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Memory for emotional events in violent offenders with antisocial personality disorder

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Abstract

Eight-eight violent offenders meeting the criteria for DSM-IV antisocial personality disorder (ASPD) were assessed using the Psychopathy Checklist-Screening Version (PCL-SV) and compared with 20 healthy controls on an emotional memory task. All participants showed enhanced memory for the emotional phase of the task. On the free and cued recall components of the task high psychopathy scorers showed most impairment on the emotional phase compared with healthy controls. Analyses of psychopathy dimensions indicated no evidence of a dimensional relationship with emotional memory, but subjects categorised as scoring on the extremes of the psychopathy scale particularly those with high emotional detachment (Factor 1) and antisocial behaviour (Factor 2) had impairments in free recall an effect that was attenuated when subjects received cues to assist recall. The results suggest that emotional memory impairments in antisocial populations may be related to a variety of emotional and attentional processes linked with the prefrontal-limbic neural circuitry.

Keywords: Emotional memory; Violence; Psychopathy; Antisocial personality

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1. Introduction

The antisocial personality disorders (antisocial personality disorder and psychopathy) are a group of overlapping disorders of personality that are associated with significant intra and interpersonal dysfunction. Antisocial Personality Disorder (ASPD) as defined in DSM-IV is more common than the dimensionally defined higher order construct of psychopathy as described by Cleckley (1976) and more recently by Hare (1991). Rates of these disorders are significantly higher in prison and forensic samples than in the general population (Hare, 1991). Half of prison inmates meet DSM-IV criteria for ASPD in the UK (Singleton, Meltzer, & Gatward, 1998). Coid (1992) reported a prevalence rate for psychopathy of 38% in maximum-security psychiatric hospital patients. Psychopathy assessed using Hare's (1991) criteria is estimated to occur in less than 20% of prisoners with a diagnosis of ASPD (Hare, 1998).

Although all these disorders are known to be a major cause of social distress—disruption to families, criminality and violence (Robins, 1990), very little is known about causation. Social factors are associated with antisocial personality disorders (Robins, 1990). However, they also occur in those with apparently normal family backgrounds and there is evidence of genetic liability (McGuffin & Thapar, 1992) suggesting a neurobiological basis for these disorders.

There are a number of theories relating to the development of antisocial behaviour, the most prominent of which are the punishment/low fear theories (see Lykken, 1995); the Response Modulation deficit hypothesis (Patterson & Newman, 1993; see Newman, 1998, for a review) and more recently the "Violence Inhibition Mechanism"(VIM) deficit proposed by Blair (1995) in which psychopathic behaviours and low empathy are perceived to be related to the failure of basic emotions (e.g. fear) to result in autonomic arousal and the inhibition of ongoing behaviour.

Many of these theories focus on the behavioural components of antisocial behaviour and few focus on the interpersonal (low empathy, callousness) components of the disorder. Blair and Frith (2000), however, suggest that the amygdala may be a core component of the neural circuit that mediates the VIM and they have proposed that early amygdala dysfunction may result in the development of core psychopathic (affective–interpersonal) traits.

Amygdala lesions in humans reduce the ability to acquire conditioned autonomic responses (Bechara et al., 1995) and impair the capacity to recall emotional material (Cahill, Babinsky, Markowitsch, & McGaugh, 1995). Functional imaging studies also confirm the notion that the amygdala is activated by affectively loaded visual stimuli (Blair, Morris, Frith, Perrett, & Dolan, 1999; Breiter et al., 1996; Morris et al., 1996; Phillips et al., 1997).

There is now fairly extensive empirical evidence that psychopaths have attenuated electrodermal responses in anticipation of aversive stimuli (see Hare, 1998, for review) and attenuated startle responses to unpleasant slides (Lang, Bradley, & Cuthbert, 1990; Patrick, Bradley, & Lang, 1993; Levenston, Patrick, Bradley, & Lang, 2000). The latter findings are consistent with a hypothesised deficit in neural circuits involved in the processing of negative affect e.g. amygdala (Davis, 1989).

To date most of the research literature on emotional information processing in antisocial samples has focussed on physiological responses to emotional visual stimuli and there has only been one study looking at memory for details of emotional events in psychopathic and