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Reactive aggression and functional, not neural, specificity

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Harenski and Kiehl (2010) provide a thoughtful commentary on my recent review regarding psychopathy, frustration, reactive aggression, and ventromedial frontal cortex (vmPFC); Blair (2010). They make four main points regarding my paper and I will consider each in turn.

First, they consider whether individuals with psychopathy might show greater frustration because they *experience more frustration* than individuals without psychopathy. I also raised this as a possibility in the review. However, as they and I noted, there are no data to support this possibility. Such data may prove important however. As I noted, none of the existent models of psychopathy, in their current form, could predict an increased experience of frustration in individuals with psychopathy. Thus, data showing that there was an increased experience of frustration would suggest that current models of this disorder are, perhaps unsurprisingly, incomplete.

Second, they suggest that individuals with psychopathy might show greater frustration/reactive aggression because emotion regulation may be dysfunctional and note that vmPFC has been implicated in successful down regulation of emotional responses. However, the situation regarding emotional regulation, psychopathy, and vmPFC is complicated. There appear to be several ways in which emotional regulation might be achieved. One way, initially proposed by Ochsner and colleagues was that emotional regulation might occur via top-down attentional control (Ochsner, Bunge, Gross, & Gabrieli, 2002). If the individual increases attention to a task relevant stimulus via representational priming, then the representation of other stimuli, including emotional stimuli, will be weakened following representational competition (cf. Desimone & Duncan, 1995). Reductions in the representational strength of an emotional stimulus will decrease the response to that stimulus. However, this form of emotional regulation should not be disrupted in psychopathy. While attentional dysfunction has been implicated in psychopathy (e.g., Newman, Brinkley, Lorenz, Hiatt, & MacCoon, 2007), the current data suggest that any existent attentional dysfunction reflects an increased, and not a decreased, propensity for top-down attention (see Blair & Mitchell, 2009).

An additional way that emotional regulation might be achieved is via a suppressive impact of vmPFC on systems involved in the emotional response, such as the amygdala. This view, that vmPFC acts as the ‘brakes’ on emotional responding, is prevalent in the clinical neuroscience literature (cf. Phillips, Ladouceur, & Drevets, 2008). However, the data are relatively inconsistent. The Ochsner *et al*. (2002) finding cited by Harenski and Kiehl (2010) indicated reduced responding in both the amygdala and vmPFC as a function of emotional

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This article is an author response to Harenski and Kiehl’s (2010) commentary on Blair (2010).

regulation. Such data are less compatible with a regulation view. In contrast, they are compatible with the position, presented in my review, that the amygdala feeds forward reinforcement expectancies to vmPFC where it is represented as value information. According to this view, the reduction in the amygdala response following regulation would be expected to reduce vmPFC responding. Typically, ANOVA designs do not reveal the inverse relationship between amygdala and vmPFC that would be required by a 'brakes type' regulatory view though it should be noted that some functional connectivity analyses have found indications of such a relationship (e.g., Urry *et al*., 2006). One thing that is clear is that the relationship of vmPFC to the amygdala is not simply suppressive. Thus, animal work clearly demonstrates that lesions of vmPFC do not lead to disinhibited/increased amygdala responding as the 'brakes type' regulatory view predicts. Instead, lesions of vmPFC actually *decrease* amygdala responding (Schoenbaum & Roesch, 2005). These data are, of course, consistent with the suggestion of integrated amygdala–vmPFC functioning proposed in my review.

Critically, very recent data have allowed a rapprochement of the view that vmPFC represents reinforcement values (some of which are provided by the amygdala; see Blair, 2010) and the view that vmPFC plays a role in emotional regulation. A recent study provided evidence that exercising self-control involves the modulation of the value signal represented by vmPFC by dorsolateral prefrontal cortex (Hare, Camerer, & Rangel, 2009). As such, vmPFC would not act as the brakes on the amygdala. Instead, dorsolateral frontal cortex, by diminishing the value representation within vmPFC would, because of the integrated relationship of vmPFC and the amygdala, also diminish the emotional response within the amygdala. If these exciting data prove robust I would agree that with Harenski and Kiehl that we might indeed predict emotional regulatory problems in psychopathy; individuals with the disorder would be less able to regulate their emotional response because dorsolateral frontal cortex would have greater difficulty interfacing with a dysfunctional emotional value representation in vmPFC.

Third, Harenski and Kiehl make several claims based on the results of a recent meta-analysis by Guy, Edens, Anthony, and Douglas (2005). They argue that this meta-analysis indicates that 'incarcerated psychopaths do not show higher levels of reactive aggression (such as physical violence and related forms of institutional misconduct) than incarcerated non-psychopaths' (p. 403). However, it is important to note that Guy *et al*. (2005) actually report a *significant association between psychopathy level and aggression/misconduct*. Moreover, Guy *et al*. (2005) do not distinguish between reactive and instrumental aggressive episodes. Thus, we do not know the nature of any change in their aggressive behaviour. Harenski and Kiehl then argue that there is no reason to assume that 'stimulus-reinforcement and response reversal failures … occur less frequently when individuals are incarcerated (the latter may be even more frequent), it may be that supervision engages top-down mechanisms that control aggression' (p. 403). However, here I would argue the exact opposite. It is unclear what triggers supervision would provide to engage those neural systems implicated in the control of aggression or how this might occur. In contrast, it is very clear that structured environments increase habitual behaviour; activity occurs at specific times to specific cues. As such behaviour is less under the control of reinforcement-based decision making. As such one might expect a reduction in reactive aggression in individuals with psychopathy in an institutional setting.

Fourth, they take exception to the claim following the recent finding that youth with psychopathic traits show appropriate responsiveness within dorsal anterior cingulate cortex (dACC) to punished reversal errors (Finger *et al*., 2008). I argued that this finding contradicts the assertion of the paralimbic hypothesis of psychopathy (Kiehl, 2006) that the entire cingulate is compromised in the disorder. They make two points. Their first point is

that Kiehl's position only stands for individuals who score above 30 on the PCL-R (most of the clinical patients in Finger *et al*. (2008) did not score above 30 on the youth equivalent of the PCL-R, the PCL-YV). Issues of group classification are important. However, it is unfortunate for their argument that they used as evidence for their position on psychopathy one of their own studies that was conducted on healthy undergraduates differentiated by their score on a self-report measure of psychopathy (Harenski, Kim, & Kiehl, 2009). This would suggest that Kiehl's position stands for healthy participants differentiated by self-report and thus might also apply to clinical patients who score above 20 rather than above 30 on the PCL.

Their second point is that 'for psychopaths to show similar levels of activity in a brain region compared to nonpsychopaths during a given task does not convincingly demonstrate a "lack of impairment" in this region' (p. 404). I agree with them. Indeed, I have argued elsewhere that it is very important not to assume dysfunction in a region in individuals with psychopathy simply because of reduced activity relative to controls; this may reflect a lack of input from other regions that are dysfunctional in psychopathy. However, it is critical to remember that the Finger *et al*. (2008) exist within a neuropsychological literature. The Finger *et al*. (2008) results indicated appropriate recruitment of dACC to the response conflict initiated by a punished reversal error. Previous neuropsychological work has shown that the mediation of response conflict in the context of Stroop paradigms is intact (and may even be superior) in adults with psychopathy (Blair *et al*., 2006; Hiatt, Schmitt, & Newman, 2004). Together, these results suggest that the mediation of response conflict by dACC appears intact in psychopathy. As such, these results suggest that it is more accurate to claim that while some functions of cingulate cortex may prove to be dysfunctional in psychopathy, the entire cingulate cortex is not compromised in this disorder.

In conclusion, I agree with Harenski and Kiehl (2010) that it is possible that individuals with psychopathy may also show emotional dys-regulation. However, it is perhaps important that we can draw this conclusion on the basis of known functional deficits mediated by a specific neural system (the representation of reinforcement information by vmPFC) rather than simply because the functioning of a specific neural system (such as vmPFC) is thought to be compromised. It is as unlikely that all functions of vmPFC are compromised in psychopathy as it is that all functions of cingulate cortex are compromised in this disorder.

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