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Emotion processing in Psychopathy Checklist — assessed psychopathy: A review of the literature

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HIGHLIGHTS

- Most studies associate psychopathy with abnormal responsiveness to emotion cues.
- Verbal tasks produce deficits more consistently than nonverbal tasks.
- Emotional stimulus complexity is associated with greater deficits.
- Overall findings are not clearly consistent with any single theoretical perspective.
- Future research should examine moderators of emotion processing in psychopathy.

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ABSTRACT

Deficient emotional experience is recognized as one of the central features of psychopathy and an impressive body of empirical findings regarding emotion processing in psychopathy has amassed over the past several decades, resulting in two broad theoretical perspectives. The *general emotional deficit perspective* postulates a globally reduced capacity for emotional experience and processing across the emotional spectrum. In contrast, according to the *specific emotional deficit perspective*, psychopathy is associated with abnormal experience of only specific types of emotion; several distinct hypotheses have been proposed under this latter perspective. We systematically and critically review findings from peer-reviewed research of emotion processing in psychopathy in relation to the two theoretical perspectives. In general, findings suggest that, compared to controls, psychopaths exhibit behavioral, psychophysiologic, and regional brain activation anomalies when processing emotion, but their ratings of self-arousal and stimulus valence/intensity do not differ from controls. However, when behavioral findings are examined separately by emotion type, the overall pattern of findings is not clearly consistent with any of the dominant theoretical perspectives of emotion processing in psychopathy. We summarize the current state of the field, including consistencies and inconsistencies in the literature, offer alternative explanations for the findings, and outline directions for future research.

Contents

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1. Introduction

Psychopathy is a personality disorder characterized by impulsive antisocial behavior, lack of emotional depth, callous treatment of others, and poor judgment. It is both a dangerous disorder and a costly one. Psychopathic inmates are charged with more crimes, more violent crimes, and a greater diversity of crimes than other inmates (Hare, 2003; Kosson, Smith, & Newman, 1990). They are more likely than other inmates to fail treatment (Ogloff & Wong, 1990) and conditional release programs (Hart, Kropp, & Hare, 1988; Porter, Birt, & Boer, 2001), and psychopathy is a strong predictor of criminal recidivism (Laurell & Daderman, 2005) and violence (Hare & Neumann, 2009; Walsh, Swogger, & Kosson, 2009).

Although the costs of psychopathy are increasingly recognized, and the construct increasingly used in forensic contexts (T. Walsh & Walsh, 2006), the mechanisms underlying the disorder are not well understood. For over 50 years, the field of psychopathy research has been dominated by clinical descriptions and theories that emphasize emotional deficits as core features of this disorder. Moreover, a growing body of empirical evidence has contributed to the development of several credible hypotheses that explain the mechanisms underlying psychopathy in terms of anomalous emotional processing. Whereas there appears to be substantial evidence consistent with each of these hypotheses, there are no recent systematic reviews of the evidence bearing on them. The purpose of this paper is to address this gap in the literature.

Contemporary hypotheses regarding emotional processing in psychopathy can be bifurcated into two primary perspectives regarding the level of specificity of observed affective deficits. According to the *general emotional deficit perspective*, psychopaths are characterized by a blunted capacity for experiencing emotion in general, which interferes with understanding the emotional significance of events and the meaning of their actions (Cleckley, 1941). As such, they are basically rational or cognitively intact, but, because they lack emotional depth, their appraisals of situations are deficient in a way that renders them unable to anticipate the emotional consequences of actions for themselves or others. According to this viewpoint, psychopaths' cognitive deficits are secondary to the pathology of the emotion processing system.[1] Alternatively, the *specific emotional deficit perspective* holds that psychopaths are characterized by an incapacity for only specific types of emotional experiences. Several distinct variants of this perspective have received substantial research attention. According to the *low fear hypothesis* (e.g. Lykken, 1957, 1995), psychopaths experience some emotions but have a reduced capacity or heightened threshold for activating fear/defense/withdrawal systems. Similarly, according to the *violence inhibition mechanism hypothesis* (VIM; Blair, 1995), later elaborated into the *integrated emotions* theory (Blair, 2005), psychopaths experience some emotions but are impaired in their understanding of interpersonal distress cues associated with sadness and fear. Consequently, psychopaths fail to recognize cues that would otherwise lead them to inhibit aggressive behavior by activating the neural networks involved in empathic processing. According to both these hypotheses, amygdala dysfunction is at the core of psychopaths' emotional deficits, resulting in impaired processing of certain kinds of fear-related emotional information (i.e., punishment cues

[1] It is noteworthy that several theorists have maintained that deficits in information processing represent a primary pathological process in psychopathy. Indeed, studies have shown that psychopathy is associated with significant situation-specific deficits in attention and behavioral inhibition (Hiatt & Newman, 2006). Newman has argued that an impaired ability to modulate dominant responses can explain psychopathic offenders' apparent emotion deficits (e.g. Newman et al., 2010). Other authors have argued for cognitive deficits when situations induce differential activation of left hemisphere-based systems (e.g., Kosson, Miller, Byrnes, & Leveroni, 2007) or have integrated hypotheses for cognitive deficits with hypotheses for affective deficits (e.g. Blair, Mitchell, & Blair, 2005; Kiehl, 2006).