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Reactive aggression in psychopathy and the role of frustration: Susceptibility, experience, and control

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This article is a commentary on ‘Psychopathy, frustration, and reactive aggression:

The role of ventromedial prefrontal cortex’ (Blair, 2010).

Blair proposes that psychopaths show high levels of reactive aggression due to their increased susceptibility for experiencing frustration. This increased susceptibility arises from cognitive impairments in stimulus-reinforcement learning and response reversal, which lead to repetitive unsuccessful goal-directed behaviours that are frustrating to the psychopath. These two forms of processing, particularly response reversal, are argued to be dependent on the ventromedial prefrontal cortex, a brain region which is known to be dysfunctional in psychopathy.

Blair and others have demonstrated psychopathy-related impairments in stimulus-reinforcement learning (Blair *et al.*, 2004; Blair, Leonard, Morton, & Blair, 2006; Newman & Kosson, 1986) and response reversal (Blair, Colledge, & Mitchell, 2001; Budhani & Blair, 2005; Budhani *et al.*, 2006; Mitchell, Colledge, Leonard, & Blair, 2002). In a recent study, they also found that children with psychopathic tendencies showed abnormal ventromedial prefrontal cortex activity during a response reversal task compared to children without psychopathic tendencies (Finger *et al.*, 2008). We concur with Blair that these impairments may indeed increase the frequency of frustrating experiences, leading to a greater likelihood of reactive aggression. We also concur that these impairments may result from aberrant function of the ventromedial prefrontal cortex, though more research demonstrating a direct link between ventromedial prefrontal impairment and deficits in stimulus-reinforcement and response reversal learning in psychopathy is needed. However, we believe it is unlikely that increased susceptibility for experiencing frustration is the sole contributing factor to reactive aggression in psychopathy. There are at least two critical processes that occur between a frustration-eliciting situation and the subsequent behaviour of reactive aggression, processes which greatly impact the probability that reactive aggression will occur. We consider each of these in turn.

First, consider a scenario in which a psychopath and a non-psychopath experience the same frustration-eliciting situation. Does the psychopath experience more frustration than the non-psychopath in response to the same situation? Blair stated that no influential models of psychopathy would predict increased responses to frustrating events. It may be more accurate to state that few models of psychopathy have addressed this issue. Theoretical perspectives on psychopathy have typically focused on emotional under reactivity that characterizes the disorder, particularly with respect to fear (Fowles, 1988; Lykken, 1995; Patrick, Cuthbert, & Lang, 1994). Other perspectives have focused on cognitive deficits (Newman, 1998), or integrated cognitive and affective deficits (Blair, 2005), but none to our knowledge have considered whether psychopaths show increased levels of any type of emotion. Thus, research is needed in which psychopaths and non-psychopaths are placed in equally frustrating situations (e.g., a task in which success or reward is equally unlikely in both groups), and behavioural, physiological, and neural responses are recorded in each group, to assess whether psychopaths report higher levels of frustration, show increased physiological responses and/or brain activity during frustrating aspects of the task than do non-psychopaths. As Blair notes, recent work has suggested that frustrating events increase activity in the ventromedial prefrontal cortex (Abler, Walter, & Erk, 2005), the same region that has been implicated as dysfunctional in psychopathy and that is proposed to underlie deficits in stimulus-reinforcement and response reversal learning. If psychopaths do show evidence of increased frustration relative to non-psychopaths, this result would represent another psychopathy-related vulnerability that increases the probability of reactive aggression.

Second, an important question concerns the psychopath's ability to control frustration responses once they are elicited. One of the 20 traits that characterize psychopaths, as defined by the Hare Psychopathy Checklist (Hare, 2003), is 'poor behavioural controls'. This trait refers to an inability to control anger and frustration, leading to aggression. Thus, regardless of whether psychopaths experience frustration more often or more intensely relative to non-psychopaths, the ability (or willingness) to down regulate the frustration response that causes reactive aggression might be dysfunctional. The relevance of emotion regulation to psychiatric disorders such as depression and anxiety has been widely discussed (Amstadter, 2008; Gross & Munoz, 1995; Nolen-Hoeksema & Corte, 2004), but it has not received much attention in psychopathy. Typically psychopaths show reduced emotional responses, and thus may require less emotion regulation skills than non-psychopaths (see also Harenski, Kim, & Hamann, 2009). However, if psychopaths experience frustration at normal levels, emotion regulation could be a critical factor in determining whether reactive aggression occurs. If psychopaths experience *higher* levels of frustration than non-psychopaths, it should be even more difficult to down regulate the response. It is also worth noting that the ventromedial prefrontal cortex has been implicated in successful down-regulation of emotional responses, such that activity in this region decreases during voluntary down-regulation of emotional responses (Ochsner, Bunge, Gross, & Gabrieli, 2002). Psychopathy-related dysfunction in this region could prevent successful down-regulation of emotional response such as frustration. Thus, emotion regulation represents another process that may be impaired in psychopathy, which would increase the likelihood of reactive aggression.

Interestingly, incarcerated psychopaths do not show higher levels of reactive aggression (such as physical violence and related forms of institutional misconduct) than incarcerated non-psychopaths (Guy, Edens, Anthony, & Douglas, 2005). However, research and clinical anecdotes clearly indicate that psychopaths act differently, namely with more reactive aggression, in the community than when incarcerated. The relatively 'normal' behaviour of psychopaths in prisons suggests that supervision is an effective management technique for reactive aggression in psychopathy. Since there is no reason to assume that stimulus-reinforcement and response reversal failures, or increased frustration, 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; mechanisms which are supported by the prefrontal cortex, particularly the anterior cingulate and orbitofrontal cortex (Siever, 2008). Unfortunately, psychopaths are generally not supervised when in the community. One could envision an experiment in which psychopaths released from prison into the community were required to wear monitoring devices and made aware that their behaviours could be tracked at any time. Would psychopaths then be less likely to display reactive aggression in frustrating situations?

An alternative to the frustration hypothesis of reactive aggression, suggested by an anonymous reviewer (cited in Blair), was that psychopaths might be hypersensitive to social but not physical threat. Consistent with this suggestion, Blair stated that Emery *et al.* (2001) found that infant monkeys with amygdala lesions show enhanced fear of unfamiliar conspecifics, suggesting that the neural systems for social threat and physical threat are different (since amygdala lesions decrease fear of physical threats). However, although the lesioned monkeys in Emery *et al.* did show more fear grimaces to unfamiliar monkeys than did control monkeys, they also made significantly more contact and affiliative gestures than those without amygdala lesions, suggesting they were not inhibited by any possible threat. Certain characteristics of psychopathy may also confer a lack of inhibition and a heightened propensity for approach behaviour. Psychopaths are unusually socially extraverted (Levenson, Kiehl, & Fitzpatrick, 1995) and they are insensitive to distress cues such as fearful facial expressions or vocalizations (Blair, 1999; Blair *et al.*, 2002, 2004). Thus, psychopaths may be less likely to inhibit an approach, whether affiliative or aggressive, towards another individual even when the individual exhibits social cues meant to discourage approach. It must be noted, however, that other's facial expressions are hardly the only, or most representative, example of social threat. Social threats come in many forms, including threats to another's social status, to interpersonal relationships, to occupational success, etc. The psychopath may display reactive aggression towards an individual who threatens them in such a manner, though this has not been studied to date.

Our commentary thus far has focused on the ventromedial prefrontal cortex, in which Blair has proposed that dysfunction contributes to impaired response reversal in psychopathy, increasing the probability of reactive aggression. However, Blair also highlighted other regions, such as the dorsomedial prefrontal cortex including the anterior cingulate, which have been activated during response reversal tasks (Budhani, Marsh, Pine, & Blair, 2007; Remijnse, Nielen, Uylings, & Veltman, 2005). In a recent study (Finger *et al.*, 2008), children with and without psychopathic tendencies both showed increased dorsal cingulate

activity during a response reversal task. This result led Blair to conclude that psychopaths show normal function in at least one part of the cingulate, which contradicts the assertion of the paralimbic hypothesis of psychopathy (Kiehl, 2006) that the entire cingulate is compromised in the disorder. There are several issues with Blair's assertion that deserve consideration.

The paralimbic hypothesis, as discussed in Kiehl (2006), pertains to adult individuals who meet clinical criteria for psychopathy, i.e., scoring 30 or above on the Hare Psychopathy Checklist-Revised. With respect to Blair's assertion that Finger *et al.*'s data contradicts the paralimbic hypothesis, we raise the following three points. First, the Psychopathy Checklist: Youth Version (PCL-YV) cut-off score used in Finger *et al.* (20 out of 40) was far below the recommended cut-off for youth (or adult) psychopathy (a score of 30 out of 40 is needed; see Forth, Kosson, & Hare, 2003). Thus, it is unlikely that there were any youth in Finger *et al.* who met criteria for psychopathy. Second, it is currently an open question whether youth with high scores on the PCL-YV will end up scoring over 30 on the Hare Psychopathy Checklist-Revised (adult version). We further note that it is exceedingly unlikely that youth with a score of 20 on the PCL-YV will climb to a score of over 30 on the Psychopathy Checklist-Revised in adulthood. Lastly, the fact that the cingulate is activated during response reversal learning does not provide strong evidence that it contributes to effective response reversal. It is often the case in functional imaging studies that one or more brain regions are activated during, but are not necessary for, task performance (see Kiehl, Stevens, Laurens, Pearlson, & Liddle, 2005, for more). Thus, for psychopaths to show similar levels of activity in a brain region compared to non-psychopaths during a given task does not convincingly demonstrate a 'lack of impairment' in this region. In order to conclude this it would be necessary to show that: (1) lesions to the cingulate impair some or all aspects of response reversal and (2) psychopaths do not show similar impairments. Thus, we disagree with Blair that the results from Finger *et al.* indicate normal function of the anterior cingulate in psychopathy. Rather, the extant literature still supports the idea that psychopathy (at clinical diagnostic levels) is associated with abnormalities in anterior cingulate (see Kiehl, 2006, for review).

To summarize, in our view there are multiple potential pathways to reactive aggression in psychopathy: (1) an increased probability of encountering frustrating situations, as proposed by Blair, (2) a heightened frustration response to frustration-eliciting situations, and (3) a reduced ability to down regulate frustration responses once they are elicited. Each of these processes involves the ventromedial prefrontal cortex, which has been shown to be dysfunctional in psychopathy. More research is needed to determine the extent to which each of these factors plays a role in reactive aggression in psychopathy, as well as the neural dysfunction underlying the deficits. These issues have important treatment implications, as they can help to identify which areas of function are most likely to benefit.

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