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Review

Deficits in facial affect recognition among antisocial populations: A meta-analysis

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Abstract

Individuals with disorders marked by antisocial behavior frequently show deficits in recognizing displays of facial affect. Antisociality may be associated with specific deficits in identifying fearful expressions, which would implicate dysfunction in neural structures that subserve fearful expression processing. A meta-analysis of 20 studies was conducted to assess: (a) if antisocial populations show any consistent deficits in recognizing six emotional expressions; (b) beyond any generalized impairment, whether specific fear recognition deficits are apparent; and (c) if deficits in fear recognition are a function of task difficulty. Results show a robust link between antisocial behavior and specific deficits in recognizing fearful expressions. This impairment cannot be attributed solely to task difficulty. These results suggest dysfunction among antisocial individuals in specified neural substrates, namely the amygdala, involved in processing fearful facial affect.

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Keywords: Facial affect; Fearful expression; Antisocial; Amygdala; Psychopathy; Emotion; Meta-analysis

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1. Introduction

Processing facial affect is crucial for socialization and normal social interaction (Corden et al., 2006; Fridlund, 1991). Aggression and other maladaptive antisocial behaviors may result from failure to be appropriately guided by the social cues of others (Blair, 2003a; Montagne et al., 2005; Walker and Leister, 1994). Some have suggested that distress-related cues, particularly fearful expressions, play an important role in inhibiting antisocial behavior (Blair, 2001; Nichols, 2001; Price et al., 2004). Accordingly, many studies find impairments in processing distress-related cues among antisocial populations. However, not all studies find this impairment. It is unclear whether these inconsistencies result from disparate methodologies, sample populations, or analytic techniques, or, alternately, from the absence of a strong relationship between fearful affect processing and antisociality. Better understanding of facial affect recognition deficits associated with antisociality would permit more precise hypotheses to be formulated regarding neurocognitive correlates of antisocial behavior. We thus conducted this meta-analysis to assess associations between antisociality and facial affect recognition deficits. We hypothesized that, beyond any general facial affect recognition deficits, antisocial individuals show specific deficits in processing fearful expressions. We also hypothesized that such deficits are not solely attributable to task difficulty.

Emotional facial expressions play an important role in modulating interpersonal behavior. Given this, extensive research has assessed the relationship between facial affect recognition and psychiatric disorders characterized by interpersonal deficits. Generalized impairments in processing facial affect have been found in antisocial populations (Kropp and Haynes, 1987; Woodbury-Smith et al., 2005; Zabel, 1979). But such impairments have also been found in disorders like autism, schizophrenia and social anxiety disorder (Gross, 2004; Singh et al., 1998; Tremeau, 2006; Easter et al., 2005). Myriad factors, including general intelligence, age, attention, verbal ability, and task-specific motivation can be associated with reductions in facial affect recognition scores (Herba and Phillips, 2004; Moore, 2001). It is thus unsurprising that many clinical populations suffer facial affect recognition deficits. However, it means that little clinically or neuropsychologically specific information regarding a particular population's impairments can be extracted from general facial affect processing deficits.

More informative would be evidence for an association between antisocial behavior and a deficit in recognizing one or more specific expressions. Likely candidates include distress cues like fear and sadness because these expressions may inhibit or avert inappropriate behavior like aggression (Blair, 2003b; Blair et al., 1997; Marsh et al., 2005; Walker and Leister, 1994). Ethologists find similar aggressioninhibiting effects of distress cues in other primates and have speculated that distress cues evolved for this purpose (Preuschoft, 2000). Developmental, behavioral, and clinical research shows that distress cues elicit empathy in those who see them (Hoffman, 1987; Marsh and Ambady, 2007; Nichols, 2001; Preston and de Waal, 2002). Empathy is generally associated with decreased antisocial behavior (Eisenberg, 2000). It has been proposed that distress cues possess perceptual properties that elicit empathy and inhibit aggression (Marsh et al., 2005). It has also been proposed that fearful and sad expressions serve as social reinforcers that condition developing children to avoid engaging in the antisocial behaviors that elicit these expressions. This process is specified by the Integrated Emotion Systems (IES) model (Blair, 2005).

Facial affect processing relies on a distributed network of structures that includes occipitotemporal cortex (particularly fusiform gyrus and superior temporal gyrus), anterior cingulate cortex, amygdala, and ventromedial prefrontal cortex (Adolphs, 2006; Murphy et al., 2003). Evidence is accumulating, however, that beyond this general network partially separable neural systems process different expressions. Detection of fearful expressions relies disproportionately on the amygdala, and the detection of disgust on the insula and basal ganglia (Adolphs, 2002; Phillips et al., 2004; Murphy et al., 2003). Evidence that antisocial individuals are impaired in processing specific expressions like fear or disgust could facilitate targeted research into neurocognitive deficits underlying antisocial behavior.

Antisocial behaviors are those that violate the rights or welfare of other individuals. Multiple studies have shown specific impairments in fearful expression processing in populations who engage in these behaviors (e.g., Blair et al., 2004; Blair and Cipolotti, 2000; Carr and Lutjemeier, 2005; Walker and Leister, 1994; Woodbury-Smith et al., 2005). These populations include those primarily classified by the presence of antisocial behaviors (e.g., aggressive, criminal, externalizing, abusive) and those classified on the basis of both antisocial behaviors and personality traits such as a lack of empathy and remorse (e.g., psychopaths). However, not all of these studies find fearful expression recognition impairments (Kosson et al., 2002). Many studies that find fear recognition deficits assess children (Blair and Coles, 2000; Blair et al., 2001; Stevens et al., 2001). This leaves questions remaining, such as whether apparently specific deficits in recognizing fearful expressions stem from task difficulty rather than fear-specific neuropsychological deficits.

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