LETTER TO THE EDITOR

Spontaneous versus deliberate vicarious representations: different routes to empathy in psychopathy and autism

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Sir, We read the article recently published by Meffert et al. (2013) with great interest. The authors used functional MRI to investigate vicarious emotional representation in psychopathic offenders, scanning 18 psychopaths and 26 non-offending control subjects. Participants were either presented with video clips of short emotional interactions or experienced similar interactions themselves. Results showed that although psychopaths showed normal patterns of neural activation in response to experience, abnormal patterns of activation were observed during observation of others’ interactions in those regions known to be involved in vicarious emotional experience. However, when participants were instructed to ‘feel with the hands in the videos’ differences in activation between psychopaths and control subjects were significantly reduced in numerous regions of interest, including the medial and anterior left insula, left anterior cingulate cortex, and bilateral angular gyri (Meffert et al., 2013). The authors conclude that these results point toward reduced spontaneous but intact deliberate vicarious representations in psychopathy.

Psychopathy represents a severe disorder of personality, characterized by a callous lack of empathy, shallow affect, and a lack of remorse or guilt (Hare, 1991, 2003). Traditional theories have emphasized poor recognition of others’ distress cues, including emotional facial expressions, as central to empathy deficits observed in psychopathy (Blair et al., 2001, 2004; Montagne et al., 2005; Dadds et al., 2006; Dolan and Fullam, 2006; see also meta-analyses by Marsh and Blair, 2008; Wilson et al., 2011). These findings are extended through the more recent results of Meffert et al. (2013), which suggest that psychopaths may exhibit a particular breakdown in the neural processes that underlie the spontaneous vicarious experience of the emotions of others. Thus, Meffert et al. (2013) findings add significantly to current understanding of empathy deficits in psychopathy, distinguishing between spontaneous and deliberate vicarious experience.

We were drawn to write a letter in response to the publication of this article because of a related line of research on individuals with autism spectrum disorder (ASD). Similar to those with psychopathy, research suggests that individuals with ASD often exhibit difficulties in the processing of emotional expressions (Hall et al., 2007; Clark et al., 2008). However, similar to the findings by Meffert et al. (2013), studies have indicated that there may also be a dissociation between spontaneous and deliberate experience in this population. For example, adults with Asperger syndrome can understand mental states such as desires and beliefs (mentalizing) when explicitly prompted to do so, despite having impairments in this domain during spontaneous tasks (Senju et al., 2009). Additionally, two studies have shown impaired spontaneous but intact deliberate empathic motor mimicry in ASD (McIntosh et al., 2006; Oberman et al., 2009).

The mechanisms underlying impaired empathic responding in autism and psychopathy appear to differ. Emotion recognition deficits in psychopathy are thought to be the result of amygdala hypoactivity, and are believed to be at the core of psychopaths’ callous lack of empathy (Blair, 2007). Alternatively, similar findings in ASD may stem from more general impairments in the processing of facial stimuli, including abnormal functioning of the fusiform face area in the fusiform gyrus (Critchley et al., 2000; Pierce et al., 2001; Dalton et al., 2005), an essential structure for the cognitive representation of facial stimuli.

Relevant to the Meffert et al. (2013) findings, several recent studies have examined the role of personal familiarity on the
activation of neural mechanisms involved in both social and empathy-related processing in ASD. For example, Pierce and Redcay (2008) used functional MRI to examine regions of interest including the amygdala, fusiform gyrus, and anterior and posterior anterior cingulate cortices in children with ASD while they viewed pictures of the faces of (i) a familiar adult; (ii) a stranger adult; (iii) a familiar child; and (iv) a stranger child. While fusiform activity in response to the face of an adult stranger was strikingly absent in the children with ASD, the results also revealed normal fusiform activity while these children viewed both their mother’s face and the faces of other children (Pierce and Redcay, 2008). These results strongly suggest a role for a social-emotional connection in mediating the activation of the social perception system in those with ASD.

The importance of social-emotional relevance in autism was further demonstrated by Oberman et al. (2008). In this study we presented video clips of both familiar and non-familiar actors performing grasping actions to children with ASD and control children, while recording EEG activity (Oberman et al., 2008). Analysis of EEG mu suppression, a putative marker of ‘mirror neuron’ functioning, revealed that children with ASD exhibited relatively intact mu suppression during the observation of the actions of people familiar to them but impaired mu suppression during the observation of the same actions of unfamiliar people. We interpreted these data as evidence that mirror neuron system activation is also modulated by the social-emotional significance of the observed individual to the person with autism (Oberman et al., 2008).

Taken together, these findings suggest that individuals with ASD exhibit relatively more intact social and empathic activations when the observed individuals are socially and emotionally relevant to them, or when they are given explicit instructions to do so. On the other hand, the spontaneous activation of these same social and empathic mechanisms is commonly and distinctly reduced or absent during the observation of individuals unknown to them. This pattern of results contrasts with the findings Meffert et al. (2013) observed in individuals with psychopathy. In these individuals, social brain regions were active under both spontaneous and deliberate conditions, whereas the activation of neural mechanisms involved specifically in empathy were active only under deliberate instruction.

The finding that psychopaths fail to spontaneously activate the empathic system despite intact social-cognitive representations and activations may account for the callous lack of empathy characteristic of psychopaths’ antisocial behaviours, including a proclivity toward instrumental, premeditated, and goal-directed acts of aggression (Cornell et al., 1996; Woodworth and Porter, 2002). Alternatively, individuals with ASD seem to activate neither the social nor the empathic system (e.g. when observing individuals of little to no social or emotional relevance to them), or to activate both the social and the empathic systems (e.g. when observing individuals of clear social and emotional relevance to them). This pattern of findings may account for the fact that, although individuals with ASD commonly fail to respond to both the social and emotional overtures of others, there is very little to no evidence to suggest that these individuals actively exhibit elevated levels of proactive aggression. Indeed, research shows that children with ASD are actually more likely to report being victims of bullying (Little, 2002; Wainscot et al., 2008; Carter, 2009; Kloosterman et al., 2013), which may represent one form of proactive aggression (Camodeca et al., 2002). That is to suggest that the key distinction between those with ASD and those with psychopathy may be that the activation of social and empathic systems and associated mechanisms are more tightly linked in individuals with ASD than in individuals with psychopathy, and that the preservation of this link in those with ASD may explain their aloof and non-malicious behavioural phenotype relative to those with psychopathy.

These results may have implications for the development of interventions that aim to improve social communication in autism and empathic functioning in psychopathy. For example, interventions in autism may seek to increase the extent to which the individual identifies with, or assigns personal significance to, unfamiliar others. This may in turn lead to increases in both empathic responding and improved social communication. For example, Bölte et al. (2006) showed that the use of a computer-based program led to significant improvements in the recognition of basic emotional expressions in individuals with ASD that were linked to changes in neural activity in the superior parietal lobe and the medial occipital gyrus (Bolte et al., 2006). These results suggest that improvements in emotion recognition in autism are dependent upon improved processing of facial stimuli, rather than an improved ability to recognize emotional expressions per se.

Similar interventions have also been undertaken in an effort to address expression recognition deficits in psychopathy. For example, Dadds et al. (2006, 2008) found that children with callous unemotional traits showed improved performance when asked to specifically focus attention on the eyes of emotionally expressive faces. Furthermore, Dadds et al. (2012) have shown that a randomized controlled trial of emotion recognition training led to improvements in empathy and conduct problems among children with high callous-unemotional traits. However, these results do not necessarily indicate an increased capacity to ‘experience’ the emotions of others. The use of neurophysiological recordings in future research may eventually add weight to the argument that such interventions can improve emotional empathy. However, it should also be noted that such physiological changes may not be interpreted in the same way among psychopathic individuals compared with healthy control subjects. Thus, while the results of Dadds et al. (2012) and Meffert et al. (2013) represent critically valuable and important steps toward therapeutic intervention in psychopathy, the experiential or emotional correlates of such changes, as yet, remain unknown.

**References**


