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Specific impairments in the recognition of emotional facial expressions in Parkinson's disease

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ABSTRACT

Studies investigating the ability to recognize emotional facial expressions in non-demented individuals with Parkinson's disease (PD) have yielded equivocal findings. A possible reason for this variability may lie in the confounding of emotion recognition with cognitive task requirements, a confound arising from the lack of a control condition using non-emotional stimuli. The present study examined emotional facial expression recognition abilities in 20 non-demented patients with PD and 23 control participants relative to their performance on a non-emotional landscape categorization test with comparable task requirements. We found that PD participants were normal on the control task but exhibited selective impairments in the recognition of facial emotion, specifically for anger (driven by those with right hemisphere pathology) and surprise (driven by those with left hemisphere pathology), even when controlling for depression level. Male but not female PD participants further displayed specific deficits in the recognition of fearful expressions. We suggest that the neural substrates that may subserve these impairments include the ventral striatum, amygdala, and prefrontal cortices. Finally, we observed that in PD participants, deficiencies in facial emotion recognition correlated with higher levels of interpersonal distress, which calls attention to the significant psychosocial impact that facial emotion recognition impairments may have on individuals with PD.

1. Introduction

One of the most basic elements of emotional functioning, and one of the components most critical to social behavior, is the recognition of the emotional states of others (Darwin, 1872/1965). In Parkinson's disease (PD), facial emotion identification deficits have been reported in several studies (Dujardin et al., 2004; Jacobs, Shuren, Bowers, & Heilman, 1995; Kan, Kawamura, Hasegawa, Mochizuki, & Nakamura, 2002; Lawrence, Goerendt, & Brooks, 2007; Sprengelmeyer et al., 2003), though not in all (Adolphs, Schul, & Tranel, 1998; Pell & Leonard, 2005). Where deficits are found, there is as yet little consensus as to whether or not they apply to the recognition of specific emotions. Kan et al. (2002) noted deficits in the recognition of fear and disgust in medicated PD participants. Sprengelmeyer et al. (2003) found that the recognition of anger and fear was disrupted in medicated PD participants, and that the recognition of fear, sadness, disgust, and anger was impaired in unmedicated PD patients. Dujardin et al. (2004) observed that unmedicated PD participants were less accurate than healthy participants in perceiving facial expressions of anger, sadness, and disgust. More recently, Lawrence et al. (2007) reported that the recognition of anger was impaired in PD patients who had been temporarily removed from dopamine replacement therapy.

It is generally argued that abnormalities in facial emotion recognition in PD arise from losses of dopaminergic neurons resulting in dysfunction of fronto-subcortical systems (e.g., Dujardin et al., 2004; Lawrence et al., 2007; Sprengelmeyer et al., 2003). With growing evidence that dissociable neural substrates are involved in the recognition of different emotions (Adolphs, 2002; Posamentier & Abdi, 2003), gaining greater clarity on emotion recognition impairments in PD would help in ascertaining which neural substrates may underlie the disruption of emotion recognition. The inconsistency in the literature may be related to the variability in medication status of PD patients across studies; however, medication status may not explain these inconsistencies entirely. Notably, it has been suggested that the typical methods of investigating emotion recognition, particularly in neurologic patient populations, may result in artifactual findings that are related to task difficulty factors rather than to impairments in emotion recognition abilities arising from disruption of specific neuroanatomical structures

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(Rapcsak et al., 2000). Inconsistent findings in the PD literature may reflect differences in difficulty levels across emotions within a study. This effect may be compounded by the differences in the difficulty level of stimuli presented (within each category of emotion), which likely vary among studies.

A second source of inconsistency may come from the confounding of emotion recognition abilities with unrelated task requirements. Executive function impairments, a hallmark of frontal dysfunction, are commonly noted in non-demented PD patients (e.g., Zgaljardic, Borod, Foldi, & Mattis, 2003). Frontally mediated impairments – mainly in decision-making and categorization abilities – may have an impact on PD patients' performance on measures of facial emotion recognition. PD patients have shown impairments on tasks of explicit decision making ability (e.g., the Game of Dice task), which correlated with decreases in executive functioning abilities (Brand et al., 2004), and on tests of implicit decision making (e.g., Iowa Gambling Task), in which PET imaging revealed decreased activation in the orbitofrontal cortex (Thiel et al., 2003). Several studies have indicated that the ability to learn categorization rules is impaired in PD (e.g., Ashby, Noble, Filoteo, Waldron, & Ell, 2003; Knowlton, Mangels, & Squire, 1996; Maddox, Aparicio, Marchant, & Ivry, 2005; Maddox & Filoteo, 2001; Price, 2006). Most recently, Filoteo et al. (2007) suggested that the presence of extraneous stimuli features may impair PD patients' performance on categorization tasks due to increased demands on selective attention processes.

Taken together, these findings are highly relevant to studies of emotion identification that require participants to categorize facial expressions. The methods most often used to assess emotion recognition abilities require that the participant call upon several skills known to be disrupted in PD. Without employing a suitable control task to studies of facial emotion perception, it is difficult to ascertain whether PD participants' poor performance on tasks of emotion recognition is due to an inability to identify emotions, or whether their difficulties arise from deficits in decision making, categorization skills, or the ability to identify the most salient features demarcating category boundaries. Impairments in the recognition of specific emotions may be related to the latter three possibilities, as any could result in increased error rates.

To help resolve these issues, the present study examined the facial emotion recognition abilities of PD and healthy normal control participants relative to performance on a non-emotional categorization test with comparable task requirements. Landscapes were chosen because they provided a sufficient number of image categories, and, like faces, they are mono-oriented and are composed of several smaller elements that can be individually assessed and integrated when categorizing the image.

A further aim of this study was to examine the relation between emotion recognition and body side of motor onset in PD. Motor symptoms in PD usually have a unilateral onset, and this pattern generally persists throughout the progression of the disease (Lee et al., 1995). There is evidence to suggest that this asymmetry is associated with reduced dopamine levels and abnormalities in the dopamine receptors of the contralateral hemisphere (Innis et al., 1993; Kempster, Gibb, Stern, & Lees, 1989), which persist even after motor symptoms have progressed to a more bilateral presentation (Antonini et al., 1995). Because differences in side of motor onset are associated with dysfunction of hemisphere-specific cognitive abilities in PD (e.g., Amick, Grace, & Chou, 2006; Amick, Schendan, Ganis, & Cronin-Golomb, 2006), in addition to the fact that the right hemisphere is thought to be more active than the left in processing emotional material, we examined the performance of PD participants with right and left motor symptom onset separately. We also examined whether men and women with PD display differences in emotion recognition, as men and women with PD may experience different disease effects (e.g., Haaxma et al., 2007; Shulman, 2007), and numerous studies have shown male–female differences in emotion recognition abilities (e.g., Hall, Carter, Horgan, & Fischer, 2000; Hall & Matsumoto, 2004; Thayer & Johnsen, 2000).

A final aim was to assess whether impairments in emotion recognition, if identified, are associated with increased difficulties in interpersonal relationships. Research in other neuropsychiatric patient populations suggests that abnormalities in facial emotion recognition correlate with declines in interpersonal interactions (Kornreich et al., 2002; Shimokawa et al., 2001). We hypothesized that similar reductions in social interaction may occur in PD patients. Based on reports that female PD patients, compared to male PD patients, tend to endorse more difficulties on overall quality of life measures, which also address social functioning (Behari, Srivastava, & Pandey, 2005; Kuopio, Marttila, Helenius, Toivonen, & Rinne, 2000; Shulman, 2007), we hypothesized that female PD patients in our group would report more interpersonal difficulties than male PD patients.

2. Participants

Participants included 20 individuals with idiopathic PD (10 men, 10 women) and 23 healthy control (HC) participants (11 men, 12 women). The groups did not differ with respect to age or education and were not demented. All attained scores above the cutoff of 27 on the Mini-Mental State Exam (Folstein, Folstein, & McHugh, 1975). PD participants additionally scored above our cutoff of 136 on the Dementia Rating Scale-2 (DRS-2) (Jurica, Leitten, & Mattis, 2001), which is well above the clinical cutoff for dementia. See Table 1 for details of participant characteristics. Participants with PD were recruited from the Parkinson's Disease Clinic at the Boston Medical Center and through support groups; the HC group was recruited from the community. All participants were right-handed except for two PD and one HC individuals. All were required to be native speakers of English. We excluded participation on the basis of history of uncorrected abnormal vision or hearing; psychiatric illness (including diagnosis of depression or anxiety); neurological

Table 1
Demographic characteristics of the participant groups

Variable	PD group (M/F = 10/10; RPD = 9, LPD = 11)		HC group (M/F = 11/12)	
	M	S.D.	*M*	S.D.
Age (years)	60.2	8.2	59.5	6.8
Education (years)	15.8	2.6	16.7	1.5
Disease duration (years)	7.3	4.2	n/a	n/a
Hoehn & Yahr rating score (Median)	2.0	n/a	n/a	n/a
Dementia Rating Scale (/144)	142.6	2.2	n/a	n/a
Mini-Mental State Exam (/30)	28.7	1.4	28.9	0.9
Beck Depression Inventory (/63)	10.0**	7.7	4.3**	4.4
Beck Anxiety Inventory (/63)	10.8**	8.6	3.2**	4.3

Note: PD = Parkinson's disease; M/F = male–female; RPD = right body side of motor onset; LPD = left body side of motor onset; HC = healthy control; *M* = mean (or median where noted). Asterisks indicate that the groups' means are significantly different at the $p < .01$ level (**).