for. However, when examining relationships with amygdala reactivity using the PCL-R-based factors, the authors, rather surprisingly, did not find any significant results.

No other functional imaging study to date has taken the FFM approach, but a number of studies have focused on single personality dimensions that have relevance for psychopathy. Most prominently, several studies have demonstrated that Neuroticism (which is negatively associated with interpersonal-affective psychopathic traits and positively associated with lifestyle-antisocial traits) is positively associated with brain reactivity to a variety of affective stimuli (e.g., emotional faces, pictures, short films) in the amygdala, hippocampus, and medial prefrontal cortex (Britton, Ho, Taylor, & Liberzon, 2007; Canli et al., 2001; Cremers et al., 2010; Haas, Constable, & Canli, 2008; Harenski et al., 2009; Hooker, Verosky, Miyakawa, Knight, & D'Esposito, 2008).

Additional studies using comparable paradigms to those employed in clinical/forensic samples, or in community samples using self-report psychopathy measures, are needed. Importantly, if psychopathy is considered as a constellation of traits from a general model of personality functioning, and given how it is thought to map onto extreme variants of Agreeableness and Conscientiousness, studies investigating the neural correlates of these personality traits (especially when they co-occur) in paradigms that are comparable to those employed in fMRI studies of psychopathy are necessary. Such studies would enable us to assess how closely the findings from fMRI studies where psychopathic traits are defined using the FFM approach correspond to findings from the studies where psychopathy/psychopathic traits are defined using measures that are directed at recording the disordered aspects of personality.

SUMMARY AND FUTURE DIRECTIONS

Neuroimaging findings from community samples typically mirror those observed in clinical/forensic samples, and they largely support the notion that psychopathy is a dimensional construct. Using different paradigms, measurement tools, and populations (both disordered and community), the weight of the evidence seems to suggest that individuals with high levels of psychopathic traits show lower activity in a number of

affect-processing areas, in particular the amygdala and anterior insula, to emotional/salient stimuli (but see exceptions: Decety, Skelly, et al., 2013; Müller et al., 2003; Schneider et al., 2000). The data also suggest that attenuated brain reactivity to emotional/salient stimuli may be dependent on the precise content of the task (e.g., the type of salient stimuli used). Extant research also implicates increased neural response in regions typically associated with reward processing and cognitive control, in particular the ventral striatum and dorsolateral prefrontal cortex in tasks involving moral processing, decision making, and reward. Finally, the data suggest that affective-interpersonal and lifestyle-antisocial facets of psychopathy may be associated with different patterns of atypical neural activity, which deserves future investigation. For example, attenuated reactivity to at least some emotional stimuli is more commonly associated with affective-interpersonal features of psychopathy, whereas exaggerated reactivity to reward appears to pertain to lifestyle-antisocial features of psychopathy.

Unfortunately, in addition to specific limitations and future directions approached in the sections above, there are a number of limitations to the present research that need to be addressed in future studies. First, different laboratories have used different stimuli and paradigms with variable control conditions. This prevents a comprehensive and systematic evaluation of the contradictory findings. Second, there is still substantial work to be done to gain a fine-grained picture of the precise cognitive-affective deficits associated with psychopathy. Many extant studies have relied on tasks that engage multiple cognitive operations and have not always included contrast conditions that would enable the parsing apart of specific cognitive-affective processes. Future studies would benefit from the development of carefully designed tasks that allow the isolation of distinct processes that contribute to complex phenomena such as empathy, moral processing, and decision making. Only with such tasks will researchers be able to systematically identify and characterize the neural correlates of psychopathy. Third, it will be important to develop more ecologically valid tasks. These could, for example, involve scenarios delivered in a virtual reality setup. Fourth, many of the studies presented here have had relatively small sample sizes. Both sample-size and task-design issues can render studies underpowered and consequently more likely to produce unreliable findings.

In sum, the variable sample sizes and type of control groups across studies, combined with the wide selection of different paradigms (and variable degree of inference that these different paradigms afford) and disparity of functional analyses approaches (e.g., whole-brain vs. region-of-interest analyses; regions-of-interest functionally vs. anatomically defined; BOLD parameter estimates extracted from the peak voxels vs. spheres around peak voxels vs. averaged across clusters of activation) make it difficult to interpret and weight any seemingly contradictory findings in the fMRI studies of

psychopathy. The field would benefit from collaborative efforts to precisely specify those cognitive-affective processes that should be interrogated with regard to psychopathic personality and the best fMRI paradigms to do that, which ideally would be tested in reasonably sized samples (from clinical/forensic and community settings and with appropriate control groups) and replicated across different laboratories.

On a final note, studies of children and youth with varying levels of conduct problems and psychopathic/callous-unemotional traits have, on the whole, produced comparable findings to those seen in adults with psychopathy (see, e.g., Blair, 2013; Viding & McCrory, 2012, for recent reviews). Future efforts should include longitudinal imaging data collection, as well as the use of comparable tasks in children and adults. Such research is of crucial interest to assess functional brain development longitudinally, including the identification of possible “brain biomarkers” that might predict future behavioral outcomes.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: Preparation of this article was supported by Grant SFRH/BD/60279/2009 from Fundação para a Ciência e Tecnologia awarded to ASC and RES-062-23-2202 from the Economic and Social Research Council awarded to EV. EV is a Royal Society Wolfson Research Merit Award holder.

Note

1. The interested reader is referred to an excellent review by Yang and Raine (2009) as well as very recent empirical articles (e.g., Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012; Gregory et al., 2012; Ly et al., 2012) regarding the structural brain imaging findings on criminal psychopathy.

References

- Ali, F., Amorim, I. S., & Chamorro-Premuzic, T. (2009). Empathy deficits and trait emotional intelligence in psychopathy and Machiavellianism. *Personality and Individual Differences, 47*, 758–762.
- Amodio, D. M., & Frith, C. D. (2006). Meeting of minds: The medial frontal cortex and social cognition. *Nature Reviews Neuroscience, 7*, 268–277.
- Benning, S. D., Patrick, C. J., Hicks, B. M., Blonigen, D.M., & Krueger, R. F. (2003). Factor structure of the Psychopathic Personality Inventory: Validity and implications for clinical assessment. *Psychological Assessment, 15*, 340–350.
- Benning, S. D., Patrick, C. J., & Iacono, W. G. (2005). Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. *Psychophysiology, 42*, 753–762.
- Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., et al. (2005). Deficient fear conditioning in psychopathy: A functional magnetic resonance imaging study. *Archives of General Psychiatry, 62*, 799–805.
- Bjork, J. M., Chen, G., & Hommer, D. W. (2012). Psychopathic tendencies and mesolimbic recruitment by cues for instrumental and passively obtained rewards. *Biological Psychology, 89*, 408–415.
- Blair, R. J. R. (2003). Facial expressions, their communicatory functions and neuro-cognitive substrates. *Philosophical Transactions of the Royal Society B: Biological Sciences, 358*(1431), 561–572.
- Blair, R. J. R. (2008). The amygdala and ventromedial prefrontal cortex: Functional contributions and dysfunction in psychopathy. *Philosophical Transactions of the Royal Society B: Biological Sciences, 363*(1503), 2557–2565.
- Blair, R. J. R. (2013). The neurobiology of psychopathic traits in youths. *Nature Reviews Neuroscience, 14*, 786–799.
- Blair, R. J. R., Mitchell, D., & Blair, K. (2005). *The psychopath: Emotion and the brain*. Malden, MA: Blackwell.
- Britton, J. C., Ho, S. H., Taylor, S. F., & Liberzon, I. (2007). Neuroticism associated with neural activation patterns to positive stimuli. *Psychiatry Research: Neuroimaging, 156*, 263–267.
- Buckholtz, J. W., Treadway, M. T., Cowan, R. L., Woodward, N. D., Benning, S. D., Li, R., et al. (2010). Mesolimbic dopamine reward system hypersensitivity in individuals with psychopathic traits. *Nature Neuroscience, 13*, 419–421.
- Canli, T., Zhao, Z., Desmond, J. E., Kang, E., Gross, J., & Gabrieli, J. D. E. (2001). An fMRI study of personality influences on brain reactivity to emotional stimuli. *Behavioral Neuroscience, 115*, 33–42.
- Carré, J. M., Hyde, L. W., Neumann, C. S., Viding, E., & Hariri, A. R. (2013). The neural signatures of distinct psychopathic traits. *Social Neuroscience, 8*, 122–135.
- Cima, M., Tonnaer, F., & Hauser, M. D. (2010). Psychopaths know right from wrong but don't care. *Social Cognitive and Affective Neuroscience, 5*, 59–67.
- Cleckley, H. (1941). *The mask of sanity* (1st ed.). St Louis, MO: Mosby.
- Costa, P. T., & McCrae, R. R. (1985). Validation of the five-factor model of personality across instruments and observers. *Journal of Personality and Social Psychology, 52*, 81–90.
- Costa, P. T., Jr., & McCrae, R. R. (1992). *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO-FFI) professional manual*. Odessa, FL: Psychological Assessment Resources.
- Costa, P. T., Jr., & McCrae, R. R. (1995). Domains and facets: Hierarchical personality assessment using the Revised NEO Personality Inventory. *Journal of Personality Assessment, 64*, 21–50.

- Craig, A. D. (2009). How do you feel—now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, **10**, 59–70.
- Cremers, H. R., Demenescu, L. R., Aleman, A., Renken, R., van Tol, M. J., van der Wee, N. J. A., et al. (2010). Neuroticism modulates amygdala-prefrontal connectivity in response to negative emotional facial expressions. *NeuroImage*, **49**, 963–970.
- Critchley, H., & Harrison, N. (2013). Visceral influences on brain and behavior. *Neuron*, **77**, 624–638.
- Critchley, H., Wiens, S., Rotshtein, P., Öhman, A., & Dolan, R. J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience*, **7**, 189–195.
- Decety, J., Chen, C., Harenski, C., & Kiehl, K. A. (2013). An fMRI study of affective perspective taking in individuals with psychopathy: Imagining another in pain does not evoke empathy. *Frontiers in Human Neuroscience*, **7**, 489.
- Decety, J., Skelly, L. R., & Kiehl, K. A. (2013). Brain response to empathy-eliciting scenarios involving pain in incarcerated individuals with psychopathy. *JAMA Psychiatry*, **70**, 638–645.
- Decety, J., Skelly, L., Yoder, K. J., & Kiehl, K. A. (2014). Neural processing of dynamic emotional facial expressions in psychopaths. *Social Neuroscience*, **9**, 36–49.
- Deeley, Q., Daly, E., Surguladze, S., Tunstall, N., Mezey, G., Beer, D., et al. (2006). Facial emotion processing in criminal psychopathy: Preliminary functional magnetic resonance imaging study. *British Journal of Psychiatry*, **189**, 533–539.
- Derefinko, K. J., & Lynam, D. R. (2006). Convergence and divergence among self-report psychopathy measures: A personality-based approach. *Journal of Personality Disorders*, **20**, 261–280.
- Eisenberg, N. (2000). Emotion, regulation, and moral development. *Annual Review of Psychology*, **51**, 665–697.
- Ermer, E., Cope, L. M., Nyalakanti, P. K., Calhoun, V. D., & Kiehl, K. A. (2012). Aberrant paralimbic gray matter in criminal psychopathy. *Journal of Abnormal Psychology*, **121**, 649–658.
- Fan, Y., Duncan, N. W., de Greck, M., & Northoff, G. (2011). Is there a core neural network in empathy? An fMRI based quantitative meta-analysis. *Neuroscience and Biobehavioral Reviews*, **35**, 903–911.
- Fullam, R. S., McKie, S., & Dolan, M. C. (2009). Psychopathic traits and deception: Functional magnetic resonance imaging study. *British Journal of Psychiatry*, **194**, 229–235.
- Glenn, A. L., Raine, A., & Schug, R. A. (2009a). The neural correlates of moral decision-making in psychopathy. *Molecular Psychiatry*, **14**, 5–6.
- Glenn, A. L., Raine, A., Schug, R. A., Young, L., & Hauser, M. (2009b). Increased DLPFC activity during moral decision-making in psychopathy. *Molecular Psychiatry*, **14**, 909–911.
- Gordon, H. L., Baird, A. A., & End, A. (2004). Functional differences among those high and low on a trait measure of psychopathy. *Biological Psychiatry*, **56**, 516–521.
- Gregory, S., Ffytche, D., Simmons, A., Kumari, V., Howard, M., Hodgins, S., et al. (2012). The antisocial brain: Psychopathy matters: A structural MRI investigation of antisocial male violent offenders. *Archives of General Psychiatry*, **69**, 962–972.
- Haas, B. W., Constable, R. T., & Canli, T. (2008). Stop the sadness: Neuroticism is associated with sustained medial prefrontal cortex response to emotional facial expressions. *NeuroImage*, **42**, 385–392.
- Han, T., Alders, G. L., Greening, S. G., Neufeld, R. W. J., & Mitchell, D. G. V. (2012). Do fearful eyes activate empathy-related brain regions in individuals with callous traits? *Social Cognitive and Affective Neuroscience*, **7**, 958–968.
- Hare, R. D. (1980). A research scale for the assessment of psychopathy in criminal populations. *Personality and Individual Differences*, **1**, 111–119.
- Hare, R. D. (1991). *The Hare Psychopathy Checklist-Revised*. Toronto, Canada: Multi-Health Systems.
- Hare, R. D. (2003). *The Hare Psychopathy Checklist-Revised* (2nd ed.). Toronto, Canada: Multi-Health Systems.
- Hare, R. D., & Neumann, C. S. (2008). Psychopathy as a clinical and empirical construct. *Annual Review of Clinical Psychology*, **4**, 217–246.
- Harenski, C. L., Harenski, K. A., Shane, M. S., & Kiehl, K. A. (2010). Aberrant neural processing of moral violations in criminal psychopaths. *Journal of Abnormal Psychology*, **119**, 863–874.
- Harenski, C. L., Sang, H. K., & Hamann, S. (2009). Neuroticism and psychopathy predict brain activation during moral and nonmoral emotion regulation. *Cognitive, Affective and Behavioral Neuroscience*, **9**, 1–15.
- Hicks, B. M., & Patrick, C. J. (2006). Psychopathy and negative emotionality: Analyses of suppressor effects reveal distinct relations with emotional distress, fearfulness, and anger-hostility. *Journal of Abnormal Psychology*, **115**, 276–287.
- Hooker, C. I., Verosky, S. C., Miyakawa, A., Knight, R. T., & D’Esposito, M. (2008). The influence of personality on neural mechanisms of observational fear and reward learning. *Neuropsychologia*, **46**, 2709–2724.
- Hyde, L. W., Byrd, A. L., Votruba-Drzal, E., Hariri, A. R., & Manuck, S. B. (2014). Amygdala reactivity and negative emotionality: Divergent correlates of antisocial personality and psychopathy traits in a community sample. *Journal of Abnormal Psychology*, **123**, 214–224.
- Justus, A. N., & Finn, P. R. (2007). Startle modulation in non-incarcerated men and women with psychopathic traits. *Personality and Individual Differences*, **43**, 2057–2071.
- Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., et al. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Biological Psychiatry*, **50**, 677–684.
- Lamm, C., Decety, J., & Singer, T. (2011). Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. *NeuroImage*, **54**, 2492–2502.
- Lilienfeld, S. O., & Andrews, B. P. (1996). Development and preliminary validation of a self-report measure of psychopathic personality traits in noncriminal populations. *Journal of Personality Assessment*, **66**, 488–524.
- Lilienfeld, S. O., & Fowler, K. A. (2006). The self-report assessment of psychopathy: Problems, pitfalls, and promises. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 107–132). New York: Guilford Press.

- Lilienfeld, S. O., & Widows, M. R. (2005). *Psychopathic Personality Inventory-Revised: Professional manual*. Lutz, FL: Psychological Assessment Resources.
- Lockwood, P. L., Sebastian, C. L., McCrory, E. J., Hyde, Z. H., Gu, X., De Brito, S. A., et al. (2013). Association of callous traits with reduced neural response to others' pain in children with conduct problems. *Current Biology*, **23**, 901–905.
- Lozier, L. M., Cardinale, E. M., VanMeter, J. W., & Marsh, A. A. (2014). Mediation of the relationship between callous-unemotional traits and proactive aggression by amygdala response to fear among children with conduct problems, *JAMA Psychiatry*, **71**, 627–636.
- Ly, M., Motzkin, J. C., Philippi, C. L., Kirk, G. R., Newman, J. P., Kiehl, K. A., et al. (2012). Cortical thinning in psychopathy. *American Journal of Psychiatry*, **169**, 743–749.
- Lynam, D. R., Hoyle, R. H., & Newman, J. P. (2006). The perils of partialling: Cautionary tales from aggression and psychopathy. *Assessment*, **13**, 328–341.
- Lynam, D. R., & Widiger, T. A. (2001). Using the five-factor model to represent the *DSM-IV* personality disorders: An expert consensus approach. *Journal of Abnormal Psychology*, **110**, 401–412.
- Mahmut, M. K., Homewood, J., & Stevenson, R. J. (2008). The characteristics of non-criminals with high psychopathy traits: Are they similar to criminal psychopaths? *Journal of Research in Personality*, **42**, 679–692.
- Malterer, M. B., Lilienfeld, S. O., Neumann, C. S., & Newman, J. P. (2010). Concurrent validity of the Psychopathic Personality Inventory with offender and community samples. *Assessment*, **17**, 3–15.
- Meffert, H., Gazzola, V., Den Boer, J. A., Bartels, A. A. J., & Keysers, C. (2013). Reduced spontaneous but relatively normal deliberate vicarious representations in psychopathy. *Brain*, **136**, 2550–2562.
- Mier, D., Haddad, L., Diers, K., Dressing, H., Meyer-Lindenberg, A., & Kirsch, P. (2014). Reduced embodied simulation in psychopathy. *World Journal of Biological Psychiatry*, **15**, 479–487.
- Miller, J. D., & Lynam, D. R. (2003). Psychopathy and the five-factor model of personality: A replication and extension. *Journal of Personality Assessment*, **81**, 168–178.
- Miller, J. D., Lynam, D. R., Widiger, T. A., & Leukefeld, C. (2001). Personality disorders as extreme variants of common personality dimensions: Can the five-factor model adequately represent psychopathy? *Journal of Personality*, **69**, 253–276.
- Molenberghs, P., Bosworth, R., Nott, Z., Louis, W. R., Smith, J. R., Amiot, C. E., et al. (2014). The influence of group membership and individual differences in psychopathy and perspective taking on neural responses when punishing and rewarding others. *Human Brain Mapping*. doi: 10.1002/hbm.22527
- Müller, J. L., Sommer, M., Döhl, K., Weber, T., Schmidt-Wilcke, T., & Hajak, G. (2008). Disturbed prefrontal and temporal brain function during emotion and cognition interaction in criminal psychopathy. *Behavioral Sciences and the Law*, **26**, 131–150.
- Müller, J. L., Sommer, M., Wagner, V., Lange, K., Taschler, H., Röder, C. H., et al. (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. *Biological Psychiatry*, **54**, 152–162.
- Osumi, T., Nakao, T., Kasuya, Y., Shinoda, J., Yamada, J., & Ohira, H. (2012). Amygdala dysfunction attenuates frustration-induced aggression in psychopathic individuals in a non-criminal population. *Journal of Affective Disorders*, **142**, 331–338.
- Osumi, T., Shimazaki, H., Imai, A., Sugiura, Y., & Ohira, H. (2007). Psychopathic traits and cardiovascular responses to emotional stimuli. *Personality and Individual Differences*, **42**, 1391–1402.
- Pardini, D. A., & Phillips, M. (2010). Neural responses to emotional and neutral facial expressions in chronically violent men. *Journal of Psychiatry and Neuroscience*, **35**, 390–398.
- Patrick, C. J., Hicks, B. M., Nichol, P. E., & Krueger, O. F. (2007). A bifactor approach to modeling the structure of the Psychopathy Checklist-Revised. *Journal of Personality Disorders*, **21**, 118–141.
- Paulhus, D. L., Neumann, C. S., & Hare, R. D. (in press). *Manual for the Self-Report Psychopathy Scale* (4th ed.). Toronto, Canada: Multi-Health Systems.
- Prehn, K., Schulze, L., Rossmann, S., Berger, C., Vohs, K., Fleischer, M., et al. (2013). Effects of emotional stimuli on working memory processes in male criminal offenders with borderline and antisocial personality disorder. *World Journal of Biological Psychiatry*, **14**, 71–78.
- Pujara, M., Motzkin, J. C., Newman, J. P., Kiehl, K. A., & Koenigs, M. (2013). Neural correlates of reward and loss sensitivity in psychopathy. *Social Cognitive and Affective Neuroscience*, **9**, 794–801.
- Pujol, J., Batalla, I., Contreras-Rodríguez, O., Harrison, B. J., Pera, V., Hernández-Ribas, R., et al. (2012). Breakdown in the brain network subserving moral judgment in criminal psychopathy. *Social Cognitive and Affective Neuroscience*, **7**, 917–923.
- Rilling, J. K., Glenn, A. L., Jairam, M. R., Pagnoni, G., Goldsmith, D. R., Elfenbein, H. A., et al. (2007). Neural correlates of social cooperation and non-cooperation as a function of psychopathy. *Biological Psychiatry*, **61**, 1260–1271.
- Ross, S. R., Benning, S. D., Patrick, C. J., Thompson, A., & Thurston, A. (2009). Factors of the Psychopathic Personality Inventory: Criterion-related validity and relationship to the BIS/BAS and five-factor models of personality. *Assessment*, **16**, 71–87.
- Rushworth, M. F., Buckley, M. J., Behrens, T. E., Walton, M. E., & Bannerman, D. M. (2007). Functional organization of the medial frontal cortex. *Current Opinion in Neurobiology*, **17**, 220–227.
- Rushworth, M. F., Kolling, N., Sallet, J., & Mars, R. B. (2012). Valuation and decision-making in frontal cortex: One or many serial or parallel systems? *Current Opinion in Neurobiology*, **22**, 946–955.
- Sadeh, N., Spielberg, J. M., Heller, W., Herrington, J. D., Engels, A. S., Warren, S. L., et al. (2013). Emotion disrupts neural activity during selective attention in psychopathy. *Social Cognitive and Affective Neuroscience*, **8**, 235–246.
- Schneider, F., Habel, U., Kessler, C., Posse, S., Grodd, W., & Müller-Gärtner, H. W. (2000). Functional imaging of conditioned

- aversive emotional responses in antisocial personality disorder. *Neuropsychobiology*, **42**, 192–201.
- Seara-Cardoso, A., Dolberg, H., Neumann, C., Roiser, J. P., & Viding, E. (2013). Empathy, morality and psychopathic traits in women. *Personality and Individual Differences*, **55**, 328–333.
- Seara-Cardoso, A., Neumann, C., Roiser, J., McCrory, E., & Viding, E. (2012). Investigating associations between empathy, morality and psychopathic personality traits in the general population. *Personality and Individual Differences*, **52**, 67–71.
- Sebastian, C. L., McCrory, E. J. P., Cecil, C. A. M., Lockwood, P. L., De Brito, S. A., Fontaine, N. M. G., et al. (2012). Neural responses to affective and cognitive theory of mind in children with conduct problems and varying levels of callous-unemotional traits. *Archives of General Psychiatry*, **69**, 814–822.
- Sommer, M., Sodian, B., Döhnel, K., Schwerdtner, J., Meinhardt, J., & Hajak, G. (2010). In psychopathic patients emotion attribution modulates activity in outcome-related brain areas. *Psychiatry Research: Neuroimaging*, **182**, 88–95.
- Uzieblo, K., Verschuere, B., van den Bussche, E., & Crombez, G. (2010). The validity of the Psychopathic Personality Inventory-Revised in a community sample. *Assessment*, **17**, 334–346.
- Viding, E., & McCrory, E. J. (2012). Genetic and neurocognitive contributions to the development of psychopathy. *Development and Psychopathology*, **24**, 969–983.
- Vieira, J. B., Almeida, P. R., Ferreira-Santos, F., Barbosa, F., Marques-Teixeira, J., & Marsh, A. A. (2013). Distinct neural activation patterns underlie economic decisions in high and low psychopathy scorers. *Social Cognitive and Affective Neuroscience*. doi: 10.1093/scan/nst093
- Vuilleumier, P., Richardson, M. P., Armony, J. L., Driver, J., & Dolan, R. J. (2004). Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nature Neuroscience*, **7**, 1271–1278.
- Widiger, T. A., & Lynam, D. R. (1998). Psychopathy and the five-factor model of personality. In T. Millon, E. Simonsen, M. Birket-Smith, & R. D. Davis (Eds), *Psychopathy: Antisocial, Criminal, and Antisocial Behaviours* (171–187). New York: Guilford Press.
- Yang, Y., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta-analysis. *Psychiatry Research: Neuroimaging*, **174**, 81–88.