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The roles of orbital frontal cortex in the modulation of antisocial behavior

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Abstract

This article considers potential roles of orbital frontal cortex in the modulation of antisocial behavior. Two forms of aggression are distinguished: reactive aggression elicited in response to frustration/threat and goal directed, instrumental aggression. It is suggested that orbital frontal cortex is directly involved in the modulation of reactive aggression. It is argued that orbital frontal cortex does not ''inhibit'' reactive aggression but rather may both increase or decrease its probability as a function of social cues present in the environment. Early dysfunction in this function of orbital frontal cortex may be linked to the development of Borderline Personality Disorder. Instrumental aggression is linked to a fundamental failure in moral socialization. However, the available data suggest that the amygdala, but not orbital frontal cortex, is required for functions such as aversive conditioning and passive avoidance learning that are necessary for moral socialization. Psychopathic individuals who present with significant instrumental aggression, are impaired in aversive conditioning and passive avoidance learning and show evidence of amygdala dysfunction. Orbital frontal cortex and the amygdala are involved in response reversal where instrumental responses must be reversed following contingency change. Impairments in response reversal are also seen in psychopathic individuals. However, it remains unclear whether impairment in response reversal per se is associated with antisocial behavior.
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1. Introduction

The level of antisocial behavior in society is a continual source of concern. More than 3 million violent crimes are committed in the US annually (Reiss, Miczek, & Roth, 1994). Twenty thousand of these involve the murder of Americans by gunfire (Sourcebook of Criminal Justice Statistics Online, 1998). There is a growing body of data indicating that there are neurobiological risk factors for antisocial behavior. In this article the role of orbital frontal cortex in the modulation of antisocial behavior will be considered.

It is necessary to first draw a distinction will be drawn between reactive and instrumental aggression (cf. Barratt, Stanford, Dowdy, Liebman, & Kent, 1999; Barratt, Stanford, Kent, & Felthous, 1997; Berkowitz, 1993; Linnoila et al., 1983). In reactive aggression (also referred to as affective aggression), a frustrating or threatening event triggers the aggressive act and frequently also induces anger. Importantly, the aggression is initiated without regard for any potential goal (for example, gaining the victim's possessions or increasing status within the hierarchy). In contrast, instrumental aggression (also referred to as proactive aggression) is purposeful and goal directed. The aggression is used instrumentally to achieve a specific desired goal (Berkowitz, 1993). This is not usually the pain of the victim but rather the victim's possessions or to increase status within a group hierarchy. Bullying is an example of instrumental aggression and, unsurprisingly, individuals who engage in bullying behaviors, frequently engage in other forms of instrumental antisocial behavior in other contexts (Roland & Idsoe, 2001).

The distinction between reactive and instrumental aggression has been criticized because of some difficulty in characterizing the nature of specific human aggressive episodes (Bushman & Anderson, 2001). However, there is considerable data that there are two relatively

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separable populations of aggressive individuals; individuals who present with solely reactive aggression and individuals who present with very high levels of instrumental aggression and also reactive aggression (Barratt et al., 1999; Crick & Dodge, 1996; Linnoila et al., 1983). The potential role of orbital frontal cortex in the modulation of both these forms of aggression will be discussed in turn.

2. Orbital frontal cortex and reactive aggression

In many respects, considerably more is known about reactive than instrumental aggression. This is because a dedicated neural circuitry mediates reactive aggression and humans share this circuitry with other mammalian species (Gregg & Siegel, 2001; Panksepp, 1998). A circuit has been identified that runs from medial amygdaloidal areas downward, largely via the stria terminalis to the medial hypothalamus, and from there to the dorsal half of the periaqueductal gray (PAG). The system is organized in a hierarchical manner such that aggression evoked from the amygdala is dependent on functional integrity of the medial hypothalamus and PAG but that aggression evoked from the PAG is not dependent on the functional integrity of the amygdala (Bandler, 1988; Gregg & Siegel, 2001; Panksepp, 1998). This circuitry mediates the animal's response to threat. At low levels of stimulation, from a distant threat, the animal will freeze. At higher levels, from a closer threat, the animal will attempt to escape the environment. At higher levels still, when the threat is very close and escape is impossible, the animal will display reactive aggression (Blanchard, Blanchard, & Takahashi, 1977).

Both the amygdala and orbital frontal cortex modulate the neural circuitry mediating reactive aggression though their roles differ. The amygdala is known to react to reinforcing as well as aversive stimuli (Everitt, Cardinal, Hall, Parkinson, & Robbins, 2000). This suggests that the amygdala would be in a position to both upgrade (as a response to an aversive stimulus) or downgrade (as a response to reinforcement) the responsiveness of the sub-cortical systems that respond to threat. Indeed, this is suggested by the augmentation of the startle reflex literature. The startle reflex is mediated by the sub-cortical systems that respond to threat. It can be modulated by the presence of visual or auditory primes that occur shortly before the startle stimulus. Aversive visual threat primes augment the magnitude of the startle reflex relative to neutral primes while appetitive visual primes reduce the magnitude of the startle reflex (Lang, Bradley, & Cuthbert, 1990). This modulation is achieved by the operation of the amygdala on the sub-cortical systems responding to threat that generate the reflex (Angrilli et al., 1996; Campeau & Davis, 1995; Davis, 2000; Funayama, Grillon, Davis, & Phelps, 2001). Given the ability of the amygdala to upgrade or downgrade the responsiveness of the sub-cortical systems that respond to threat as indicated by the startle reflex literature, amygdala lesions might therefore reduce the probability of reactive aggression in threatening circumstances by reducing the patient's sensitivity to learned threat. Learned threats would not activate the amygdala and through the amygdala, the sub-cortical system mediating reactive aggression. However, amygdala lesions might also increase the probability of reactive aggression in non-threatening circumstances. The amygdala lesion would prevent the suppression of reactive aggression as a function of amygdala activation by appetitive stimuli in the environment. This suggests that amygdala lesions might either increase or decrease the probability of reactive aggression depending on the contextual parameters the animal is exposed to. The literature certainly indicates that amygdala lesions can both increase or decrease the probability of reactive aggression. Thus, bilateral amygdalectomies have been reported to decrease aggressive behavior in 70–76% of cases (Ramamurthi, 1988). However, very severe amygdalar atrophy is found in a significant subgroup of aggressive patients with temporal lobe epilepsy (van Elst, Woermann, Lemieux, Thompson, & Trimble, 2000). Moreover, unilateral damage to the central nucleus of the amygdala in cats increases the expression of reactive aggression (Zagrodzka, Hedberg, Mann, & Morrison, 1998).

Both the animal, and human neuro-psychological literature, suggest that frontal cortex is involved in the modulation of the sub-cortical circuit mediating reactive aggression (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Grafman, Schwab, Warden, Pridgen, & Brown, 1996; Gregg & Siegel, 2001; Panksepp, 1998; Pennington & Bennetto, 1993). Certainly, damage to medial frontal and orbital frontal cortex is associated with increased risk for the display of reactive aggression in humans whether the lesion occurs in childhood (Anderson et al., 1999; Pennington & Bennetto, 1993) or adulthood (Grafman et al., 1996). In addition, there are considerable neuro-imaging data assessing the neural functioning of patients with reactive aggression. These data have revealed reduced frontal functioning in patients presenting with reactive aggression (Soderstrom, Tullberg, Wikkelso, Ekholm, & Forsman, 2000; Volkow & Tancredi, 1987; Volkow et al., 1995). Interestingly, this reduced frontal functioning is not observed in patients presenting with predominantly instrumental aggression (Raine et al., 1998). This is consistent with neuro-psychological data that indicate that psychopathic individuals, individuals who present with marked instrumental aggression, do not present with poor performance on general measures of frontal lobe functioning (Kandel & Freed, 1989; LaPierre, Braun, & Hodgins, 1995; Mitchell, Colledge, Leonard, & Blair, 2002).