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Psychopathy: An overview

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PSYCHOPATHY is a disorder characterised by callousness, shallow affect, lack of guilt, antisocial behaviour and impulsivity. Since the early characterisation of the disorder, psychopathy has intrigued medical professionals and the general public alike. Understanding the mechanisms that underpin psychopathy is critical: these individuals commit a disproportionate amount of crime and are more likely to reoffend than criminals without psychopathic traits. Indeed, with the cost of antisocial behaviour in the UK an estimated £3.4 billion (www.police-foundation.org.uk), delineating the mechanisms of psychopathy could help to develop treatments and potentially reduce the burden of antisocial behaviour. This discussion paper will give a brief overview of research on the development of psychopathy, how psychopathy is measured and diagnosed, and studies examining the behavioural and neural profile associated with psychopathy.

WHILE THERE is no clinical diagnosis of psychopathy in childhood, there is abundant evidence that psychopathic traits and behaviours can be observed in children. In childhood, high levels of antisocial behaviour may be diagnosed as conduct disorder (DSM-5). Particular subsets of children with conduct disorder can also have elevated levels of psychopathic traits, which are termed callous-unemotional traits in research studies and 'limited prosocial emotions' in the new DSM-5 guidelines. Callous-unemotional traits in children can persist into adulthood (Lynam, Caspi, Moffitt et al., 2007) and are highly heritable (Viding, Blair, Moffitt & Plomin, 2005). In contrast, antisocial behaviour in children without callous-unemotional traits appears to be primarily driven by environmental influences (Viding et al., 2005).

In terms of the pathways through which psychopathy develops, researchers have proposed that individuals with psychopathy have an atypical experience of distress, such as fear or sadness (Blair, 2013; there may also be impairments in the processing of other emotions, but a discussion of this is beyond the scope of this article), underpinned by dysfunction in specific neural systems. Genetic and environmental factors influence the development of these neural systems. Over development, the reduced ability to experience emotions results in impaired associations between antisocial actions and outcomes of causing distress in other people (Bird & Viding, 2014; Blair, 2013). Reduced distress in an infant also results in fewer opportunities in the environment for learning which cues reliably signal distress in other people (Bird & Viding, 2014; Blair, 2013). Researchers have argued that it is the reciprocal interaction between atypical emotional reactivity and the resulting interactions with the environment that can lead to the development of psychopathy (Bird & Viding, 2014; Blair, 2013).

Measuring psychopathy

In forensic settings, the most widely used and validated instrument for assessing psychopathy is the Hare Psychopathy Checklist Revised (PCL-R; Hare, 2003). The PCL-R conceptualises psychopathy as consisting of two dimensions, Factor 1 and Factor 2. Factor 1 is characterised by affective and interpersonal features, including reduced empathy and guilt. Factor 2 is characterised by antisocial behaviour and impulsivity (Hare, 2003).

In the typical population, psychopathic traits can be reliably measured, with these traits existing on a continuum. Self-report measures suitable for non-forensic samples include the Self-Report Psychopathy Scale (Paulhus, Neumann & Hare, in press), the Psychopathic Personality Inventory (Lilienfeld & Widows, 2005) and the Levenson Self-Report Psychopathy Scale (Levenson, Kiehl & Fitzpatrick, 1995). However, there is considerable debate as to the best method of assessment of psychopathic traits in typical populations. Part of the disagreement is due to low correlations of some of these scales with the PCL-R or different conceptualisations of psychopathy all together (Lilienfeld & Fowler, 2006). This raises the question of whether the construct of psychopathy measured by the PCL-R in forensic samples is the same as that measured by self-report scales in community samples. Nevertheless, findings from studies in community samples often mirror those observed in forensic samples in both behavioural and neural profiles (e.g. Lilienfield & Fowler, 2006; and see discussion below), lending support to the claim that there is a common underlying construct.

Behavioural profile associated with psychopathy

In terms of the behavioural and neural profile associated with psychopathy, studies have examined forensic samples with high psychopathic traits, community samples with high psychopathic traits, and children with conduct disorder and varying levels of callous-unemotional traits. In the following two sections, studies from these different populations will be discussed, together with the assumption that they can all contribute to informing us about the profile of psychopathy.

Research on the behavioural and cognitive manifestations of psychopathy has mainly focused on emotion and reward processing impairments. For example, as discussed earlier, one of the most striking features of individuals with psychopathy is their reduced physiological response to others' emotions (Blair et al., 1997). Similarly, in community samples, high levels of psychopathic traits are related to weaker affective responses to fearful faces and happy stories (Seara-Cardoso, Neumann, Roiser et al., 2012) and a general impairment across empathic responses to other people regardless of valence (Lockwood, Bird, Bridge & Viding, 2013a). Intriguingly, this affective empathic deficit does not appear to extend to problems with understanding other people's thoughts – which distinguishes individuals with psychopathy/psychopathic traits from those with autism spectrum disorders/autistic traits (Lockwood et al., 2013a).

Reward processing impairments have been observed in children with conduct disorder during reinforcement-based decision making tasks, including the Iowa Gambling Task (Blair et al., 2001). In violent offenders with psychopathy, impairments have been observed in response reversal and passive avoidance learning (De Brito et al., 2013). Taken together, results from studies investigating emotion and reward processing suggest atypical emotion and reward processing is associated with psychopathic traits.

Neural profile associated with psychopathy

There has been a rapid expansion in neuroimaging studies investigating the brain basis of psychopathy in criminal psychopathy, community samples with high psychopathic traits and children with callous-unemotional traits. In line with the behavioural studies investigating psychopathy, neuroimaging research has mainly focused on emotion and reward processing impairments (although other areas of cognitive impairments have been investigated, they are beyond the scope of this overview). Broadly, atypical responses have been observed in the amygdala, insula, orbitofrontal cortex, anterior cingulate cortex and striatum – regions involved with emotion and reward processing in typical

populations. For example, relative to controls, those with psychopathy/high psychopathic traits show reduced amygdala response to emotional faces and social situations (Viding et al., 2012; Sebastian et al., 2012). When processing images of other people in pain, those with psychopathic traits have shown atypical neural responses in regions including the anterior insula and anterior cingulate cortex (Lockwood et al., 2013b; Decety, Skelly & Kiehl, 2013).

Using paradigms that index reward processing and decision-making, Pujara, Motzkin, Newman, Kiehl & Koenigs (2013) observed positive correlations between PCL-R scores and left ventral striatum activation to gains-losses. In community samples, Buckholtz et al. (2010) reported increased ventral striatum responses to monetary reward anticipation, and this response was associated with psychopathic traits. Finally, youths with psychopathic traits have been found to show reinforcement reward learning impairments in the caudate and ventromedial prefrontal cortex (White et al., 2013). This latter study was the first to use model-based fMRI in a sample of children with conduct problems. Model-based fMRI uses trial-by-trial behaviour to model parameters such as prediction error – the degree to which reinforcement is better or worse than expected. The model generated from the behaviour can then be applied to neuroimaging data to see which regions of the brain vary parametrically over time, with the size of the prediction error. The advantage of this approach is that it can tell us not just where in the brain there might be differences between those with and without psychopathy but also how different cognitive process are implemented (O'Doherty, Hampton & Kim, 2007).

Conclusions and future directions

Overall, the defining features of psychopathy include callousness/shallow affect and impulsive antisocial behaviour. The development of psychopathy is likely to result from genetic predispositions, dysfunction in specific neural systems, and the interplay between reduced emotional reactivity and resulting interactions with the environment. Atypical emotion and reward processing in forensic samples, community samples with high psychopathic traits, and children with conduct disorder and varying levels of callous-unemotional traits has been observed in both behavioural and neuroimaging studies.

Looking forward, the use of computational modelling techniques could help us to link behavioural and brain processes that might be disrupted in psychopathy, and perhaps get us closer to the specific mechanisms causing atypical processing. Intriguingly, recent evidence from neuroimaging suggests that reduced neural responses to others' pain in individuals with psychopathy can be changed dependent on the instructions given to participants, and in particular if participants are explicitly instructed to empathise (Meffert, Gazzola, den Boer, Bartels & Keysers, 2013). However, it remains to be seen whether effortfully activating the neural response to others' pain can foster empathy and empathic behaviour in individuals with high psychopathic traits. Nevertheless, factors that motivate effortful empathy in these individuals could be a key target for future research. In terms of understanding the development of psychopathy, researchers have argued that a longitudinal and genetically informed approach has the best chance of helping us elucidate how the developmental vulnerability to psychopathy unfolds. Ultimately, a key aim for the future will be to enable the findings from basic science to be translated into research informed approaches for clinical intervention in order to help individuals with psychopathy.

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