



Facial expression recognition in people with medicated and unmedicated Parkinson's disease

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Abstract

Recognition of facial expressions of emotion was investigated in people with medicated and unmedicated Parkinson's disease (PD) and matched controls (unmedicated PD, $n = 16$; medicated PD, $n = 20$; controls, $n = 40$). Participants in the medicated group showed some visual impairment (impaired contrast sensitivity) and performed less well on perception of unfamiliar face identity, but did not show significant deficits in the perception of sex, gaze direction, or familiar identity from the face. For both Parkinson's disease groups, there was evidence of impaired recognition of facial expressions in comparison to controls. These deficits were more consistently noted in the unmedicated group, who were also found to perform worse than the medicated group at recognising disgust from prototypical facial expressions, and at recognising anger and disgust in computer-manipulated images. Although both Parkinson's disease groups showed impairments of facial expression recognition, the consistently worse recognition of disgust in the unmedicated group is consistent with the hypothesis from previous studies that brain regions modulated by dopaminergic neurons are involved in the recognition of disgust.

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1. Introduction

Neuropsychological studies have shown deficits affecting the recognition of specific emotions. People with lesions involving the amygdala show differentially severely impaired recognition of facial expressions of fear [2,3,9,10]. Investigation of one of these cases also revealed impaired recognition of fear (and anger) to auditory stimuli [37] and Sprengelmeyer et al. [42] reported deficits in recognition of facially, vocally and gesturally displayed fear in a further patient with bilateral gliosis of the amygdala. These neuropsychological results are paralleled by demonstrations of activation of the human amygdala in response to facial and vocal expressions of fear in functional imaging studies of normal subjects [8,26,32,33].

In contrast, people with symptomatic Huntington's disease [40,43] and pre-symptomatic Huntington's disease gene carriers [17] are particularly poor at recognising facial expressions of disgust. The findings from Huntington's

disease point to the possibility that fronto-striatal regions and especially the basal ganglia (widely considered to form the core site of pathology in Huntington's disease) are implicated in recognition of disgust. This hypothesis is supported by findings of impaired recognition of disgust in people with Gilles de la Tourette's syndrome with co-present obsessive-compulsive behaviours and for people with obsessive-compulsive disorder [41], since neurophysiological and neuropsychological studies of these disorders also highlight fronto-striatal abnormalities [25,34]. Supporting evidence that the basal ganglia are involved in recognition of facial expressions of disgust comes from two functional imaging (fMRI) studies in which activation of the putamen in response to faces depicting disgust has been reported [33,39].

Because of this evidence that the basal ganglia are involved in recognition of facial expressions of disgust, we investigated recognition of facial expressions by people with Parkinson's disease (PD). The pathology in Parkinson's disease especially affects the dopaminergic system, allowing us to study the possible contribution of this neurotransmitter system to processes of facial expression recognition in general and to the recognition of disgust, in particular.

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To provide evidence concerning the involvement in face related information processing of brain regions modulated by dopaminergic neurons, we looked in detail at face perception and recognition of emotions in people with Parkinson's disease who were and were not receiving dopamine replacement therapy using an extensive battery of tasks to explore the perception of age, gender, unfamiliar face identity, gaze direction and recognition of emotional expression from the face. We were able to compare the performance of people with medicated and unmedicated Parkinson's disease to that of neurologically normal controls, and to compare the performance of medicated and unmedicated Parkinson's disease groups to each other. The latter comparison is of particular interest to identifying the potential contributions of the dopaminergic system.

2. Subjects

Thirty-six people suffering from idiopathic Parkinson's disease and 40 controls gave their informed consent to take part in the study. Twenty of these participants who were in the more advanced stages were receiving medication for Parkinson's disease (the *medicated* group), and 16 people in the early stages of the disease were not receiving dopaminergic medication (the *unmedicated* group). Note that five participants from the unmedicated group were receiving anti-depressants; the terms 'medicated' and 'unmedicated' refer to dopaminergic medication for Parkinson's disease.

The mean age of the participants in the unmedicated Parkinson's disease group ($n = 16$; 8 female, 8 male) was 56.7 years (S.D. 10.6 years) and mean duration of schooling was 8.3 years (S.D. 0.8 years). Their average IQ was 100.0 (S.D. 9.6) as assessed by the Kurztest für allgemeine Intelligenz (KAI, [22]).

Severity of the disease was rated using both the Hoehn and Yahr scale [19] and the first three subscales of the Unified Parkinson's Disease Rating Scale (UPDRS, [15]). The people with unmedicated Parkinson's disease ranged between 1 and 2.5 on the Hoehn and Yahr scale, with a mean of 1.7 (S.D. 0.5). Mean scores for the three subscales of the UPDRS were 1.8 (S.D. 2.2) for scale 1 (cognition and mood, maximum score 16), 7.4 (S.D. 4.6) for subscale 2 (activities of daily living, maximum score 52), and 16.4 (S.D. 9.5) for subscale 3 (motor disability, maximum score 72), indicating very mild to mild motor impairment. Duration of the disease varied between 0.5 and 8 years, with a mean of 3.0 years (S.D. 2.6).

Depression was estimated using the Beck Depression Inventory (BDI, [5]). The mean depression score of the unmedicated Parkinson's disease group was 13.6 (S.D. 9.3) with individual scores ranging from 0 to 30. Eight of the people with unmedicated Parkinson's disease were classified as depressed according to the criteria of the BDI.

The mean age of the participants in the medicated Parkinson's disease group ($n = 20$; 11 female, 9 male) was

56.9 years (S.D. 9.9 years) and mean duration of schooling was 9.0 years (S.D. 1.3 years). Their average IQ was 103.2 (S.D. 10.6 years).

People with medicated Parkinson's disease ranged between 1 and 4 on the Hoehn and Yahr scale, with a mean of 2.6 (S.D. 0.9). Mean scores for the three subscales of the UPDRS were 3.1 (S.D. 2.2) for scale 1 (cognition and mood, maximum score 16), 17.2 (S.D. 6.9) for subscale 2 (activities of daily living, maximum score 52), and 30.0 (S.D. 15.8) for subscale 3 (motor disability, maximum score 72), indicating mild to moderate motor impairment. Duration of the disease varied between 1 and 20 years, with a mean of 9.3 years (S.D. 4.6 years).

The mean depression score of the medicated Parkinson's disease group was 11.9 (S.D. 7.7) with individual scores ranging from 0 to 28. Eight of the people with medicated Parkinson's disease were classified as depressed according to the criteria of the BDI.

Statistical analyses showed that the medicated and unmedicated groups differed significantly from each other in terms of disease severity as measured by the three subscales UPDRS (cognition and mood: $t = 1.99$, $P = 0.05$; activities of daily living: $t = 4.82$, $P < 0.001$; motor disability: $t = 3.03$, $P < 0.01$) and the Hoehn and Yahr scale ($t = 3.69$, $P < 0.01$) as well as duration of the disease ($t = 4.87$, $P < 0.001$). However, no significant differences on BDI scores could be found ($t = -0.61$, $P = 0.55$) between medicated and unmedicated Parkinson's disease.

The control group consisted of 40 healthy adults (22 female, 18 male) free of neurologic and psychiatric disorders. The mean age of the controls was 55.3 years (S.D. 7.7 years) and mean duration of schooling was 9.0 years (S.D. 1.6 years). Mean IQ of the control group was 105.7 (S.D. 9.7).

Analyses of variances showed no significant differences between controls and Parkinson's disease groups with respect to age ($F(2, 73) = 0.25$, $P = 0.77$), years of formal education ($F(2, 73) = 1.76$, $P = 0.18$), or intelligence ($F(2, 73) = 2.23$, $P = 0.11$).

Basic visual processing was assessed using the Vistech VCTS 6000 contrast sensitivity chart. This measures the degree of contrast at which the orientations of stationary sinusoidal gratings can be detected at each of five spatial frequencies (1.5, 3.0, 6.0, 12.0, 18.0 cycles per degree). No significant differences were found between the unmedicated Parkinson's disease group and controls. Visual contrast sensitivity was found to be impaired in the group of medicated people with Parkinson's disease compared to controls at 6 cycles per degree ($t = 2.76$, $P < 0.01$) and 12 cycles per degree ($t = 2.11$, $P < 0.05$). A comparison between people with medicated and people without medicated Parkinson's disease showed impairments for the medicated Parkinson's disease group at 12 cycles per degree ($t = 2.28$, $P < 0.05$) and 18 cycles per degree ($t = 2.53$, $P < 0.05$).

To establish that people with Parkinson's disease were able to understand the meanings of verbal emotion terms used for responses, all participants were tested for their

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