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Putting the brakes on antisocial behavior: Secondary psychopathy and post-error adjustments in reaction time

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Abstract

Psychopathy is both a clinical disorder and an individual difference dimension that is strongly predictive of antisocial behavior. The present work focuses on a dimensional understanding of psychopathy and recognizes the distinction between primary and secondary psychopathy. Following from the conceptualization of secondary psychopathy as a type of impulsivity which renders an individual less capable of learning from prior mistakes, the authors hypothesized that individuals high in secondary psychopathy would be less likely to adjust behavioral performance following errors. Two studies, involving a total of 104 undergraduate volunteers, were conducted. As hypothesized, individuals high in secondary (but not primary) psychopathy exhibited a reduced tendency to slow behavioral performance following errors. The findings highlight the manner in which an error self-regulation model can shed light on the processing basis of secondary psychopathy and its correlates.

Keywords: Psychopathy; Self-regulation; Cognitive control; Errors; Impulsivity

1. Introduction

When Cleckley (1941) originally described the construct of psychopathy, it is unlikely that he could have appreciated the degree to which this individual difference variable would capture the

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public imagination. Countless movies (e.g., Psycho; Silence of the Lambs) and television programs (e.g., Law and Order) have depicted criminals who possess many of the traits that Cleckley described as characteristic of the prototypical psychopath. Such characters exhibit pervasive antisocial tendencies even in the face of extreme societal censure. Moreover, they are frequently depicted as cold-hearted and impulsive, often acting aggressively on the basis of whims or fleeting desires.

Although these popular depictions as well as the preponderance of psychological research has focused on criminal psychopaths, Cleckley (1941) viewed psychopathy as a continuous dimension of personality that varies from extreme clinical cases to more modest sub-clinical cases. Empirical support for this dimensional view is relatively recent. Studies have shown that psychopathy has dimensional characteristics that can be reliably measured among normal populations (e.g., Levenson, Kiehl, & Fitzpatrick, 1995; Marcus, John, & Edens, 2004). Furthermore, such research has shown that sub-clinical psychopathy measures are predictive of individual variation in antisocial behavior (Levenson et al., 1995). Finally, authors have converged on the idea that many of the mechanisms proposed to underlie criminal psychopathy also appear to underlie sub-clinical variations in this construct (Benning, Patrick, & Iacono, 2005; Vanman, Meija, Dawson, Schell, & Raine, 2003). In the current investigation, we sought to further contribute to this dimensional view of psychopathy.

Psychopathy is thought to encompass two distinct but related dysfunctions (Frick, 1995; Harpur, Hare, & Hakstian, 1988). The first dysfunction, commonly termed primary psychopathy, refers to a type of emotional callousness involving a lack of empathy, guilt, and remorse. Individuals high in primary psychopathy are thought to engage in antisocial actions precisely because they lack the emotional reactions (e.g., guilt) that stop most individuals from engaging in behavior harmful toward others (Blair, Mitchell, & Blair, 2005). The second dysfunction, commonly termed secondary psychopathy, has been characterized less in terms of emotional deficits and more in terms of impulsive tendencies towards antisocial behavior (e.g., Fowles & Dindo, 2006). From this view, secondary psychopathy is linked to a lack of behavioral restraint and an inability to modulate existing behavioral patterns (Fowles & Dindo, 2006; Morgan & Lilienfeld, 2000).

Previous research has primarily focused on the emotional deficit thought to underlie primary psychopathy (Benning et al., 2005; Vanman et al., 2003). In the present research, we instead focus on the impulsive deficit thought to be characteristic of individual differences in secondary psychopathy. Researchers have often suggested that cognitive control deficits may underlie this dimension (Colledge & Blair, 2001; Morgan & Lilienfeld, 2000). Considering the high recidivism rates associated with secondary psychopathy (Walters, 2003), it is likely that such deficits should involve an inability to modulate behavior that has resulted in negative outcomes in the past (e.g., Newman & Lorenz, 2003). Based on this line of reasoning, we hypothesized that secondary psychopathy might be systematically linked to deficits in the modulation of error-prone behavior.

A great deal of cognitive and neurocognitive research has contributed to a systematic understanding of the manner in which individuals recognize and reduce their tendencies toward error (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Such research indicates that the anterior cingulate cortex (ACC) can detect performance-related errors quite quickly (i.e., within 100 ms of the commission of the error; Gehring, Goss, Coles, Meyer, & Donchin, 1993). Moreover, it is apparent that the detection of an error triggers efforts to avoid errors in the future (Botvinick et al., 2001; Kerns et al., 2004).