Disrupted Prefrontal Regulation of Striatal Subjective Value Signals in Psychopathy

Highlights

- Ventral striatal subjective value signals are amplified in incarcerated psychopaths
- Medial cortico-striatal intrinsic connectivity is weak in psychopathic individuals
- Cortico-striatal regulation of striatal activation is disrupted in psychopathy
- Diminished cortico-striatal regulation is associated with more criminal convictions

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In Brief

Psychopaths are notorious for their criminal behavior and poor self-control, but underlying neural mechanisms remain unresolved. Using fMRI in incarcerated offenders, Hosking et al. show that regulatory cortico-striatal connectivity is weakened in psychopathy, driving heightened striatal value encoding during decision making.
Disrupted Prefrontal Regulation of Striatal Subjective Value Signals in Psychopathy

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Article

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SUMMARY

Psychopathy is a personality disorder with strong links to criminal behavior. While research on psychopathy has focused largely on socio-affective dysfunction, recent data suggest that aberrant decision making may also play an important role. Yet, the circuit-level mechanisms underlying maladaptive decision making in psychopathy remain unclear. Here, we used a multi-modality functional imaging approach to identify these mechanisms in a population of adult male incarcerated offenders. Psychopathy was associated with stronger subjective value-related activity within the nucleus accumbens (NAcc) during inter-temporal choice and with weaker intrinsic functional connectivity between NAcc and ventromedial prefrontal cortex (vmPFC). NAcc-vmPFC connectivity strength was negatively correlated with NAcc subjective value-related activity; however, this putative regulatory pattern was abolished as psychopathy severity increased. Finally, weaker cortico-striatal regulation predicted more frequent criminal convictions. These data suggest that cortico-striatal circuit dysregulation drives maladaptive decision making in psychopathy, supporting the notion that reward system dysfunction comprises an important neurobiological risk factor.

INTRODUCTION

Crime is estimated to cost the United States more than a trillion dollars a year (Anderson, 1999). Even apart from its traumatizing effects on victims, the enormous economic burden associated with criminal behavior renders it a critical target for scientific investigation. One particularly robust predictor of criminal behavior and recidivism is psychopathy. Psychopathy is a personality disorder comprising both interpersonal-affective and impulsive-antisocial symptoms; these include superficial charm and egocentric grandiosity, callousness and diminished empathy, disinhibition, heightened sensation seeking, and persistent antisocial behavior (Viding et al., 2014). Psychopathy is notoriously difficult to treat (Brazil et al., 2016), and intervention efforts are hampered by our limited understanding of the cognitive and neurobiological underpinnings of the disorder.

Neuroscientific work to date has largely focused on the interpersonal-affective dimension of psychopathy. These studies have found relatively consistent evidence for amygdala and ventromedial prefrontal cortex (vmPFC) dysfunction, which is thought to underlie aberrant fear learning, moral decision making, and empathic responsiveness in the disorder (Blair, 2008; Koenigs et al., 2011; Viding et al., 2014). Morphological findings indicate decreased amygdala and vmPFC gray matter volume, and lower vmPFC cortical thickness, in psychopathy (Ermer et al., 2012; Yang and Raine, 2009). Likewise, psychopaths show reduced recruitment of amygdala and vmPFC during fear conditioning and moral decision making (Birbaumer et al., 2005; Glenn et al., 2009; Harenski et al., 2010), blunted amygdala responsiveness during affective perspective taking (Decety et al., 2013a), and weaker vmPFC engagement in response to empathogenic (Decety et al., 2013b) and facial emotion stimuli (Carré et al., 2013; Viding et al., 2014). Reduced functional and structural connectivity between amygdala and vmPFC has also been reported in psychopathy (Motzkin et al., 2011). Notably, there is some evidence that the observed relationships between psychopathy, task-related brain activity, and vmPFC-amygdala connectivity are driven by the interpersonal-affective features of the disorder (Carré et al., 2013; Decety et al., 2014; Hyde et al., 2014). This observation accords well with functional and structural brain imaging work in youth and adolescents with callous-unemotional traits (Blair, 2013), which are considered by some to be a developmental precursor of the interpersonal-affective symptoms in adult psychopathy (Frick and Viding, 2009). On the whole, these findings make a compelling case that social and affective deficits in psychopathy arise from cortico-limbic circuit dysfunction (Blair, 2013; Viding et al., 2014).

While the neurobiological mechanisms underlying interpersonal-affective symptoms in psychopathy are coming increasingly
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