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# The recognition of facial emotion expressions in Parkinson's disease

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**Abstract**

A limited number of studies in Parkinson's Disease (PD) suggest a disturbance of recognition of facial emotion expressions. In particular, disgust recognition impairment has been reported in unmedicated and medicated PD patients. However, the results are rather inconclusive in the definition of the degree and the selectivity of emotion recognition impairment, and an associated impairment of almost all basic facial emotions in PD is also described. Few studies have investigated the relationship with neuropsychiatric and neuropsychological symptoms with mainly negative results. This inconsistency may be due to many different problems, such as emotion assessment, perception deficit, cognitive impairment, behavioral symptoms, illness severity and antiparkinsonian therapy. Here we review the clinical characteristics and neural structures involved in the recognition of specific facial emotion expressions, and the plausible role of dopamine transmission and dopamine replacement therapy in these processes. It is clear that future studies should be directed to clarify all these issues.


## 1. Introduction

Parkinson's disease (PD) is a neurodegenerative disorder clinically characterized by motor symptoms, such as bradykinesia, rigidity, and rest tremor, which usually arise unilaterally, and spread to the opposite side of the body and the axial muscles with disease progression. These symptoms depend mostly on the degeneration of the dopamine-containing neurons in the substantia nigra pars compacta in the mesencephalon (Hughes et al., 1992), where cytoplasmic inclusions of ubiquinated proteins, the Lewy bodies, have been identified. Recent neuropathological data, however, indicate that the Lewy bodies can be found in a number of brainstem structures, including the olfactory nuclei, the dorsal motor nucleus of the vagus, the locus coeruleus, the raphe nuclei, and the reticular formation, and in several cortical regions (Braak et al., 2003, 2004). Therefore, the original description

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of PD as due to the selective damage of the dopaminergic neurons in the mesencephalon should be updated into the concept of a more severe and multisystemic neurodegenerative disorder, whose clinical symptoms reflect the progression of the pathological damage from the medulla oblongata to neocortical areas. Thus, PD patients frequently experience non-motor symptoms, including autonomic, behavioral, and cognitive dysfunctions, and one of the most intriguing aspects of the studies by Braak et al. (2003, 2004) is that neuropathological alterations outside the substantia nigra are soundly correlated with some of these non-motor symptoms of PD.

Depression is a neuropsychiatric symptom frequently observed in PD patients, occurring in up to 45% of cases (Burns, 2002). Depression in PD is not necessarily related to the severity of motor impairment, and it is often misdiagnosed since hypomimia and reduction of voluntary movements are common to both PD and functional depression. Therefore, the identification of depression in PD patients is essentially based upon subjective perception of depressive symptoms, such as the feeling of incapability, the reduced reaction to emotional stimuli, and the impairment to experience pleasure from things or events (anhedonia). Also, cognitive impairment is frequent in PD and is usually characterized by 'frontal' cortical dysfunction, including visual–perceptual and executive deficits, whereas episodic memory and verbal fluency are generally spared (Levin and Katzen, 1995). However, in the late stages of PD, severe cognitive impairment may be observed, giving rise to the PD-dementia complex. Because of the frequent involvement of these behavioral and cognitive manifestations, in recent years interest is growing on the characterization of the possible impairment of the processing of facial emotion expressions in PD. In an evolutionary perspective, emotions play a significant role for the adaptation of a subject and for the establishment of relationships within a community. Moreover, emotions have a regulatory function on the mechanisms of motivation, on promoting transgeneration relationships, and on modulating individual behavior as to increase the chances of survival. Thus, this aspect is of more than a theoretical importance.

Patients suffering from PD may have alteration in areas strategic for the identification of specific facial expressions, such as the insula, the amygdala, the ventral striatum, the inferior orbitofrontal cortex, and the anterior cingulate cortex.

## 2. Method

With the aim to review all pertinent papers in the field of facial emotion expressions recognition and its correlation with neuropsychiatric and neuropsychological data, a detailed search of the literature was conducted. For our purposes, the database was selected using PubMed Services utilizing the following key words: Parkinson's disease, facial expression, emotion recognition, cognitive deficits, depression, anxiety. We also hand-searched relevant journals. In addition, the bibliographies of all important articles were searched for further publications.

The articles were restricted to English language and spanned the period from January 1989 to December 2007. We chose papers reporting sign-based definitions relevant empirical neurological and neuropsychological data, and results of clinical trials. Historically remarkable or conceptually related articles were included as well. All articles cited in this manuscript were judged by F.E.P. and G.S. to be relevant and to meet the scientific and conceptual criteria listed.

## 3. Impaired facial emotion recognition in PD patients

A total of 14 studies assessed the topic of facial emotion recognition in PD patients from 1989 to 2007. Eleven studies (among these 2 investigated pre-operative Deep Brain Stimulation (DBS) PD patients) reported data on individual facial emotion expressions with the aim of analyze recognition impairment. Three studies analyzed this topic on patients with DBS of the subthalamic nuclei. Two further studies reported ancillary data on particular subpopulations of patients or using alternative methods. Table 1 summarizes results of these studies.

Notably, the recognition of facial emotion expressions was evaluated using different methods in different studies reviewed herein (Table 2).

The first study assessing this topic was that of Blonder et al. (1989), who suggested that PD patients suffer from a selective impairment in facial emotion recognition. The results of this first study, indicating an impairment of facial emotion expressions recognition in PD patients, have been replicated by the vast majority of the subsequent studies. However, partial discordance exists on the type of emotions involved. Indeed, the study by Kan et al. (2002) showed a significant impairment in the recognition of facial expression of fear and disgust. Globally, fear was the emotion least recognized by both PD and controls. Instead, Yip et al. (2003) described a compromising recognition in all facial emotion, in particular fear and sadness, in 56 PD patients with bilateral motor symptoms while 8 right-sided PD patients were particularly impaired in the recognition of sadness and disgust. Similarly, Sprengelmeyer et al. (2003), demonstrated the presence of a wide impairment in the recognition of facial emotion expressions in PD patients. Indeed, fear, sadness, anger, and disgust recognition were all compromised. A sub-analysis showed that unmedicated PD patients showed a specific deficit in the recognition of disgust compared to medicated PD. Thus, authors suggested that dopaminergic treatment of PD may improve disgust recognition which should be considered the core facial emotion recognition impairment in PD. Dujardin et al. (2004a) noticed an impairment in PD in the recognition of all investigated facial emotions (i.e. disgust, anger, and sadness). Post-hoc analyses showed that higher intensity of emotional facial expressions did not help PD patients in the recognition. On the contrary, healthy controls were facilitated in facial emotion recognition by the higher emotional expression intensity. Interestingly, the same authors (Dujardin et al., 2004b) described no impairment in the recognition of facial emotion expressions in 12 PD patients in the pre-operative phase of DBS of the subthalamic nuclei in comparison with 12 healthy controls. Differently, Suzuki et al. (2006a), showed a selective impairment in the recognition of disgust in PD patients. Finally, Lawrence et al. (2007), reported a selective deficit in the recognition of anger facial emotion in PD patients.

On the contrary, Adolphs et al. (1998) firstly indicated that 18 patients with PD were not impaired in the recognition