PAPER

# Taste responses in patients with Parkinson's disease

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**Objective:** Preclinical studies indicate that dopaminergic transmission in the basal ganglia may be involved in processing of both pleasant and unpleasant stimuli. Given this, the aim of the present study was to assess taste responses to sweet, bitter, sour, and salty substances in patients with Parkinson's disease (PD).
**Methods:** Rated intensity and pleasantness of filter paper discs soaked in sucrose (10–60%), quinine (0.025–0.5%), citric acid (0.25–4.0%), or sodium chloride (1.25–20%) solutions was evaluated in 30 patients with PD and in 33 healthy controls. Paper discs soaked in deionised water served as control stimuli. In addition, reactivity to 100 ml samples of chocolate and vanilla milk was assessed in both groups. Taste detection thresholds were assessed by means of electrogustometry. Sociodemographic and neuropsychiatric data, including cigarette smoking, alcohol consumption, tea and coffee drinking, depressive symptoms, and cognitive functioning were collected.
**Results:** In general, perceived intensity, pleasantness, and identification of the sucrose, quinine, citric acid, or sodium chloride samples did not differ between the PD patients and controls. Intensity ratings of the filter papers soaked in 0.025% quinine were significantly higher in the PD patients compared with the control group. No inter-group differences were found in taste responses to chocolate and vanilla milk. Electrogustometric thresholds were significantly ($p = 0.001$) more sensitive in the PD patients.
**Conclusions:** PD is not associated with any major alterations in responses to pleasant or unpleasant taste stimuli. Patients with PD may present enhanced taste acuity in terms of electrogustometric threshold.

According to the dopamine hypothesis of reward, midbrain dopaminergic neurones constitute a critical part of the brain reward system and mediate the rewarding effects of food, water, sex, and drugs of abuse.[1–5] In its simplest form, the dopamine hypothesis of reward postulates that the rewarding properties of any stimulus are a direct consequence of dopamine release evoked by this stimulus in the striatum.[2 4 5] In addition, the theory states that dopamine deficits may produce anhedonia, a condition defined as a decreased experience of pleasure after presentation of natural or "chemical" rewards.[2 5–7]

The dopamine hypothesis of reward prompted some researchers to speculate that patients with idiopathic Parkinson's disease (PD) may show symptoms of anhedonia.[2 8] It has been found repeatedly that PD leads to progressive atrophy of both nigrostriatal and mesolimbic dopaminergic pathways.[9–11] However, experimental evidence linking PD with anhedonia comes from a single study on the rewarding properties of a dopaminergic psychostimulant, methylphenidate. The drug tended to produce weaker rewarding effects in PD subjects compared with healthy controls.[12] Surprisingly little is known about acute reactivity of PD patients to other chemical or natural rewards. Sweet taste and pleasant odours are considered phylogenetically old natural rewards and their hedonic evaluation is regarded as the indicator of the reward system function.[13–16] However, any study on hedonic responses to olfactory stimuli in PD could be complicated by the fact that PD patients present severe deficits in odour detection, discrimination, and identification.[17–21] Taste function in PD has not been assessed in detail as yet. Given these factors, we decided to evaluate taste responses (rated intensity, pleasantness, and identification) in PD patients and in age matched controls. Both groups rated the intensity and pleasantness of filter paper discs soaked in sucrose (sweet), quinine (bitter), citric acid (sour), and sodium chloride (salty) solutions. The subjects were also exposed to 100 ml samples of chocolate and vanilla milk. In order to evaluate perithreshold taste sensitivity, detection thresholds were assessed by means of electrogustometry.[22 23]

## METHODS

### Participants

Patients with idiopathic PD, stages I–III according to Hoehn and Yahr,[24] were taken from a local registry established in the Institute of Psychiatry and Neurology for the Warsaw district. A consecutive series of 35 subjects was invited to participate. All subjects were ambulatory and living with their families. In each case, the diagnosis was confirmed by two neurologists from the study team (WK, HS-J). One patient refused to take part in the study, one was excluded because of cognitive impairment, and three were excluded after neurological examination. Thus, a group of 30 patients (17 males, 13 post-menopausal females) was finally recruited for the study. Twenty six of those patients were being treated with L-dopa, 11 with selegiline, two with amantadine, and one with biperiden. The four subjects who were not treated with L-dopa received selegiline ($n = 2$), amantadine ($n = 1$), or selegiline and amantadine in combination ($n = 1$). Patients with clinical fluctuations were examined in the "on" state. Baseline characteristics of the PD group are shown in table 1. A control group was recruited through all institutions involved in the study from families of staff members. Thirty three controls (20 males, 13 post-menopausal females) were selected from a group of 34 subjects. One potential participant was excluded after neurological examination.

**Abbreviations:** ANOVA, analysis of variance; AUDIT, Alcohol Use Disorder Identification Test; BDI, Beck Depression Inventory; L-dopa, L-3,4-dihydroxyphenylalanine; MMSE, Mini Mental State Examination; PD, Parkinson's disease; rCBF, regional cerebral blood flow

**Table 1** Baseline characteristics of the control and PD group

| Parameter | Controls (n = 33) | Patients (n = 30) | p |
|---|---|---|---|
| Age (years) | 64.0 (1.3) | 64.0 (1.5) | 0.68 |
| Women (%) | 42.4 | 43.3 | 0.96 |
| Married (%) | 63.3 | 80.0 | 0.56 |
| University degree (%) | 39.4 | 43.3 | 0.84 |
| Weight (kg) | 73.3 (3.0) | 73.2 (2.8) | 0.87 |
| Height (cm) | 170.0 (1.8) | 170.1 (1.6) | 0.90 |
| No. of medical conditions*¶ | 1.4 (0.3 | 0.4 (0.1) | 0.004 |
| No. of drugs taken | | | |
| excluding APAs¶ | 1.8 (0.3) | 0.9 (0.20) | 0.02 |
| including APAs | 1.8 (0.3) | 2.5 (0.20) | 0.11 |
| Duration of Parkinson's disease (years), mean (SEM) (range) | – | 7.3 (0.9) (0.5–16) | – |
| Hoehn and Yahr stage†, mean(SEM) (range) | – | 2.1 (0.20) (1–3) | – |
| Schwab and England rating‡, mean(SEM) (range) | – | 85.8 (2.0) (60–100) | – |
| Duration of L-dopa treatment (years),§ mean (SEM) (range) | – | 4.9 (0.2) (0.02–12) | – |
| L-dopa daily dose (mg)§, mean (SEM) (range) | – | 572 (71) (100–1500) | – |
| Sialorrhea (%) | – | 43.3 | – |
| MMSE score | 28.3 (0.3) | 28.4 (0.3) | 0.78 |
| BDI score | 12.7 (1.4) | 13.2 (1.4) | 0.64 |
| AUDIT score | 2.7 (0.4) | 2.4 (0.4) | 0.54 |
| Current smokers (%) | 27.3 | 6.7 | 0.07 |
| Tea drinking (cups per day) | 3.9 (0.3) | 3.3 (0.3) | 0.22 |
| Coffee drinking (cups per day) | 0.6 (0.1) | 0.7 (0.1) | 0.59 |
| Adding sugar to caffeinated beverages (spoonful per cup) | 1.1 (0.1) | 1.2 (0.2) | 0.96 |
| Sweet craving on the day of testing | 1.4 (0.3) | 2.5 (0.5) | 0.10 |
| Self reported smell impairment (%) | 6.1 | 36.7¶ | 0.01 |
| Self reported taste impairment (%) | 6.1 | 13.3 | 0.37 |

Values are expressed as means (SEM) unless otherwise indicated.
The Mann-Whitney U test or the $\chi^2$ test was used for inter-group comparisons.
*The sum of chronic medical conditions other than Parkinson's disease was calculated for each subject.
†Stage 1–11 patients, stage 2–7 patients, stage 3–12 patients.
‡Schwab and England.[75]
§Calculated for the 26 patients treated with the drug.
¶Indicates significant inter-group differences.
APAs, anti-parkinsonian agents.

The subjects in both groups were white, aged 44−75 years, with no prior history of psychiatric disorders except nicotine dependence, and had had no acute medical conditions over the previous 30 days. Only non-demented subjects whose Mini Mental State Examination (MMSE) scores[25] were ⩾24 were included to the study.

The study was carried out in accordance with the Declaration of Helsinki, and the study protocol was reviewed and approved by the Ethics Committee on Human Studies (protocol no. IPiN/13/2001). Each participant read and signed an informed consent form after study procedures had been fully explained. The subjects were paid for their participation (100 PLN = €22).

## General design

A single test session was conducted between 10 am and 12.30 pm in a quiet, well ventilated, and temperature controlled room. The participants were asked to refrain from eating, drinking, and smoking for at least 1 hour prior to the test session.

The subjects were questioned regarding basic sociodemographic variables, chronic medical conditions and drugs taken, drinking alcohol, coffee, and tea, smoking cigarettes, adding sugar to caffeinated beverages, and subjective taste or smell impairment. Craving for sweets on the day of testing was rated on an 11-point numerical scale (0 = "not at all", 10 = "very much"). The Alcohol Use Disorders Identification Test (AUDIT)[26] was used to assess alcohol consumption, then the 21 item Beck Depression Inventory (BDI)[27–29] was completed by each participant.

## Preparation of taste samples

Identical discs (1.3 cm in diameter) were cut from filter paper sheets (Filtrak® no. 388, Spezialpapier-Filtrak GmbH, Post Bärenstein, Germany). Twelve solutions, three for each basic tastant, were prepared with sterile deionised water (Polfa, Lublin, Poland) and stored at room temperature. The paper discs were dipped into the sucrose (10, 25, 60%, w/v; Sigma, Poznan, Poland), quinine hydrochloride (0.025, 0.1, 0.5%; Polfa), citric acid (0.25, 1.0, 4.0%; Sigma), or sodium chloride (1.25, 5.0, 20.0%; Polfa) solutions until they were completely soaked. Another set of discs were soaked in deionised water only, to serve as control cues. The filter papers were allowed to dry at room temperature, packed in separate airtight envelopes, and stored at 4℃. The filter paper methodology was used instead of tastant solutions to avoid olfactory stimulation via the so-called retronasal route,[18] which could be a confounding factor, as PD has been linked to early diminution of olfactory function.[17 19 21]

A row of the "sweet", "bitter", "sour", "salty", and "water" discs was prepared for each participant 1 hour before the test and stored at room temperature. Each participant received and rated 13 different filter paper discs. The order of sample administration was counterbalanced across the subjects, although the "bitter" papers were always administered at the end. The 100 ml samples of chocolate and vanilla milk were prepared for each subject from commercially available ultra heat treated milk products (Mlekovita, Wysokie Mazowieckie, Poland). The same volume of deionised water (Polfa) was prepared as another control stimulus.

It should be mentioned that validated tests of taste function are not commercially available. The method used

in the present study has not been formally assessed in terms of its reliability and validity in a white population. The concentrations of the tastants were selected on the basis of our preliminary studies in a group of adult volunteers. The samples were identified by most of the subjects and intensity ratings varied with concentration (Bienkowski *et al*, unpublished). The same was true for the groups tested in the present study (see below).

### Electrogustometry

The electrogustometer (TR-06; Rion Co., Ltd., Tokyo, Japan) used in the study is a commercially available device for human taste examination.[22 23] The apparatus allows delivery of anodal currents of low intensity (from −8 dB to 34 dB, in 2 dB steps; 4–400 μA) at known duration. In the present study, the stimulus duration was kept at 0.5 s.[22 30]

The electrogustometer was equipped with a stainless steel, flat, circular stimulus rod (5 mm in diameter) and a larger indifferent electrode (a neck band). During the test, the stimulus probe was placed on the tongue tip,[23] while the indifferent electrode was attached to the subject's neck. The participant signalled any new taste sensation on the tongue with the aid of a response button connected to a small buzzer.

### Test session

The taste examination started 15 minutes after completion of the BDI. Each participant was familiarised with all procedures and rating scales before the start of the test.

Firstly, a modified version of initially ascending, single staircase detection threshold procedure was used to assess electrogustometric threshold. The subject was asked to signal any new taste sensation on the tongue by pressing the response button. Care was taken to confirm that each PD patient could really control the button. The current intensity was increased if no response occurred within 3 seconds. The current intensity was decreased (reversal) if the subject signalled detection of the stimulus. The reported threshold (in μA) was an average of the last four of the six reversals.

Five minutes after completion of electrogustometry, delivery of the filter paper discs started. Each paper was applied on the tongue tip with sterilised tweezers. Neither the experimenter nor the participant was aware of the actual content of the filter papers. The participant was asked to saturate the paper with saliva for 10–15 s and to taste the liberated tastant within the entire oral cavity. The subject rated taste intensity and pleasantness on 11 point numerical scales labelled at the ends (for intensity 0 = "not at all" to 10 = "extremely", and for pleasantness 0 = "extremely unpleasant" to 10 = "extremely pleasant"). In addition, the subject was asked to describe the taste of the paper using one of five categories: "sweet", "bitter", "sour", "salty", or "none of the above". "Sweet", "bitter", "sour", and "salty" was assumed to be a correct description of the sucrose, quinine, citric acid, and sodium chloride samples, respectively. "None of the above" was considered a correct descriptor of the neutral paper stimulus. The subjects were not required to perform any test related manual tasks as their responses were registered on the response forms by the experimenter. The testing of each paper disc was followed by a 60 second interval during which the subjects rated and identified the tastant, rinsed their mouths with deionised water, and waited for the next sample. The test was performed by the experimenter blinded to the actual content of the paper discs and no feedback was given to the participants as to the correctness of their taste responses.

Five minutes after presentation of the last filter paper, the participant was asked to drink slowly and taste carefully the 100 ml sample of chocolate milk. The same volume of distilled water then served as the neutral stimulus, and was followed by the sample of vanilla milk. Perceived intensity and pleasantness of the samples was rated on the 11 point scales. The subject was also required to identify the taste of the sample as described above. "Sweet" was assumed to be a correct descriptor of the milk samples. "None of the above" was considered a correct descriptor of the water sample. The testing of each sample was followed by a 120 second interval during which the subjects rated and identified the tastant, rinsed their mouths with deionised water, and waited for the next sample.

### Statistics

The Friedman analysis of variance was used to analyse whether rated intensity of a given tastant varied with its concentration. The Mann-Whitney U test or the $\chi^2$ procedure (for non-parametric data) was chosen for inter-group comparisons of sociodemographic parameters and taste responses. The Spearman R test was employed to search for correlations between the clinical parameters (table 1) and those taste responses in the Parkinsonian subjects for which significant inter-group differences had been identified. In addition, correlations between responses to sweet tastes and L-dopa dose and treatment duration were calculated for the PD group. The non-parametric tests were chosen after the Kolmogorov-Smirnov test had revealed that most sociodemographic parameters and taste responses were not normally distributed. A probability level $<0.05$ was considered significant. No correction for multiple comparisons was applied. All statistical analyses were performed with the aid of the Statistica software package for Windows (version 5.0; StatSoft, Tulsa, OK, USA).

## RESULTS

Baseline characteristics of the control and PD group are shown in table 1. The two groups were similar with respect to age, proportion of men and women, marital status, university education, height, weight, coffee or tea drinking, adding sugar to caffeinated beverages, and sweet craving on the day of testing. The BDI, AUDIT, and MMSE scores did not differ between the groups.

The mean number of chronic medical conditions and drugs taken (excluding PD and the antiparkinsonian medications, respectively) was significantly higher in the controls than in the PD group (table 1). No difference between the groups was observed when the antiparkinsonian drugs were included in the analysis. There was a non-significant trend towards a higher percentage of current smokers in the control group ($p = 0.07$).

More than one third of the patients reported subjective smell impairment (table 1) compared with only two controls reporting that symptom ($p = 0.01$). Taste problems were indicated by four PD and two control subjects ($p = 0.37$). Notably, only one PD subject reported isolated taste impairment; the other three patients reported both smell and taste diminution.

Table 2 shows electrogustometric thresholds, taste intensity ratings, and proportions of correct taste identifications in both groups. The patients presented significantly lower electrogustometric threshold compared with the controls. The Friedman one way analysis of variance confirmed that intensity ratings varied with concentration for each tastant including the milk samples ($p<0.01$). No inter-group differences were found in terms of perceived intensity of the control cues or of the sucrose, citric acid, sodium chloride, or chocolate or vanilla milk samples ($p >0.05$). The same was true for rated intensity of the paper discs soaked in the two higher quinine concentrations. The PD group rated the papers soaked in 0.025% quinine as more intense ($p = 0.04$). In general, the subjects correctly identified most of the taste

**Table 2** Electrogustometric threshold, taste intensity and identification in the control and PD group

| | Intensity (0–10) | | | Correct identification (%) | | |
|---|---|---|---|---|---|---|
| | Controls | Patients | p | Controls | Patients | p |
| Filter paper discs | | | | | | |
| Neutral (water-soaked papers) | 0.5 (0.2) | 0.8 (0.2) | 0.19 | 91 | 67 | 0.42 |
| Sucrose | | | | | | |
| 10.0% | 3.9 (0.4) | 4.0 (0.4) | 0.96 | 85 | 83 | 0.96 |
| 25.0% | 6.3 (0.4) | 6.0 (0.5) | 0.64 | 88 | 97 | 0.79 |
| 60.0% | 7.1 (0.4) | 6.1 (0.5) | 0.11 | 94 | 93 | 0.99 |
| Quinine | | | | | | |
| 0.025% | 1.9 (0.4) | 3.2 (0.5)* | 0.04 | 39 | 60 | 0.34 |
| 0.1% | 6.2 (0.5) | 6.9 (0.5) | 0.30 | 94 | 93 | 0.99 |
| 0.5% | 8.3 (0.4) | 7.6 (0.5) | 0.23 | 97 | 100 | 0.93 |
| Citric acid | | | | | | |
| 0.25% | 2.4 (0.3) | 3.2 (0.4) | 0.11 | 64 | 73 | 0.72 |
| 1.0% | 5.6 (0.5) | 6.2 (0.4) | 0.45 | 79 | 70 | 0.76 |
| 4.0% | 8.4 (0.3) | 8.2 (0.3) | 0.33 | 91 | 83 | 0.81 |
| Sodium chloride | | | | | | |
| 1.25% | 3.5 (0.5) | 3.9 (0.5) | 0.54 | 64 | 67 | 0.91 |
| 5.0% | 6.1 (0.4) | 6.1 (0.4) | 0.93 | 82 | 80 | 0.95 |
| 20.0% | 8.4 (0.3) | 8.2 (0.3) | 0.80 | 91 | 90 | 0.98 |
| Liquid samples | | | | | | |
| Chocolate milk | 6.6 (0.3) | 6.2 (0.4) | 0.44 | 94 | 97 | 0.94 |
| Water | 0.8 (0.2) | 2.2 (0.4) | 0.06 | 85 | 90 | 0.87 |
| Vanilla milk | 6.5 (0.4) | 6.6 (0.4) | 0.93 | 100 | 100 | 1.00 |

The electrogustometric threshold (μA) was 95.6 (20.0) for controls and 27.0 (6.5) for patients; p = 0.001 (significant difference).
Intensity ratings are expressed as means (SEM).
Correct identification refers to the percentage of subjects who described the control stimuli as "none", the sucrose samples as "sweet", the quinine samples as "bitter" etc. "Sweet" was assumed to be the correct answer in the case of the milk samples.
*Significant inter-group differences.

samples, with no significant differences between the controls and PD patients ($p>0.3$). The Spearman R test revealed that neither electrogustometric thresholds nor intensity ratings of the 0.025% quinine papers correlated with the clinical parameters specified in table 1 ($R<0.3$, $p>0.1$).

Table 3 shows pleasantness ratings of all tastants. The Mann-Whitney U test indicated that pleasantness ratings did not differ between the two groups ($p>0.1$). Taste responses to the sucrose and milk samples recorded for the PD patients did not correlate with L-dopa dose and treatment duration ($R<0.3$, $p>0.1$).

**Table 3** Taste pleasantness in the control and PD group

| | Controls | Patients | p |
|---|---|---|---|
| Filter paper discs | | | |
| Neutral (water-soaked papers) | 4.7 (0.2) | 4.4 (0.3) | 0.70 |
| Sucrose | | | |
| 10.0% | 7.0 (0.3) | 6.6 (0.2) | 0.25 |
| 25.0% | 7.1 (0.3) | 7.4 (0.3) | 0.61 |
| 60.0% | 7.8 (0.3) | 7.3 (0.2) | 0.11 |
| Quinine | | | |
| 0.025% | 4.0 (0.3) | 3.4 (0.3) | 0.14 |
| 0.1% | 2.1 (0.3) | 1.8 (0.3) | 0.35 |
| 0.5% | 1.3 (0.2) | 1.4 (0.3) | 0.89 |
| Citric acid | | | |
| 0.25% | 4.6 (0.2) | 4.9 (0.3) | 0.43 |
| 1.0% | 4.6 (0.3) | 4.4 (0.4) | 0.67 |
| 4.0% | 3.7 (0.5) | 4.5 (0.5) | 0.29 |
| Sodium chloride | | | |
| 1.25% | 4.8 (0.3) | 4.2 (0.3) | 0.18 |
| 5.0% | 4.9 (0.4) | 4.0 (0.3) | 0.18 |
| 20.0% | 3.8 (0.5) | 3.0 (0.5) | 0.37 |
| Liquid samples | | | |
| Chocolate milk | 8.6 (0.2) | 8.1 (0.3) | 0.20 |
| Water | 5.1 (0.3) | 5.5 (0.3) | 0.61 |
| Vanilla milk | 8.0 (0.2) | 7.8 (0.3) | 0.85 |

Pleasantness ratings are expressed as means (SEM).

## DISCUSSION

The two groups recruited for the present study did not differ in terms of several important sociodemographic and biological parameters including age, gender ratio, marital status, university education, height, weight, cognitive status, alcohol related problems, and coffee and tea drinking. Given the hypothesis tested in the study, it should be stressed that adding sugar to caffeinated beverages and sweet cravings on the day of testing were also similar in the two groups. As might be expected, smoking tended to be less frequent in the PD group. In this respect, our findings support previous reports on the negative association between smoking rates and PD.[31 32] It was also not surprising that the number of chronic medical conditions other than PD was lower among the patients. It has been reported that PD is associated with equal (for ischaemic stroke, hypertension, and diabetes mellitus) or lower (for myocardial infarction, coronary artery disease, atrial fibrillation, and cancer) cumulative incidence of various physical illnesses compared with the general population.[33 34] The difference in the number of medical conditions fits with the lower number of non-parkinsonian medications taken by the PD group. On the other hand, when the antiparkinsonian drugs were included in the analysis, the PD patients tended to consume more drugs than the controls. It should be noted that all these differences may modify the pattern of taste responses.

Notably, the PD patients and controls did not differ in depressive symptoms as measured by the BDI.[27 28] BDI scores in the present study were similar to those reported previously for PD patients with similar clinical characteristics.[35 36] It has repeatedly been shown that PD subjects present more depressive features than age matched controls; however, estimates of frequency of depression in PD vary widely, from 2.7% to 70%.[37–39] Hence, although we may not exclude the possibility that other questionnaires would have identified inter-group differences in depressive symptoms, it seems

reasonable to suggest that depression was not a major factor contributing to the present results.

Contrary to the hypothesis formulated in the Introduction, perceived pleasantness of the sweet samples (sucrose, chocolate milk, and vanilla milk) did not differ between the PD and control group. In addition, hedonic ratings of the other samples (bitter, sour, and salty) were also similar in the PD patients and controls. Thus, it seems that dopaminergic dysfunction in the PD subjects[2 9] did not lead to any obvious alteration in perceived pleasantness/aversiveness of gustatory stimuli. Animal studies with the taste reactivity paradigm have revealed that neither dopamine receptor antagonists nor dopamine depletion in the striatum altered appetitive taste responses to sucrose solutions.[1 40–42] Moreover, dopamine receptor antagonists fail to diminish the rewarding effects of amphetamine or cocaine in humans.[4 43 44]

Our results are also in accordance with a recent report of Künig *et al*,[45] who used a simple operant task to measure regional cerebral blood flow (rCBF) in response to monetary reward. PD patients and age matched controls presented distinct patterns of rCBF increases in response to monetary reward, but the subjective value of earnings did not differ between the groups. The PD group earned slightly less money during the study.[45] In line with the latter finding, Czernecki *et al*.[46] have shown that PD patients were impaired on stimulus−reward learning and reversal in an operant procedure in which points and tones served as symbolic rewards. However, the Parkinsonian group presented normal extinction of responding and actually tended to emit more responses when rewarding feedback was withdrawn. In another study,[36] PD patients and osteoarthritis controls with comparable degrees of disability did not differ in mean hedonic tone scores on the Snaith-Hamilton Pleasure Scale.[47] In contrast, the PD group had significantly higher levels of apathy, defined as reduced interest in purposeful behaviours, which were positively correlated with executive impairment. A subgroup of the PD patients with higher levels of apathy showed more anhedonia on the Snaith-Hamilton Pleasure Scale.[36] The inverse relationship between apathy and executive functioning was also demonstrated in a larger sample of Parkinsonian individuals.[48]

More apathy in PD subjects was also found by Isella *et al*.[8] In the same study, the Parkinsonian patients presented mild symptoms of physical anhedonia as measured by the Physical Anhedonia Scale,[49] but they were also more depressed and demented. The PD group had worse performance in tests of executive function, and physical anhedonia tended to correlate with executive impairment.[8] Thus, the results of the present and previous studies[1 4 8 36 45 46 50–52] may indicate that it is learning and/or motivational deficit (apathy) that impairs processing of rewarding cues in PD and that hedonic tone may be largely unaffected in PD subjects. This notion fits well with theories linking striatal dopaminergic transmission to preparatory phase of motivational behaviours and reward associated learning, but not to subjective pleasure and euphoria.[1 3 4 40 42 43]

Contrary to the data discussed above, it has been reported that PD patients present decreased sensitivity to the rewarding effects of oral methylphenidate.[12] In that study, two pairs of outliers were eliminated from the PD and control group and the final group size was relatively small ($n = 10$ subjects/group). The inter-group differences after that adjustment were still modest and not dose dependent.[12] Moreover, in an earlier experiment,[53] only PD subjects with major depression reported less euphoria and activation after intravenous methylphenidate administration. Subjective responses to methylphenidate in PD patients without major depression were similar to those observed in healthy controls. Surprisingly, the rewarding effects of methylphenidate in the depressed subjects without PD remained unaffected.[53] The latter observation corresponds with reports of other researchers indicating that depressed individuals may even show enhanced reactivity to natural and chemical rewards. Hospitalised, depressed patients rated high concentration sucrose solutions as more pleasant than did non-depressed controls.[54] Recently, strong positive correlations between severity of major depression and the amphetamine rewarding effects have been reported by Tremblay *et al*.[55] Given the above, it may be hypothesised that neither atrophy of dopaminergic neurones in PD[2 9 10] nor serotonergic/noradrenergic dysfunction in major depression[56] is sufficient for the development of clinically relevant anhedonia. Only the combined impairment of the three monoaminergic systems, which is probably present in depressed PD individuals,[2 9–11 37 39 57–60] invariably leads to anhedonia.[53] This hypothesis needs further validation in multidisciplinary studies.

Notably, the PD group recruited for the present study did not show any major sensory deficit as assessed by intensity ratings and identification of the gustatory samples. Indeed, the PD patients rated the filter papers soaked in the lowest quinine concentration as more intense, compared with the control group. This finding corresponded with lower electrogustometric thresholds observed in the patient group. Neither electrogustometric thresholds nor reactivity to the quinine papers correlated with the basic clinical characteristics of the PD group.

Lower electrogustometric threshold in the PD group does not mean that the patients had enhanced taste sensitivity. It is possible that for some reason threshold taste responses were diminished in the control group; however, our recent results argue against this latter possibility. A mean electrogustometric threshold in a control group (mean age 46.2 years) recruited to another study was similar (102.1 μA; Bienkowski *et al*, unpublished) to that reported in the present study. Lower taste thresholds in the PD group are difficult to reconcile with several reports that olfactory, auditory, visual, and tactile perception may be compromised in PD.[17 19 21 61–67] In the present study, the percentage of subjects reporting subjective smell impairment was significantly higher in the PD group (36.7%) and similar to that reported by other researchers (28%).[62] In contrast, there was no inter-group difference in the percentage of subjects reporting taste dysfunction. Furthermore, subjective taste impairment in some PD patients could have been secondary to olfactory deficits.[68] Bearing in mind the functional[18 69] and neuroanatomical[15 18] interconnections between taste and olfaction, it may be speculated that olfactory deficits in PD[17 62 64] might be compensated by enhanced taste reactivity (like in the present study). However, we did not find any difference in electrogustometric threshold between the PD patients reporting and not reporting olfactory deficits ($p = 0.71$). Thus, although attractive, the "compensatory" hypothesis needs validation in future studies in which correlations between individual olfactory and taste responses would be calculated.

There is no consensus on oral health status in PD. In one study, PD subjects had significantly more teeth and less caries compared with age matched controls. On the other hand, salivary secretion rate was reduced in a subgroup of patients with more severe parkinsonian symptoms.[70] More mucositis was also found in the oral cavities of PD patients.[71] Similarly, a rather inconsistent relationship was observed between PD and dietary habits, although some association with protein rich and carbohydrate rich foods cannot be excluded.[72–74] The present results may indicate that diminution of taste function is not responsible for altered dietary choices and oral health status in PD.

Limitations of the present study involve, firstly, some clinical characteristics of the recruited group. All but one PD patients were treated with L-dopa or other dopaminergic therapeutics and all patients were examined in the "on" state. Hence, it could be hypothesised that dopaminergic treatment normalised any pre-existing hedonic deficit in the PD patients. Although we cannot rule out the above hypothesis, it should be borne in mind that L-dopa dose and treatment duration did not predict taste responses to sweet tastes in our patients. Other limitations include the inter-group differences in the number of chronic medical conditions, drugs taken, and cigarette smoking. It should also be borne in mind that the methods used in the study needs further validation and may not reflect all aspects of taste responses in real life conditions. Thus, further research is needed to confirm the present findings.

Concluding, the present results suggest that: (*a*) PD does not lead to any profound alterations in perceived intensity, pleasantness, or identification of suprathreshold gustatory stimuli, and (*b*) PD patients may show enhanced taste acuity as assessed by electrogustometry.

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