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Neuronal correlates of reward and loss in Cluster B personality disorders: A functional magnetic resonance imaging study

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Abstract

Decision making is guided by the likely consequences of behavioural choices. Neuronal correlates of financial reward have been described in a number of functional imaging studies in humans. Areas implicated in reward include ventral striatum, dopaminergic midbrain, amygdala and orbitofrontal cortex. Response to loss has not been as extensively studied but may involve prefrontal and medial temporal cortices. It has been proposed that increased sensitivity to reward and reduced sensitivity to punishment underlie some of the psychopathology in impulsive personality disordered individuals. However, few imaging studies using reinforcement tasks have been conducted in this group. In this fMRI study, we investigate the effects of positive (monetary reward) and negative (monetary loss) outcomes on BOLD responses in two target selection tasks. The experimental group comprised eight people with Cluster B (antisocial and borderline) personality disorder, whilst the control group contained fourteen healthy participants. A key finding was the absence of prefrontal responses and reduced BOLD signal in the subcortical reward system in the PD group during positive reinforcement. Impulsivity scores correlated negatively with prefrontal responses in the PD but not the control group during both, reward and loss. Our results suggest dysfunctional responses to rewarding and aversive stimuli in Cluster B personality disordered individuals but do not support the notion of hypersensitivity to reward and hyposensitivity to loss.

Keywords: fMRI; Borderline personality disorder; Antisocial personality disorder; Positive reinforcement; Orbitofrontal cortex

1. Introduction

Rewarding and punishing stimuli result in an increase or decrease of the probability of antecedant actions, thereby shaping behaviour. In recent years, functional neuroimaging studies in humans using primary (e.g. O'Doherty et al., 2001a, 2002) and abstract rewards (e.g. Breiter et al., 2001; Elliott et al., 2000, 2003) have advanced our understanding of the neuronal correlates of reinforcement processing and have corroborated previous findings from single-cell electrophysiological and lesion studies in animals. This research has implicated a network of interconnected brain regions mediating the behavioural and motivational effects of reward, including ventral striatum, dopaminergic midbrain, amygdala and orbitofrontal cortex (OFC; for a review see O'Doherty, 2004). Distinct functions have been attributed to these different regions. For instance, amygdala, striatum and midbrain have been found to respond to the presence of reward regardless of value (e.g. Elliott et al., 2000, 2003); in

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Table 1
Behavioural results

Variable	PD group	Control group
	$N=8$ [a]	$N=14$ [b]
RT reward blocks	531.4 (57.3)	498.9 (62.5)
RT no-reward blocks	562.2 (58.8)	545.2 (66.3)
OE reward blocks	0.0	0.0
OE no-reward blocks	0.0	0.0
CE reward blocks	0.0	0.5
CE no-reward blocks	0.0	1.0
RT loss blocks	492.4 (79.4)	459.9 (56.3)
RT no-loss blocks	539.5 (58.7)	510.3 (45.6)
OE loss blocks	0.0	0.0
OE no-loss blocks	0.0	0.0
CE loss blocks	1.0	0.0
CE no-loss blocks	0.0	0.0

RT: Reaction time, means in ms (standard deviation in brackets).
OE: Omission error, CE: commission error; median.
[a] No-reward block OE and CE: 7 subjects after exclusion of one outlier with exceptionally high error rates in each of these blocks.
[b] Loss block CE: 13 subjects included in analysis after exclusion of one outlier.

contrast, a more complex pattern of responses has been identified in medial and lateral OFC suggesting a possible role for higher order processing of reinforcing stimuli, such as the integration of stimulus attributes and emotional value (e.g. Elliott et al., 2003; Kringelbach et al., 2003; O'Doherty, 2004). This in turn allows the salience of reinforcing stimuli to be updated and modulated following changes in contingencies and the subsequent use of this information in action selection.

The role of reward system components in the response to punishment or loss is less clear. Several fMRI studies in humans have suggested the striatum has an important role. Jensen et al. (2003) have identified ventral striatum responses in anticipation of sensory aversive stimuli. Other authors have shown ventral striatum activity associated with anticipation and following the presentation of both monetary rewards and punishments (e.g. Knutson et al., 2001; Delgado et al., 2003). Decreased BOLD signal in dorsal and ventral striatum has been observed following punishing feedback (Delgado et al., 2000). These findings suggest that this structure is not functionally specific to reward but may have a more general role in the processing of reinforcing stimuli. Other authors have identified BOLD signal changes in lateral OFC following punishment (e.g. O'Doherty et al., 2001b; Remijnse et al., 2005). This might reflect the involvement of this structure in response inhibition (Aron et al., 2003). In addition, anterior cingulate and thalamus (Knutson et al., 2000), right amygdala (Zalla et al., 2000), insula (O'Doherty et al., 2003) and hippocampus/parahippocampus (Elliott et al., 2000) have been associated with the experience of loss or punishment in humans.

Dysfunctional responses to reinforcing stimuli have been proposed to underlie the psychopathology in substance use and impulsivity-related personality disorders (Petry, 2002). The latter encompass two personality disorders within Cluster B of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994): antisocial (ASPD) and borderline (BPD) personality disorder. These two disorders share some common characteristics, particularly high levels of impulsive behaviour; some authors have argued that ASPD and BPD are manifestations of the same underlying pathology in male and female individuals respectively (e.g. Paris, 1997). There is considerable co-morbidity between these two personality disorders: in male individuals a co-occurrence of up to 50% has been identified (Zanarini et al., 1998; Chabrol and Leichsenring, 2006). Becker et al. (2005) noted that symptoms related to impulsive behaviour in BPD were not significantly more efficient in diagnosing BPD than ASPD. It therefore seems justified to consider these two disorders together as an impulsivity-related personality disorder as has previously been suggested by other authors (e. g. Goethals et al., 2005).

A number of aetiological models of impulsivity-related personality disorders have been put forward. Early accounts (Gray, 1987) postulated two distinct motivational systems: a behavioural activation system (BAS) which is sensitive to reward cues and a behavioural inhibition system (BIS) which is sensitive to punishment. In this model, impulsive–aggressive behaviour as observed in impulsive Cluster B PD is proposed to result from an imbalance of these two systems, either due to an oversensitivity of the BAS or due to hyporesponsiveness of the BIS.

More recent models of impulsive personality disorders have focused on behavioural choice in the context of reward and punishment. The reward dominance theory suggests that antisocial individuals show greater responsivity to reward and decreased sensitivity to punishment in situations where both types of stimuli are available (Scerbo et al., 1990). Impulsive individuals focus on the prospect of reward even if environmental cues indicate possible later punishment (Budhani and Blair, 2005). Preference for shorter delays in reward-choice tasks has been demonstrated in borderline (Dougherty et al., 1999) and antisocial personality disorder (Moeller et al., 2002) and in probation and parole groups (Cherek et al., 1999, 1997). Other authors have shown that antisocial groups perform poorly on passive avoidance tasks by failing to inhibit punishable responses (e. g. Dikman and Allen, 2000).