Disgust-specific impairment of facial expression recognition in Parkinson’s disease

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There is contradictory evidence regarding whether the impairments of the recognition of emotional facial expressions in Parkinson’s disease are specific to certain emotions such as disgust and fear. Generally, neurological case reports on emotion-specific impairments have been suspected of being confounded with the factor of task difficulty. Using a refined assessment method in which the difficulty factors were controlled by means of mixed facial expressions and item response theory, we attempted to clarify whether Parkinson’s disease disproportionately impaired the recognition of specific emotions. We studied 14 patients with Parkinson’s disease and 39 healthy controls who were matched in terms of gender, age, years of education and intelligence quotient. Whereas the refined method revealed that the patients with Parkinson’s disease displayed significantly lower scores in disgust recognition alone, conventional methods failed to detect this impairment. In addition, control measures including face recognition abilities did not statistically explain the impairment observed in the patients. The results indicate that Parkinson’s disease can indeed selectively impair the recognition of facial expressions of disgust; this provides concrete evidence for emotion-specific impairments that sufficiently withstands criticisms regarding the difficulty artefacts. Furthermore, the results support the proposed role of the basal ganglia–insula system in disgust recognition. This study effectively demonstrates the benefits of refining neuropsychological assessment by taking advantage of the modern psychometric theory.

Keywords: Parkinson’s disease; facial expression recognition; emotion-specific impairment; disgust; item response theory

Abbreviations: GRM = graded-response model; IRT = item response theory; SDS = Self-report Depression Scale


Introduction

Parkinson’s disease, typically considered a movement disorder, causes a range of mild cognitive deficits, and there is growing interest in its role in impaired emotional and social behaviours (Benke et al., 1998; Saltzman et al., 2000; Pell and Leonard, 2003; Brand et al., 2004). Among other things, impairments of the recognition of emotional facial expressions have been previously noted in Parkinson’s disease patients (Scott et al., 1984; Beatty et al., 1989). Given the recent emphasis on the role of dissociable neural substrates in the recognition of their corresponding emotions (Calder et al., 2001; Adolphs, 2002; Posamentier and Abdi, 2003), the most intriguing, yet debatable, issue is whether or not Parkinson’s disease disproportionately impairs the recognition of facial expressions of specific emotion(s) (Kan et al., 2002; Sprengelmeyer et al., 2003; Pell and Leonard, 2005). In addition to those on Parkinson’s disease, neurological reports on emotion-specific impairments in general have always been suspect owing to difficulty artefacts (Rapcsak et al., 2000). Therefore, this paper aims to help resolve this controversial issue by taking advantage of our recently developed and elaborate assessment method for facial expression recognition (Suzuki et al., 2005).

At present, it appears that substantial evidence indicating the role of dissociable neural substrates in the recognition of facial expressions of fear and disgust has been accumulated (Calder et al., 2001; Adolphs, 2002; Posamentier and Abdi, 2003). Since Adolphs et al. (1994) demonstrated a disproportionate impairment of the recognition of fear in a patient with selective amygdala damage, a number of neurological (Calder et al., 1996; Broks et al., 1998; Adolphs et al., 1999; Sato et al., 2002) and functional imaging studies (Morris et al., 1996, 2001; Whalen et al., 1998) have replicated the inextricable
link between the amygdala and fear recognition. Consequently, there is a growing emphasis on the role of the amygdala in fear recognition (Calder et al., 2001), although some exceptions (Hamann et al., 1996; Siebert et al., 2003) are yet to be explained.

A disproportionate impairment of disgust recognition was first observed in patients with Huntington’s disease, a hereditary neurodegenerative disorder associated with pathological changes in the basal ganglia. Sprengelmeyer et al. (1997b) reported that Huntington’s disease patients without general cognitive disturbances were impaired in the recognition of disgust and, to a lesser extent, fear. A more selective impairment of disgust recognition was later observed in pre-symptomatic Huntington’s disease gene carriers (Gray et al., 1997). Functional imaging research (Phillips et al., 1997; Sprengelmeyer et al., 1998; but see also Winston et al., 2003) revealed the activation of the insula as well as that of the basal ganglia in response to facial expressions of disgust. The proposed contributions of the basal ganglia and the insula to disgust recognition were recently supported by a single case report (Calder et al., 2000) that revealed a disproportionate impairment in the recognition of disgust after damage to these neural substrates.

The pathological hallmark of Parkinson’s disease is the loss of dopamine neurones in the substantia nigra, resulting in the depletion of dopamine in the striatum (caudate and putamen) of the basal ganglia. Recent evidence also suggests that the limbic system, including the amygdala, may not function normally in Parkinson’s disease patients (Braak et al., 1994, 1996; Harding et al., 2002; Saito et al., 2003). Cumulatively, the pathology of this disease predicts disproportionate impairments of disgust recognition and, perhaps to a lesser extent, fear recognition. However, so far, the results have been rather mixed, ranging from non-specific, broad impairments (Scott et al., 1984; Beatty et al., 1989; Blonder et al., 1989; Jacobs et al., 1995; Breitenstein et al., 1998; Dujardin et al., 2004) to an entirely intact performance (Dewick et al., 1991; Madeley et al., 1995; Adolphs et al., 1997; Pell and Leonard, 2005). We (Kan et al., 2002) reported the most selective deficit, namely, that Parkinson’s disease patients had impairments of the recognition of disgust and fear in the case of both still and moving images of facial expressions. Furthermore, Sprengelmeyer et al. (2003) investigated the effect of dopamine medication and observed a lower recognition of disgust and anger among unmedicated patients than among medicated patients. They also observed that fear recognition among both medicated and unmedicated patients was significantly worse than that among the healthy controls.

At present, it is argued that the apparent inconsistency in the results may reflect factors such as disease duration and/or severity (Breitenstein et al., 1998), and medication status (Sprengelmeyer et al., 2003; Dujardin et al., 2004). However, none of these factors alone may be sufficient to explain the inconsistency in the results (Pell and Leonard, 2005). In this paper, we focus on the problems related to measurement, which are likely to be responsible for this inconsistency. That is, the earlier methods used in the assessment of facial expression recognition share two major problems: (i) the ceiling effect and (ii) the differential difficulty levels across emotions (Suzuki et al., 2005). The first problem relates to the preferential use of prototypical, intense facial expression stimuli in earlier studies (Dewick et al., 1991; Madeley et al., 1995; Adolphs et al., 1998; Pell and Leonard, 2005), which frequently resulted in a ceiling effect. Consequently, Dewick et al. (1991) concluded that owing to this ceiling effect, their method did not appear to be sensitive enough to detect mildly impaired facial expression recognition in Parkinson’s disease patients.

The second problem is more critical and is prevalent in neurological research in general: sceptics have argued that the apparently disproportionate impairments of the recognition of specific emotions may be mere artefacts that simply reflect the differential difficulty levels across emotions (Rapcsak et al., 2000; Milders et al., 2003). It is a well-documented fact that even among the healthy population, facial expressions of fear are the most difficult to recognize (Russell, 1994; Biehl et al., 1997). Thus, it is possible that owing to its marked difficulty, the recognition of fear may be vulnerable to general impairments after brain damage. Indeed, there are reports that various brain lesions other than to the amygdala also serve to disproportionately impair the recognition of fear (Adolphs et al., 1996; Rapcsak et al., 2000; Milders et al., 2003).

The criticism described above is directly applicable to the findings regarding Parkinson’s disease. Thus, Sprengelmeyer et al. (2003) speculated that the deficit in fear recognition observed in Parkinson’s disease patients may be influenced by the difficulty factors. However, their data could not exclude the possibility that the impairment of fear recognition could be attributed to pathological changes in the amygdala. Along with other researchers (Tessitore et al., 2002), we (Yoshimura et al., 2005) supported the above view by demonstrating that Parkinson’s disease patients lacked the amygdala activation observed among healthy controls when viewing facial expressions of fear.

In brief, the existing methods for the assessment of facial expression recognition can (i) readily detect a fear recognition deficit because of its marked difficulty and (ii) cannot distinguish between the two conflicting interpretations of emotion-specific impairments and difficulty artefacts. As in the case of Parkinson’s disease, comorbid impairments of fear and other emotions further complicate the issue by obscuring the relevant impairment that is specifically related to the pathological changes under consideration.

In order to address these two problems, namely, the ceiling effect and the differential difficulty levels, we recently developed a new method for the assessment of facial expression recognition by taking advantage of morphing and item response theory (IRT) (Suzuki et al., 2005). Morphing refers to the technique of synthesizing an interpolated computer image from multiple images, which we used to create mixed, ambiguous facial expressions from prototypical facial expressions. Specifically, we utilized facial expression...
megamix (Young et al., 1997), that is, a set of mixed facial expression stimuli constructed from all the possible paired combinations of the six basic emotions (i.e. happiness, surprise, fear, anger, disgust and sadness). As compared with previous studies in which morphing was limited to combinations between two closely related basic emotions (facial expression hexagon; Calder et al., 1996; Sprengelmeyer et al., 1997b, 2003), facial expression megamix yields many stimuli with various difficulty levels and helps in rectifying the two problems.

In addition, we have based the scoring method on a state-of-the-art psychometric technique known as IRT (Baker, 1992; Embretson and Reise, 2000), in which the participants’ performances are scored according to both their responses and the stimulus properties. Although, at present, IRT is primarily used in educational testing and personality measurement, there is a growing interest in its applicability to neuropsychological assessment (Mungas et al., 2000; Yonekura et al., 2005). In IRT, the stimulus properties that constitute the difficulty levels are first estimated from a raw dataset of participants’ responses using a statistical procedure known as marginal maximum-likelihood estimation (Bock and Aitkin, 1981). The participants’ performances are then scored taking into account the estimated stimulus properties. Thus, in contrast with simple counting/summing that disregards the differences in the stimulus properties, IRT scoring will greatly contribute to the cancellation of the differential difficulty levels across stimuli (emotions).

Figure 1 illustrates one of the main findings of a normative study in which our method was used (n = 421; Suzuki et al., 2005). It presents the distributions of the performance scores (referred to as sensitivity scores) for happiness and fear recognition. In previous methods, the scores for the recognition of happiness have commonly exhibited a ceiling effect owing to the ease of the recognition, and those for the recognition of fear have displayed a positively skewed distribution owing to the difficulty of the recognition. In Fig. 1, however, both the score distributions are roughly symmetrical and contain substantial variations; this indicates the resolution of the two problems. In this paper, we use this refined method to provide an unbiased description of facial expression recognition in Parkinson’s disease patients and thereby contribute to the resolution of the long-standing controversy over emotion-specific impairments.

### Methods

#### Participants

We studied 14 patients with idiopathic Parkinson’s disease (5 men, 9 women; aged 57–77 years) who were recruited from among outpatients who were diagnosed and regularly treated at the Showa University Hospital. The recruitment criterion was the patient’s competency in understanding the nature of this study and being able to provide informed consent on his/her own behalf; an experienced neurologist (M.K.) and a tester (A.S.) were the judges for ascertaining which patients met this criterion. This procedure resulted in the recruitment of early Parkinson’s disease patients without a particular recognition problem; the severity of their disease corresponded to Hoehn and Yahr stages I–III (I: 7; II: 6; III: 1; Hoehn and Yahr, 1967). All the patients were being treated with levodopa or a dopamine agonist medication. The patients were compared with 39 healthy controls (18 men, 21 women; aged 58–78 years) who reported no history of neurological or psychiatric diseases.

The background data for the two groups of participants are summarized in Table 1. The two groups were matched with respect to...
gender ratio \(\chi^2(1) = 0.457, P = 0.499\), age \(t(51) = -0.306, P = 0.761\), years of education \(t(51) = -1.470, P = 0.148\), and intelligence quotient (IQ), as estimated by the revised Wechsler Adult Intelligence Scale subscales of Information and Picture Completion \(t(51) = -0.943, P = 0.350\). In addition, the scores on the Mini-Mental State Examination (MMSE; Folstein et al., 1975) were equally high in both groups [Patients, range 24–55; Controls, range 26–30; \(t(51) = 0.172, P = 0.864\)], indicating no signs of clinical dementia. Finally, the two groups did not display any significant difference with regard to depression, as measured by a modified version of the Self-rating Depression Scale (SDS; Zung et al., 1965), from which one item concerning suicidal ideation was removed for ethical reasons [Patients, range 20–55; Controls, range 20–49; \(t(51) = 0.457, P = 0.656\)], and by the participant’s sensitivity to the relevant emotion (\(\theta_i\)). According to the model, the probability \(P_{ij}\) that the participant will select intensity points higher than or equal to \(j\) is expressed as follows:

\[
P_{ij}^* = \frac{1}{1 + \exp[-1.7\alpha_i(\theta_i - \beta_j)]}
\]

The above equation reflects the assumption that the probability of choosing higher intensity points increases monotonically with \(\theta_i\). Figure 2 illustrates this equation, wherein the horizontal axis represents \(\theta_i\), and the vertical axis represents \(P_{ij}^*\).

The item parameters represent the stimulus properties constituting the difficulty and determine the exact shapes of the \(P_{ij}^*\) curves. For example, larger \(\beta_j\) parameters shift \(P_{ij}^*\) curves to the right (as seen in Fig. 2A and B) and lower the probability of choosing higher intensity points; this indicates an increasing difficulty. In IRT, the item parameters are first estimated from a raw dataset of ratings, and each participant’s sensitivity is then scored on the basis of the estimated item parameters. Thus, IRT-based scoring is free from difficulty artefacts.

In this study, the participants’ sensitivity to particular emotions were scored using the estimates of item parameters obtained in our normative research (Suzuki et al., 2005; unpublished work). To illustrate this, Fig. 3 presents concrete examples of the \(P_{ij}^*\) curves based on the intensity ratings of happiness for a 60% happiness and 40% disgust morph (easy stimulus) and for a

![Graphical representations of the functional relationship between \(P_{ij}^*\) and \(\theta_i\).](http://brain.oxfordjournals.org/)

(A) \(\alpha_i = 1.5, \beta_{11} = 2.0, \beta_2 = 0.9, \beta_3 = 0, \beta_4 = -1.2, \beta_5 = -2.0\). (B) \(\alpha_i = 1.5, \beta_{11} = 3.5, \beta_2 = 2.4, \beta_3 = 1.5, \beta_4 = 0.3, \beta_5 = -0.5\).
Facial expression hexagon

For the purpose of comparison with the IRT method, we also scored the data obtained in assessing ‘sensitivities to basic emotions’ in an alternative manner, namely, by simulating a conventional method utilizing the facial expression hexagon (Calder et al., 1996). As described earlier, the facial expression hexagon is a sequence of morphed facial expressions that combine two closely related emotions; specifically, the sequence runs happiness–surprise–fear–sadness–disgust–anger–happiness. Calder et al. (1996) devised a forced-choice identification task of morphed facial expressions belonging to the hexagon; this task has been applied to a variety of neuropsychiatric populations (Calder et al., 2000, 2004; Sprengelmeyer et al., 1997a, b, 1999, 2003).

We simulated the method of Calder et al. (1996) as follows. Of the 72 photographs that were used in the rating task in this study, 36 belonged to the facial expression hexagon. With respect to these 36 photographs, we dichotomized the intensity rating responses as ‘correct’ when the maximal rating was made on the dominant emotion (i.e. 100% or 60% morphed emotion) and as ‘incorrect’ otherwise. This scoring method yielded a maximum score of 6 for each emotion because there were two prototypes (i.e. 100% morphs) and four 60% morphs for any given emotion.

Control tasks for face recognition

Parkinson’s disease is known to interfere with visual recognition (Cummings and Huber, 1992), and general deficits in face recognition, in particular, can partially account for the impairments of facial expression recognition in Parkinson’s disease patients (Beatty et al., 1989). Therefore, we included two control tasks in order to measure face recognition abilities in general.

Facial identity matching

The task is designed to be a Japanese version of the Benton Test of Face Recognition (Benton and Van Allen, 1968). The participants were asked to match a facial photograph (target) to an array of six facial photographs (references) of different persons of the same gender. The photographs used in this task were selected from the Facial Information Norm Database (FIND; Yoshida et al., 2004) distributed by Nihon University. The matching involved three conditions (i.e. a front-view target with front-view references, a front-view target with side-view references, and an upward target with front-view references); each condition comprised 10 trials. The 30 pairs consisting of a target and references were presented to the participants in either of two pseudo-randomized orders, and the orders assigned were counterbalanced across participants. The total number of correct responses was scored across all the three conditions.

Gender intensity rating

We used a task named ‘Gender Intensity Rating’ as another test of face recognition. The participants were asked to rate a facial photograph with respect to the intensity of gender (male and female) on a 6-point scale. This procedure was similar to the task in which the ‘sensitivities to basic emotions in faces’ were evaluated. A total of 16 photographs were used as stimuli: 4 male faces and 4 female faces were selected from among Japanese and Caucasian neutral faces (JACNeuF; Matsumoto and Ekman, 1988), and 8 male–female morphed faces were synthesized from them. The order in which the 16 photographs were presented was pseudo-randomized but
was identical for all the participants. The average of the intensity ratings for each gender was scored.

Results

Control tasks for face recognition

Table 1 presents the means and standard errors of the scores in the control tasks that measured face recognition abilities. Although the scores were consistently lower in the Parkinson’s disease patients, the difference was not statistically significant [Facial Identity Matching: $t(51) = -0.458, P = 0.649$; Gender Intensity Rating: Male, $t(51) = -1.058, P = 0.295$; Gender Intensity Rating: Female, $t(51) = 0.886, P = 0.380$]. The results suggest that face recognition per se was relatively unaffected among the Parkinson’s disease patients in our sample.

Sensitivities to basic emotions

Figure 4 presents the means and standard errors of the estimated sensitivity scores for the five basic emotions among the two groups of participants. The degree to which the Parkinson’s disease patients displayed lower sensitivity scores than the healthy controls for corresponding emotions varied, and a marked difference was observed with respect to the emotion of disgust. A multivariate analysis of variance was conducted on the sensitivity scores; this revealed a significant effect of groups [$\text{Pillai–Bartlett’s } V = 0.264$, Wilks’ $\Lambda = 0.736$, $F(5,47) = 3.368, P = 0.011$]. Post hoc comparisons ($t$-tests) confirmed a significant decrease in the sensitivity scores for disgust among the Parkinson’s disease patients [$t(51) = 3.408, P = 0.001$]. None of the other comparisons reached statistical significance ($P > 0.2$).

We further examined the decreased sensitivity scores for disgust among the Parkinson’s disease patients in relation to the background data and face recognition abilities. Table 1 presents the correlation coefficients between the sensitivity scores for disgust and the other variables. Certain variables, such as the years of education [$r = 0.279, t(51) = 2.075, P = 0.043$] and the male intensity rating [$r = 0.270, t(51) = 2.003, P = 0.051$], displayed significant or marginally significant correlations. However, an analysis of covariance revealed that a covariation of the two variables could not account for the significant decrease in the sensitivity scores for disgust among the Parkinson’s disease patients [$F(1,49) = 7.631, P = 0.008$].

Conventional measures of facial expression recognition

Figure 5 allows for a comparison of the methods by presenting the means and standard errors of the scores from the two conventional methods: Facial Expression Identification (Fig. 5A) and Facial Expression Hexagon (Fig. 5B). The figure reveals that the performance of the two groups was almost identical, although the largest difference was still observed in the case of the emotion of disgust. A multivariate analysis of variance that was conducted on the scores from each method revealed an insignificant effect of groups [Facial Expression Identification: $\text{Pillai–Bartlett’s } V = 0.095$, Wilks’ $\Lambda = 0.905$, $F(6,46) = 0.809, P = 0.568$; Facial Expression Hexagon: Pillai–Bartlett’s $V = 0.106$, Wilks’ $\Lambda = 0.894$, $F(6,46) = 0.908, P = 0.497$]. No post hoc comparison ($t$-test), including that of the scores for disgust, reached statistical significance ($P > 0.15$).
Impaired disgust recognition in Parkinson’s disease

The aim of the current study was to clarify the exact nature of the impairments of facial expression recognition among patients with Parkinson’s disease by using a refined assessment method, that is, by evaluating the ‘sensitivities to basic emotions in faces’ (Suzuki et al., 2005). This method has two significant advantages: (i) it eliminates the ceiling effect and is sensitive enough to detect mild impairments, and (ii) it effectively controls the differential difficulty levels across emotions and is appropriate for the examination of emotion-specific impairments. These advantages enabled us to clearly demonstrate that the Parkinson’s disease patients were selectively impaired in the recognition of facial expressions of disgust; that is, the sensitivity scores for disgust were significantly lower among the Parkinson’s disease patients than among the healthy controls, whereas the scores for happiness, fear, anger, and sadness were not. In contrast, the two conventional methods of Facial Expression Identification and Facial Expression Hexagon did not reveal any difference between the patients and controls, although the difference appeared to be the largest in the case of the emotion of disgust.

Earlier studies were consistent with the current findings and revealed an impairment of disgust recognition in Parkinson’s disease patients (Kan et al., 2002; Sprengelmeyer et al., 2003); however, comorbid impairments in the recognition of other emotions, particularly fear, were always observed. The comorbidity of the impairments of disgust and fear recognition is also prevalent among Huntington’s disease patients (Sprengelmeyer et al., 1996, 1997b; Milders et al., 2003; Wang et al., 2003). Given the claims that a fear recognition deficit can often be attributed to its marked difficulty (Rapcsak et al., 2000; Milders et al., 2003), our success in revealing a disgust-specific impairment may be partially owing to the fact that the difficulty levels across emotions were well controlled in our assessment method [advantage (2)]. However, this may not be the only factor since we did not observe any impairment of fear recognition in the Parkinson’s disease patients even when using the conventional methods, wherein the difficulty levels were not controlled.

Furthermore, we believe that the excellent sensitivity of our method [advantage (1)] enabled us to detect a mild impairment of disgust recognition in ‘high-functioning’ Parkinson’s disease patients who were in the early stages of the disease. In this study, the mental abilities of the Parkinson’s disease patients were comparable with those of the healthy controls in terms of a relatively high IQ, a rare incidence of depression and spared face recognition. Their well-preserved mental abilities may indicate that the Parkinson’s disease patients are in the early stages of their disease and/or currently respond well to dopamine medication. It is possible that impairments of facial expression recognition may be mild and specific to disgust in such early-stage Parkinson’s disease patients with well-preserved mental abilities; the assessment methods similar to those used in previous studies such as Facial Expression Identification (Dewick et al., 1991) and Facial Expression Hexagon (Sprengelmeyer et al., 2003) may have failed to detect such mild impairments owing to their low sensitivity. Parkinson’s disease patients suffering from other ailments such as depression and disturbed vision (Sprengelmeyer et al., 2003) may be likely to suffer from deficits in other emotions such as fear. In particular, in the case of patients with depression, Parkinson’s disease is observed to be associated with amygdala dysfunction (Remy et al., 2005), which we will discuss further in the next section. Nonetheless, from the current findings, it can be deduced that the impairment of disgust recognition is primarily associated with the early stages of Parkinson’s disease, and that it is dissociable from the impairment of fear recognition.

Beatty et al. (1989) demonstrated that impairments of facial expression recognition in Parkinson’s disease patients may partly reflect the deficits in face recognition in general. In addition, Adolphs et al. (1998) raised the possibility that the inadequate control of demographic factors in some studies may yield spurious results. These reservations are not applicable to the current findings because the Parkinson’s disease patients in our sample had relatively intact abilities with regard to face recognition, and they matched the healthy controls in terms of the demographic variables. Although we observed that the sensitivity scores for disgust had a significant correlation with the years of education and the male intensity rating, the intervening effects of these factors did not explain the lower sensitivity scores among the Parkinson’s disease patients. We believe that ultimately, it is unlikely that general factors can fully account for the disproportionate impairment of disgust recognition among Parkinson’s disease patients; this may require more specific explanations.

Neural and functional accounts

Given the well-known pathological changes that occur in Parkinson’s disease, the basal ganglia are plausible candidates for the neural substrate responsible for impaired disgust recognition (Kan et al., 2002; Sprengelmeyer et al., 2003). Several neuropsychiatric diseases that are commonly associated with the dysfunction of the basal ganglia, such as Huntington’s disease (Sprengelmeyer et al., 1997b), obsessive-compulsive disorder (Sprengelmeyer et al., 1997a), and Wilson’s disease (Wang et al., 2003), have also been known to cause a disproportionate impairment in disgust recognition. Meta-analyses of functional imaging studies lend further support to the above neurological findings by demonstrating the frequent activation of the basal ganglia in response to facial expressions of disgust (Phan et al., 2002; Murphy et al., 2003). Consequently, the latest examinations of neural systems for facial expression recognition postulate that the basal ganglia play a special role in disgust recognition.
expressions of disgust. Furthermore, Calder et al. (2003) presented novel evidence linking a part of the basal ganglia (the ventral striatum) with the recognition of anger and not disgust.

However, to date, there still exists a controversy regarding the relative contributions of the basal ganglia and the insula to impaired disgust recognition. Wang et al. (2003) suggested that in the case of Wilson’s disease, the degree of subcortical (i.e. basal ganglia) atrophy, and not cortical (i.e. insula) atrophy, was correlated with impaired facial expression recognition. Hennenlotter et al. (2004) also allowed for the possibility of the involvement of the basal ganglia because the activation of the putamen in the recognition of disgust, which was significant in the case of healthy controls, was absent in Huntington’s disease gene carriers. With regard to Parkinson’s disease, there has been little emphasis on pathological changes in the insula (but see Braak et al., 1996), with the inexplicable exception of the relative over-activation in the insula during motor (Hanakawa et al., 1999) and sensory (Boecker et al., 1999) tasks. However, this was usually interpreted as a compensatory mechanism rather than a dysfunction. Nevertheless, in Parkinson’s disease patients, the functional levels of the insula, indexed by the regional cerebral blood flow (Oishi et al., 2004) and dopamine D2 receptor availability (Kaasinen et al., 2004), are observed to be positively correlated with psychological traits such as performance IQ and novelty-seeking personality, respectively. Thus, a clarification of the roles of the basal ganglia and the insula in impaired disgust recognition will require further attempts at large-scale structural lesion mapping and functional imaging to be applied to the neuropsychiatric populations of interest. Given the extensive interconnections between the basal ganglia and the insula (Chikama et al., 1997; Fudge et al., 2005), at present, we can safely suggest that both neural substrates may be involved as a system in the recognition of disgust signals from facial expressions and, perhaps, from other modalities as well (Calder et al., 2000).

Another issue left unresolved by the current study concerns the impact of the amygdala pathology on facial expression recognition in Parkinson’s disease (Braak et al., 1994, 1996; Harding et al., 2002; Saito et al., 2003). The essential role of the amygdala in fear recognition is extensively supported by findings based on studies of lesions (Adolphs et al., 1994, 1999; Calder et al., 1996; Saito et al., 2002) as well as functional imaging studies (Morris et al., 1996, 2001; Whalen et al., 1998), and the absence of amygdala activation in response to facial expressions of fear was demonstrated in Parkinson’s disease patients (Tessitore et al., 2002; Yoshimura et al., 2005). These facts are apparently incompatible with our observation of intact fear recognition among the Parkinson’s disease patients. Given that both the above studies of activation failed to observe a fear recognition impairment among the Parkinson’s disease patients in their samples, the influence of the amygdala dysfunction on observable behaviours may be very subtle in Parkinson’s disease, especially when the patients are treated with dopamine medication. Functional imaging studies in the healthy population revealed that the amygdala...
activation in response to the negative emotional stimuli was potentiated (Hariri et al., 2002) and attenuated (Takahashi et al., 2005) by the administration of dopamine agonist and antagonist, respectively. Tessitore et al. (2002) further demonstrated that dopamine repletion partially restored the amygdala function in Parkinson’s disease patients. Thus, it is possible that dopamine medication may have influenced the current findings by mitigating the amygdala dysfunction and fear deficits associated with Parkinson’s disease.

In addition, pathological changes in the amygdala may not be clearly evident in our sample of Parkinson’s disease patients with well-preserved mental functions. Specifically, the rare incidence of depression in the current sample (measured by a modified version of SDS, M = 32.8) is noteworthy; this is in contrast with the relatively high incidence of depression in the sample studied by Kan et al. (2002) (measured by SDS, M = 53.9), in which impaired fear recognition was observed. Moreover, in this study, one Parkinson’s disease patient who was considered as depressed displayed lower sensitivity score for fear (−1.24), although the exclusion of the patient did not affect the overall results. Remy et al. (2005) recently provided evidence that depressed Parkinson’s disease patients suffered from a more severe loss of dopamine and noradrenaline innervation, specifically in the limbic system, including the amygdala, as compared with non-depressed patients. Cumulatively, a complete account of the influence of amygdala dysfunction on fear recognition in Parkinson’s disease will require further research investigating the possible modulatory role of dopamine medication and depression.

Finally, we would like to highlight the potential involvement of neural substrates other than the basal ganglia, the insula and the amygdala because Parkinson’s disease is known to affect various components of the limbic system such as the entorhinal region, the hippocampal formation, the thalamus and the anterior cingulate (Braak et al., 1996). It is further proposed that in Parkinson’s disease the pathology may initially occur in the extranigral regions such as the anterior olfactory nucleus and the lower brainstem (Braak et al., 2003), and that olfactory impairment and sleep disorder (Boeve et al., 2001; Stiasny-Kolster et al., 2005) may be the earliest preclinical symptoms of the disease (for review, see Abbott, 2005). Thus, it is interesting to determine whether or not impaired facial expression recognition also constitutes one of such preclinical symptoms of Parkinson’s disease, which may shed light on the unsolved contribution of the brainstem nuclei to emotional behaviours (Damasio et al., 2000).

Conclusions
This is the first neurological study that examines the emotion-specific impairments of facial expression recognition by using a refined assessment method that sufficiently withstands existing methodological criticism. Our data clearly reveal that Parkinson’s disease can selectively impair the recognition of emotional expressions, thereby providing concrete evidence for emotion-specific impairment that is not confirmed with difficulty artefacts. The findings successfully demonstrate the benefits of applying an elaborate assessment method that incorporates modern psychometric theory into neuropsychological testing, such as that presented in this study.

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