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Impaired recognition of facial expressions of anger in Parkinson's disease patients acutely withdrawn from dopamine replacement therapy

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Abstract

We have previously reported that acute dopaminergic blockade in healthy volunteers results in a transient disruption of the recognition of facial expressions of anger, whilst leaving intact the recognition of other facial expressions (including fear and disgust) and facial identity processing. Parkinson's disease (PD) is characterised by cell loss in dopaminergic neuronal populations, and hence we predicted that PD would be associated with impaired anger recognition. We reasoned that treatment with dopamine replacement therapy (DRT) could mask any deficit present in PD, and therefore studied facial expression recognition in a group of PD patients transiently withdrawn from DRT. Seventeen PD patients were compared to 21 age- and IQ-matched controls on the Ekman 60 task, which required the forced-choice labelling of 10 exemplars of each of six facial expressions (anger, disgust, fear, sadness, happiness, surprise). In line with our predictions, PD patients showed a selective impairment in the recognition of facial expressions of anger. This deficit was not related to the PD patients' performance on the Benton unfamiliar-face matching task, which was normal, nor was the deficit related to overall disease severity, or to depression symptoms. However, as predicted by simulation theories, impaired anger recognition in PD was related to reduced levels of the anger-linked temperament trait, exploratory excitability. The results extend our previous findings of a role for dopamine in the processing of facial expressions of anger, and demonstrate the power of adopting a phylogenetic, comparative perspective on emotions.

Keywords: Anger; Dopamine; Emotion; Novelty seeking; Reward; Simulation theory

1. Introduction

Neuropsychology has provided evidence of strikingly selective impairments in the recognition of facial expressions of fear (Adolphs, Tranel, Damasio, & Damasio, 1994; Calder et al., 1996; Sprengelmeyer et al., 1999) and disgust (Adolphs, Tranel, & Damasio, 2003; Calder, Keane, Manes, Antoun, & Young, 2000). Clear double dissociations have been found between impaired recognition of facial expressions of these emotions (Calder, Lawrence, & Young, 2001), suggesting that different neural systems are specialised, at least in part, for the recognition of different classes of emotion expression, presumably related to their evolutionary history (Lawrence & Calder, 2004). A notable feature of such impairments in the recognition of conspecific facial expressions is that they reliably co-occur with alterations in the experience of the same emotion. These findings have been taken as support for 'simulationist' models of facial emotion recognition, in which the emotion states of others are recognised by mental simulation, i.e. by generating similar states in oneself (Goldman & Sripada, 2005; Gordon, 1992).

An outstanding question is whether evidence of selective impairments in the recognition of facial expressions of emotions other than fear and disgust can be found. Following Griffiths (2001), we have argued (Lawrence & Calder, 2004) that research on emotion expression recognition could benefit from the adoption of a phylogenetic perspective—that is, a search for emotion processing systems in humans that are elaborations of mechanisms seen in other species.

Two main motivational systems govern mammalian conspecific aggression. One controls offensive aggression and the other defensive aggression (Blanchard & Blanchard, 1984, 1988, 1989, 2003). Offensive aggression occurs in the context of conspecific challenge over adaptively important resources. It involves a set of species-typical behaviours enabling the individual to pursue and contact particular body sites on the opponent where blows (in humans including slaps and over-arm

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or closed-fist blows) are delivered. Its successful outcome is the termination of the resource dispute. There is evidence for behavioural homology in the expressions/signals (the two terms need not be strongly differentiated—Ekman, 1997) of offensive attack across several mammalian species, including, in primates, characteristic facial expressions (Blanchard & Blanchard, 1984, 1988, 1989; Chevalier-Skolnikoff, 1973; Darwin, 1872/1965; Ekman, 1972). The emotional basis of offensive aggression is thought to be homologous (in mammals) with (certain instances of) human anger (Blanchard & Blanchard, 1984).

In contrast, defensive attack occurs in the context of immediate threat to the individual, from either a conspecific or non-conspecific, e.g. a predator. Defensive attack is seen only when the individual is defending its own body, not when it is attacking another animal to "defend" a disputed resource. The latter situation involves offensive attack. Defensive attack includes a salient threat component (not seen in offensive attack), and involves a set of species-typical behaviours different from those seen in offensive attack, e.g. loud vocalizations and display of "weapons" (e.g. claws). The bites or blows delivered tend to be made on different body sites on the opponent than those contacted in offensive aggression. The successful outcome of defensive aggression is discouragement of the body-threatening conspecific or predator. There is evidence for behavioural homology in the expressions of defensive attack across several mammalian species, including, in primates, characteristic facial expressions (Blanchard & Blanchard, 1984, 1988, 1989; Blanchard, Hynd, Minke, Minemoto, & Blanchard, 2001; Chevalier-Skolnikoff, 1973; Darwin, 1872/1965; Ekman, 1972). The emotional basis of defensive aggression is thought to be homologous (in mammals) with (certain instances of) human fear (Blanchard et al., 2001).

Comparative neuropsychological research has suggested that specific mammalian neural systems are involved in offensive aggression. For example, post-mortem radioenzymatic and in vivo microdialysis experiments in rats have shown that hypothalamic and ventral striatal dopamine levels are elevated in anticipation of, and during, conspecific aggressive encounters (Barr, Sharpless, & Gibbons, 1979; Ferrari, van Erp, Tornatzky, & Miczek, 2003; Louilot, LeMoal, & Simon, 1986; van Erp & Miczek, 2000). Further, acute administration of dopamine antagonists, including those of the D2 receptor, such as sulpiride, or dopamine release inhibitors, selectively impairs offensive aggression in rodents (Aguilar, Miñarro, Pérez-Iranzo, & Simòn, 1994; Felip, Rodríguez-Arias, Aguilar, & Miñarro, 2001; Kudryavtseva, Lipina, & Koryakina, 1999; Masur, Maroni, & Benedito, 1975; Redolat, Brain, & Simón, 1991; Simón, Minarro, Redolat, & Garmendia, 1989), whilst leaving intact general locomotion. In primates, the dopamine-rich rhinal cortex (Lewis & Sesack, 1997) is another region implicated in offensive aggression (Meunier & Bachevalier, 2002). The system(s) involved in offensive aggression appear dissociable from an amygdala-centred system underpinning fear states, including those motivating defensive aggression against both conspecifics and predators (Blanchard & Takahashi, 1988; Meunier & Bachevalier, 2002; Oakes & Coover, 1997).

Given the role of dopamine in mammalian offensive aggression, together with evidence for behavioural homology in mammalian expressions of offensive aggression, and the predictions of simulation theory – that impairments in the ability to generate an emotion state would impair recognition of that same emotion expressed in the faces of conspecifics – we predicted that dopaminergic manipulations that reduce offensive aggression in other mammals would impair recognition of conspecific offensive aggression expressions, specifically, facial expressions of anger, in humans. In line with these predictions, we have shown that acute dopaminergic blockade in healthy volunteers results in a transient disruption of the recognition of facial expressions of anger (Lawrence, Calder, McGowan, & Grasby, 2002). In a within-subject, double-blind, placebo-controlled cross-over experiment, we found that acute dopaminergic blockade, produced by the administration of sulpiride (a dopamine D2 receptor antagonist) produced a selective disruption in the recognition of anger facial expressions, but left intact the recognition of other facial expressions (including fear and disgust), and facial identity processing (as measured by the Benton unfamiliar-face matching task).

Parkinson's disease (PD) is characterised by cell loss in dopaminergic neuronal populations (Hornykiewicz & Kish, 1986) and hence the prediction is that PD would be associated with impaired anger recognition. We reasoned that treatment with dopamine replacement therapy (DRT) could mask any deficit present in PD, and therefore studied facial expression recognition in a group of PD patients transiently withdrawn from DRT. We tested the hypothesis that, when acutely withdrawn from DRT, PD patients, relative to age- and IQ-matched healthy volunteers, would show impaired recognition of facial expressions of anger, whilst showing relatively spared recognition of other emotions and facial identity processing. Further, based on previous findings of paired deficits in emotion recognition and emotion experience, consistent with simulationist models of emotion recognition, we predicted that impaired anger recognition in PD would be linked to impaired anger experience. Exploratory excitability, a sub-component of novelty seeking (NS), as measured by Cloninger's tridimensional personality questionnaire (TPQ, Cloninger, 1987b) is an anger-linked temperament trait (Cloninger, 1987a), levels of which are reduced in PD (Fuji, Harada, Ohkoshi, Hayashi, & Yoshizawa, 2000). It predicts trait levels of outwardly directed anger in large samples of healthy volunteers (Giancola, Zeichner, Newbolt, & Stennett, 1994; Svrakic, Przybeck, Whitehead, & Cloninger, 1999). Notably, a similar relationship between exploratory excitability and offensive aggression appears to exist in rats (Kazlauckas et al., 2005). We predicted that anger recognition in PD would be correlated with levels of the anger-linked trait exploratory excitability.

2. Methods

2.1. Participants

Seventeen PD patients (7 females) were compared to 21 age ($t(36) = 0.60$, $P = 0.56$) and reading-estimated IQ (Nelson, 1991) ($t(36) = 0.29$, $P = 0.78$)-