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Research article

Facial expressions and identities recognition in Parkinson disease

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ABSTRACT

Parkinson's Disease (PD) is associated with motor and non-motor symptoms. Among the latter are deficits in matching, identification, and recognition of emotional facial expressions. On one hand, this deficit has been attributed to a dysfunction in emotion processing. Another explanation (which does not exclude the former) links this deficit with reduced facial expressiveness in these patients, which prevents them from properly understanding or embodying emotions. To disentangle the specific contribution of emotion comprehension and that of facial expression processing in PD's observed deficit with emotions we performed two experiments on non-emotional facial expressions. In Experiment 1, a group of PD patients and a group of Healthy Controls (HC) underwent a task of non-emotional expression recognition in faces of different identity and a task of identity recognition in faces with different expression. No differences were observed between the two groups in accuracies. In Experiment 2, PD patients and Healthy Controls underwent a task where they had to recognize the identity of faces encoded through a non-emotional facial expression, through a rigid head movement, or as neutral. Again, no group differences were observed. In none of the two experiments hypomimia scores had a specific effect on expression processing. We conclude that in PD patients the observed impairment with emotional expressions is likely due to a specific deficit for emotions to a greater extent than for facial expressivity processing.

1. Introduction

Parkinson's disease (PD) typically encompasses a spectrum of symptoms, including both motor [1,2] and non-motor [3] manifestations. While the latter are frequently underestimated, research indicates that every patient experiences at least one non-motor symptom [4], which can have an adverse effect on their quality of life [5]. Non-motor symptoms encompass affective and cognitive impairments [6] and are frequently linked to challenges in social cognition [7].

One of the most studied abilities in PD is recognizing emotional facial expressions [8–10]. Deficits have been observed in the

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identification, discrimination, and recognition of emotional facial expressions for patients with PD when compared to healthy controls [11,12]. Different factors influence facial emotion recognition abilities in PD. These include, disease duration and the degree of its severity, mood disturbances and pharmacological therapy [13]. Various explanations have been proposed to account for the impairment of emotional facial expression recognition in individuals with Parkinson's disease. The emotions more consistently impaired in the mentioned studies are the so-called negative emotions [11], with some authors attributing this specific impairment to the effects of PD on their neural substrate. For instance, disgust is reported to be subserved by insula and striatum, strictly connected with basal ganglia, the system impaired in PD [14,15]. In line with this, Martins and colleagues [16] have suggested that the reduction in dopamine levels in the brain resulting from the disease leads to the inability to recognize certain emotions, such as anger and fear. Furthermore, various hypotheses regarding the neural structures involved in this deficit extend beyond the basal ganglia to encompass areas like the Orbitofrontal Cortex, the Anterior Cingulate Cortex, the Prefrontal Cortex, and the amygdala.

While not all studies have investigated the recognition of all six basic emotions [17], results in favour of a bigger impairment with the negative emotions appear to be robust [18].

An alternative explanation for the deficit in emotion recognition in PD could be its potential association with impairments in emotion expressivity [19]. This interpretation relies on the embodied simulation theory, which posits that emotion recognition is improved through internal simulations of the expressions being observed. These simulations should occur when viewing an emotional facial expression which in turn activates the corresponding emotion in the viewer [11]. Therefore, deficiencies in emotional expression recognition may stem from impaired facial expressiveness. On the one hand, signs commonly observed in PD patients are amimia, hypomimia, or facial bradykinesia [20–22] leading to reduced facial expressiveness. On the other hand, it's possible that the reduced facial expressiveness in PD patients compared to controls is linked to a dysfunction in the mirror neuron system [23]. Whatever the cause, it seems that blocking facial mimicry impacts emotion recognition. Indeed, numerous studies have observed reduced mimicry in PD patients when compared to control subjects [24,25]. This reduction has subsequently been associated with a decline in emotion recognition within this patient group [19,26–29]. It is worth noting that there is variability in the outcomes, with some studies reporting a decrease in the ability to mimic specific emotions exclusively [30,31].

In conclusion, despite extensive research on emotional facial expression recognition in Parkinson's Disease, there remains ongoing debate regarding the interpretations of the findings. A way to shed light on it is by investigating expression recognition using non-emotional expressions. In this way, we can disentangle the contribution of a deficit in emotional processing in PD [32] from that of an expression simulation deficit. If the deficit primarily concerns emotions, we anticipate that PD patients would not encounter difficulties in processing non-emotional facial expressions. Conversely, if the deficit primarily concerns expressions, we expect PD patients to exhibit the same challenges in processing non-emotional facial expressions as they do with emotional ones. To the best of our knowledge, there are limited studies on non-emotional facial expressions. One study investigated facial expression processing encompassing both emotional and non-emotive expressions [25] and concluded that there were no differences in expression recognition in PD patients compared with controls. This outcome could provide insight into a deficit in PD patients that is specifically associated with the processing of emotions rather than the simulation of expressions for comprehension. Conversely, another study investigated ERPs in PD patients in response to both emotional and non-emotional expressions. The authors divided PD patients into LPD (i.e., patients with disease insurgence on the right hemisphere causing initial symptoms on the left side) and RPD (i.e., patients with disease insurgence on the left hemisphere causing initial symptoms on the right side). They found that LPD but not RPD were impaired in dynamic expression processing compared to controls [33], hinting at a causal role of expression simulation and a major role of the left hemisphere.

Furthermore, although numerous studies reported challenges in identifying emotional facial expressions in PD, only a limited number presented data regarding identity processing. Some authors found specific difficulties in unfamiliar identity recognition measured by both the Benton Face Recognition test [14,34] and the Cambridge Face Memory Test [17,35]. Others found difficulties in configural processing and link them, at least partially, to emotion recognition [36,37]. However, it is unclear whether these are deficits proper of the disease or if the results obtained are a consequence of aging and developing Parkinson's Disease Dementia [38].

Thus, the aim of Experiment 1 was to investigate whether there were differences in recognition of non-emotional facial expressions and identities in PD patients compared to healthy controls. If the observed deficit in PD for the recognition of emotional expressions is mainly caused by the difficulty with emotions, we would expect them not to show difficulties in recognition of non-emotional facial expressions. On the other hand, if the deficit in emotional expression recognition is mainly caused by the mimicry component, we would expect our patients to show difficulties in recognition of non-emotional facial expressions.

Moreover, it must be noted that models of face recognition postulate that e identify faces through two distinct yet interconnected systems: a ventral system responsible for discerning the structural features of faces, and a dorsal system dedicated to processing facial expressions [39–41]. Those systems interact with each other in the recognition of facial identity. Hence, if the deficit in recognizing emotional expressions stems from a problem specific to expressions, we would anticipate that these patients would likewise encounter difficulties in identifying face identity when faces are presented with an expression. That is why we designed Experiment 2, where participants needed to recognize faces presented as neutral, with a dynamic non-emotional facial expression, and a control condition for the motion component (i.e., a rigid head movement). Expressions and rigid head movements were presented as in motion because it has been documented in the literature an advantage of moving faces in their recognition [42]. PD patients have deficits in the recognition of facial expressions and if it is true that the dorsal system for face recognition is involved in the recognition of the identity of faces encoded through an expression, we would expect PD patients to have difficulties in recognizing faces encoded through an expression but not through static features (i.e., neutral condition). Again, we used non-emotional facial expressions to distinguish the role of expressions from that of emotional processing.

2. Experiment 1

2.1. Aim

We hypothesized that the PD patients' hypomimia could impair the recognition of changeable features in the faces of others. The first aim of Experiment 1, indeed, was to verify whether PD patients show a deficit in non-emotional facial expressions recognition. To do so, we tested them through a discrimination test of non-emotional expressions displayed in different facial identities and compared them with healthy controls. The second aim of Experiment 1 was to investigate the ability of facial identity recognition in PD, net of facial expressions. To do so, we administered a task of facial identity recognition displayed with different expressions to our patients and a group of healthy controls. We also considered the relation between hypomimia scores and scores in the experimental tasks.

2.2. Methods

2.2.1. Participants

Twenty-two patients and twenty-four control participants took part in the study. The sample size was determined through the software Gpower (parameters: power = 0.90, α = 0.05, medium effect size = 0.25) for a 2*2 design where the dependent variable was accuracy and the independent variables were the two types of experiments (expressions vs identities) and the two groups (patients vs controls).

22 patients (12 females, mean age = 62,09; sd = 9,42) were recruited through the institute "Istituti Clinici Zucchi" of Carate Brianza, Italy. Twenty-four patients were initially collected: however, two had to be excluded for not finishing the experimental tasks. Patients with PD met clinical criteria for mild to moderate idiopathic PD (Hoehn & Yahr Stage 1–3, [43]).

Motor disability was measured through the Unified Parkinson's Disease Rating Scale (UPDRS, [44]). All individuals with PD were taking medications for their motor symptoms and were in the "on" state during testing.

Exclusion criteria for the patient group included: non-corrected visual or auditory problems; history of alcohol or drug abuse; DBS implant; neurological (other than PD) or psychiatric illness; a deficit in a screening test for cognitive functioning (i.e., Montreal Cognitive Assessment, MoCa, [45]); history of traumatic brain injury or intracranial surgical operations.

24 control participants (11 females, mean age = 61,74; sd = 9,60) were recruited through caregivers. Exclusion criteria for the control group included not having a neurological or psychiatric illness; never suffering from traumatic brain injury; not having a deficit in a cognitive screening test (i.e., MoCa, [45]).

Table 1 displays the demographic and clinical characteristics of the PD group and control group and a statistical comparison between the two groups.

All participants were native Italian speakers. Moreover, all participants provided written informed consent and the study was approved by the Ethics Committee of the University of Milan-Bicocca (protocol number 0126263/21). All the procedures used in the study were in accordance with the Declaration of Helsinki.

2.2.2. Stimuli

Stimuli were 10 avatars (5 females) created, starting from the Chicago Face Database [46], through the software Character Creator 3. All avatars were rendered to be without hairs to prevent a facilitatory effect due to the hair cue in the recognition task. For each of the 10 identities, 10 facial expressions were created. Those expressions were previously validated to be non-emotional in a pilot task on an independent sample of participants. An example of stimuli can be seen in Fig. 1.

2.2.3. Procedure

After signing the informed consent, participants were administered a cognitive screening test (i.e., MoCa, [45]). After doing that, they were administered the Benton Facial Recognition Test (BFRT, [47,48]). Parkinson's disease patients were also administered the Questionnaire for impulsive-compulsive disorders in Parkinson's Disease (QUIP-RS, [49]). All participants were then administered the two experimental tasks in counterbalanced order. Both tasks were administered through the program Opensesame (version 4.0.1, [50]). In the expression recognition task, participants looked at a face for 5 s. Right afterward, they were presented with two alternatives and had to say which one represented the same facial expression. Identity changed from target to recognition, but one of the two alternatives represented a face with the same facial expression. An example of an expression recognition trial is represented in Fig. 2. A total of 10 expression was presented (half females), 4 times each with different alternatives in randomised order. No timeout for response was set. In the face recognition task, participants looked at a face for 5 s and, right afterward, were presented with two

Table 1
Means (and standard deviations) of demographic and clinical characteristics of the PD group and control group (HC); T-test comparisons between the two groups and p values are reported.

	PD	HC	T	*p*
Mean age, year (SD)	62.09 (9.42)	61.74 (9.60)	−0.17	0.86
Mean education, year (SD)	11.41 (4.03)	12.29 (4.46)	1.01	0.32
Mean MoCa (SD)	23.86 (2.27)	24.37 (3.06)	0.92	0.36
Mean BFRT (SD)	47.43 (4.48)	48.71 (3.79)	1.47	0.15

Fig. 1. Example of two male and two female identities showing 4 of the 10 non-emotional facial expressions created for the present experiment.

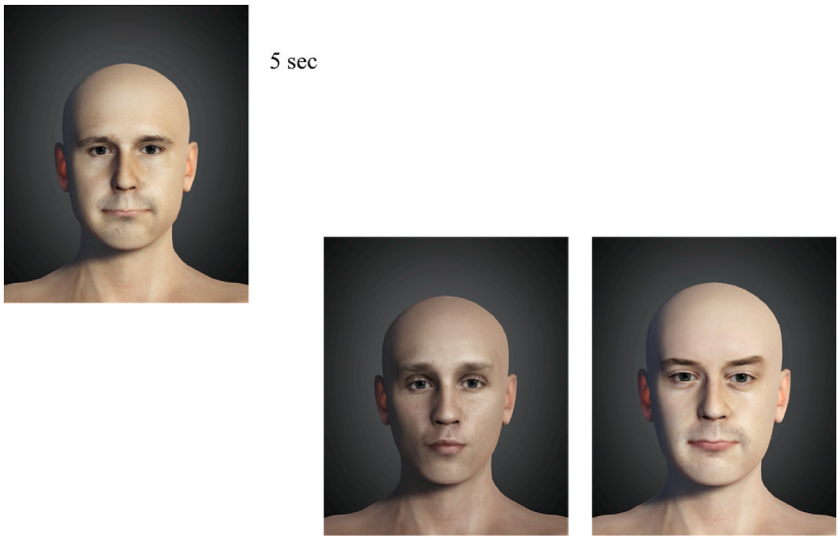

Fig. 2. An example of an experimental trial from the expression recognition task.

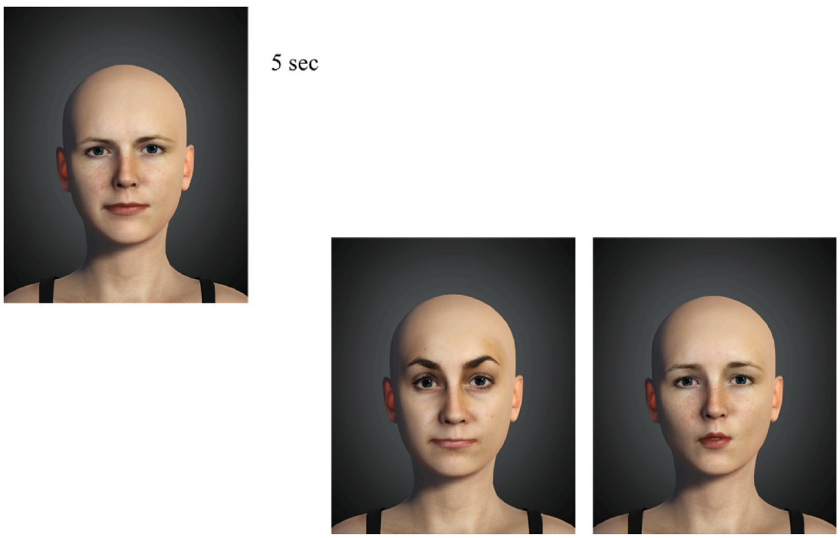

Fig. 3. An example of an experimental trial from the identity recognition task.

faces. They were asked to indicate which of them represented the same person just seen. The two alternatives presented different facial expressions compared to the target, but one represented the same identity. An example of a trial can be seen in Fig. 3. A total of 10 identities was presented (half females), 4 times each with different alternatives in randomised order. No timeout for response was set.

In the case of PD patients, the time when they last took their pharmacological therapy was noted as well as the year and month (when possible) when the first symptoms emerged.

2.3. Results

Statistical analyses were performed through the software R (version 4.0.4) and the packages *lme4* [51], *lmertest* [52], and *emmeans* [53].

Patients and controls were compared on age, education, and scores in the MoCa and BFRT tests by means of independent samples *t*-test. Results indicate that patients and controls did not differ in age, education, MoCa, or BFRT (see Table 1). Data relative to the QUIP questionnaire were not considered as they were almost all 0s (mean QUIP score = 1.05, sd = 3.62).

The dependent variable that was analyzed was the accuracy in the two tasks (Expressions and Identity recognition tasks). Accuracy was calculated as the number of correct responses divided by total number of responses. Analyses were performed excluding an outlier control participant scoring an accuracy of 0.55 in recognizing expressions and 0.97 in recognizing identities. We hypothesized that this control participant might have a selective deficit in recognizing facial expressions compared to identity recognition. For this reason, she was excluded from the analyses. Accuracies were analyzed through a linear mixed effect model where task (i.e., expression or identity) and the group (i.e., HC or PD) were entered as fixed factors, and age, education, MoCa, and Benton were entered as covariates. Single subjects were entered as random factors. Results revealed a main effect of condition ($F_{(1,43)} = 29.652$, $p < 0.0001$). Post-hoc tests revealed that the expression task ($emmean_{(39)} = 0.818$) was harder on average than the identity one ($emmean_{(39)} = 0.902$; t ratio = -5.445, $p < 0.0001$). However, no interaction between the condition and the group was found ($F_{(1,43)} = 2.294$, $p = 0.137$). Results are represented in Fig. 4. Moreover, the BFRT covariate had a significant effect on accuracies ($F_{(1,39)} = 6.875$, $p = 0.0124$). A simple regression with accuracy as the dependent variable and BFRT as the independent variable revealed that the relation between these two variables was positive (estimate = 0.0066, T = 2.523, $p = 0.0134$) that is, the higher the score in BFRT the higher the accuracy in the two experimental tasks. This relation is represented in Fig. 5.

Additionally, the relation between accuracy and the time when the last medication was taken was investigated in the PD group through a simple linear regression where accuracy was the dependent variable and the time since the last assumption was the independent variable. Results reveal no effect of the time since the last assumption on accuracies ($F_{(1)} = 0.612$, $p = 0.4384$). Moreover, the relation between the distance from the insurgence of the disease (calculated from the insurgence of symptoms) and accuracies was investigated through a simple linear regression where accuracy was the dependent variable and the distance from the insurgence the independent variable. Results reveal no effect of the distance from the insurgence of the disease on accuracies ($F_{(1)} = 0.919$, $p = 0.3433$). Eventually, the relation between accuracy and UPDRS hypomimia score and accuracy was investigated in the PD group through a simple linear regression where accuracy was the dependent variable and UPDRS hypomimia score was the independent

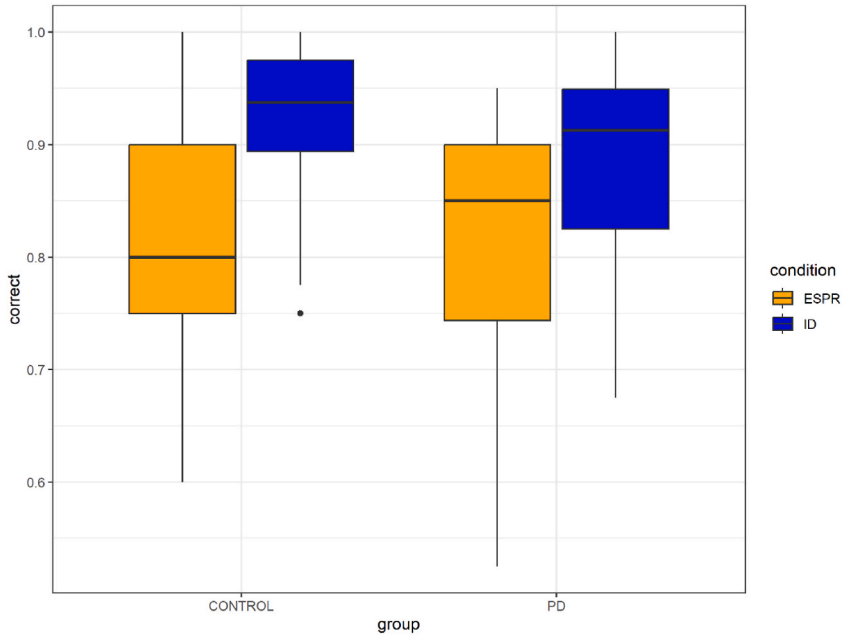

Fig. 4. Graphical representation of results of Experiment 1. A significant effect of condition was found (ID easier than ESPR), while no significant effect of group (PD vs CONTROL) emerged.

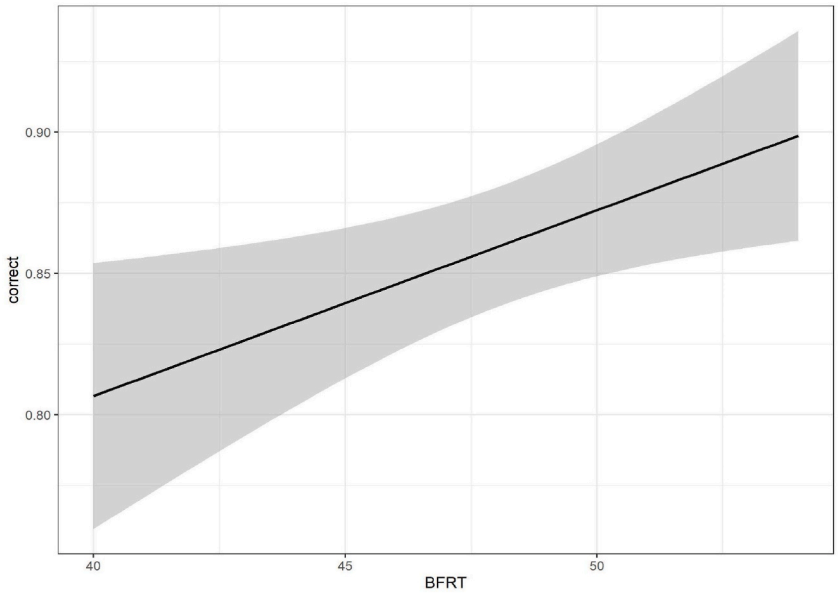

Fig. 5. Graphical representation of the relation between scores in the Benton Face Recognition Test and accuracy scores.

variable. Results reveal no effect of the UPDRS hypomimia scores on accuracies ($F(1) = 0.836$, $p = 0.366$).

3. Experiment 2

3.1. Aim

Experiment 1 showed no differences between PD patients and the control group in recognizing facial identities and non-emotional expressions, however, in that study, only static stimuli were used. The aim of Experiment 2 was to understand whether PD patients show differences in processing faces with a dynamic non-emotional expression. To do so, we tested them and a group of healthy controls in a task where they had to recognize the identity of faces encoded as neutral, with a non-emotional expression, or with a rigid movement.

3.2. Methods

3.2.1. Participants

Twenty-four patients and twenty-five control participants took part in the study. The sample size was determined through the software Gpower (parameters: power = 0.95, $\alpha = 0.05$, medium effect size = 0.25) for a 3*2 (3 experimental conditions * 2 groups) design.

24 patients (10 females, mean age = 67,33; sd = 9.95) were recruited through the institute "Centro Parkinson Bignami" of Milan, Italy. Motor disability was measured through the Unified Parkinson's Disease Rating Scale (UPDRS). All individuals with PD were taking medications for their motor symptoms and were in the "on" state during testing.

Exclusion criteria for the patient group included non-corrected visual or auditory problems; history of alcohol or drug abuse; DBS implant; neurological (other than PD) or psychiatric illness; a deficit in a screening test for cognitive functioning (i.e., MoCa, [45]); history of traumatic brain injury or intracranial surgical operations.

25 control participants (18 females, mean age = 64,64; sd = 9.53) were recruited through caregivers. Exclusion criteria for the control group included: not having a neurological or psychiatric illness; never suffering from traumatic brain injury; not having a deficit in a cognitive screening test (i.e., MoCa, [45]).

Table 2
Means (and standard deviation) of demographic and clinical characteristics of the PD group and control group (HC); T-test comparisons between the two groups and *p* values.

	PD	HC	T	*p*
Mean age, year (SD)	67.33 (9.95)	64.79 (9.67)	−1.48	0.14
Mean education, year (SD)	12.79 (3.45)	14.17 (3.97)	1.66	0.10
Mean MoCa (SD)	23.67 (2.88)	24.96 (2.40)	3.19	0.0017
Mean BFRT (SD)	42.54 (3.78)	44.37 (4.30)	2.97	0.0035

Table 2 displays the demographic and clinical characteristics of the PD group and control group and their statistical comparison. Moreover, a table with details about the pharmacological therapy of patients and their disease severity measured by means of the modified Hohen & Yahr [43] score can be found in the supplementary materials.

All participants were native Italian speakers. Moreover, all participants provided written informed consent and study was approved by the Ethics Committee of the University of Milan-Bicocca (protocol number 0041468/20). All the procedures used in the study were in accordance with the Declaration of Helsinki.

3.2.2. Stimuli

Stimuli were created by selecting 60 identities (30 males) from the Chicago Face Database [46], matched for attractiveness and trustworthiness. Once identities were selected, an avatar was created for each of them using the program Character Creator 3. 10 avatars (5 males) were morphed to assume 6 non-emotional facial expressions, and 25 captures were taken, each one with a different and growing level of intensity of the expressions. The 25 captures were presented in rapid succession (every 60 ms) to induce a perceived movement. A pilot study was conducted to ensure that expressions were non-emotional. In addition to the non-emotional expressions, 10 avatars (5 males) were morphed to produce a rigid head movement on the three rotational axes. This condition served as a movement control condition. In fact, the stimuli displayed a rigid movement, but the facial expression remained neutral. As for the expressions, 25 captures were taken for as many degrees of rotation and presented in rapid succession. To ensure that both expressions and rigid movements were indeed perceived as in motion, a pilot study was run. Eventually, 10 identities (5 males) were created as neutral and static.

Moreover, the static version of all identities was created, and 30 more static neutral identities (15 males) were created to serve as distractors. All images were on a greyscale to avoid recognition driven by color cues. For an example of the three categories of stimuli, see Fig. 6.

3.2.3. Procedure

Both groups underwent cognitive evaluation through MoCa [45], the BFRT [47,48], and the following experimental procedure. The experiment was administered through the program Inquisit (Inquisit 5 [Windows] (2016). Retrieved from https://www.millisecond.com.).

Participants were presented with three blocks in randomized order. Each block belonged to one of the three conditions (i.e., non-emotional facial expression, rigid head movement, or neutral) and was divided into two parts. In the first part, a fixation cross was presented for 500 ms at the centre of the screen. It was followed by the face presented in the experimental condition belonging to the block. Faces in the expression and rigid movement conditions were presented as a sequence of 25 frames, one each 60 ms for a total of 1500 ms. Each face was shown two times to reach a total exposure of 3000 ms [54]. No timeout for response was set. Faces were presented twice because presenting a single face for 3000 ms created a movement that was too slow and, therefore, unnatural. To create movement at a more natural speed, faces were shown twice for 1500 ms each time. For the neutral static condition, the faces were presented for 1500 ms two times to maintain the same exposure scenario as for the other conditions. After viewing each face twice, participants were asked to make a male/female decision to be sure that participants were processing the faces [55]. Once the 10 faces belonging to the experimental condition were judged, the second part began, where the neutral static version of each face was presented for 3000 ms together with the distractors. All the faces that were presented in part 1 (a total of 30) were presented with 30 distractors for a total of 60 stimuli. Participants were asked to say whether they already saw that face or not. For an example of the experimental procedure, see Fig. 7 or a demo version of the experiment at the following OSF link: https://osf.io/6p24r/.

In addition to this, PD patients were administered the Questionnaire for impulsive-compulsive disorders in Parkinson's Disease (QUIP-RS, [49]).

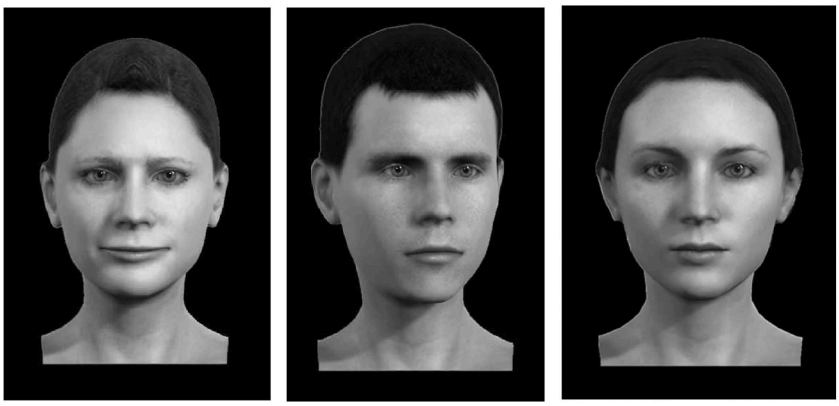

Fig. 6. Examples of the stimuli. On the left is one of the validated non-emotional expressions, in the middle a rigid head movement around the horizontal axis, and a neutral expression on the right. Non-emotional facial expressions and rigid movement stimuli were dynamic, composed by a succession of 25 images (one each 60 ms), while neutral faces were static.

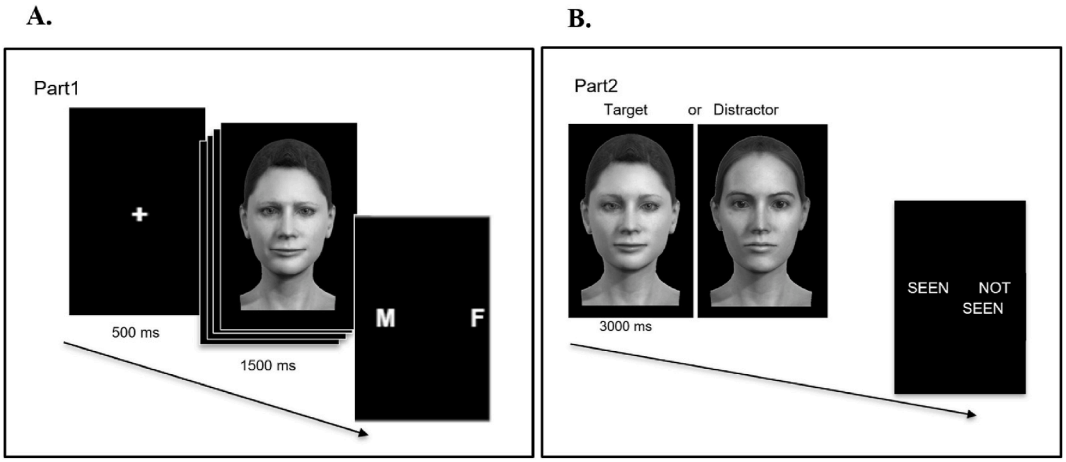

Fig. 7. An example of the experimental procedure. Each block was divided into two parts: in the first part (displayed in panel A) participants saw a fixation cross for 500 ms, followed by a stimulus. Each stimulus was presented twice for 1500 ms. After the stimulus, the letters M and F appeared on the screen, and the participants had to indicate whether the seen stimulus was a male or a female. After seeing the 10 identities belonging to the running block, participants were administered the second part (displayed in panel B). In this part, participants saw a neutral static face, which could be the neutral static version of the already seen stimulus or a distractor. After 3000 ms the stimulus disappeared and a "seen" or "not seen" text was displayed for response.

3.3. Results

Statistical analyses were performed through the software R (version 4.0.4) and the packages *lme4* [51], *lmertest* [52], *emmeans* [53], *singcar* [56], and *psycho* [57].

Patients and controls were compared on age, education, scores in the MoCa and BFRT tests by means of independent samples *t*-test. Results indicate that patients and controls did not differ in age, or education. However, they differed in MoCa raw scores, and BFRT raw scores (see Table 2). Data relative to the QUIP questionnaire were not considered as they were almost all 0s (mean QUIP score = 2.55, sd = 5.09).

The dependent variable considered was the d'prime score [58], and it was calculated for each participant for each condition (i.e., non-emotional expression, rigid head movement, or neutral). D'prime scores were entered in a linear mixed-effect model as the

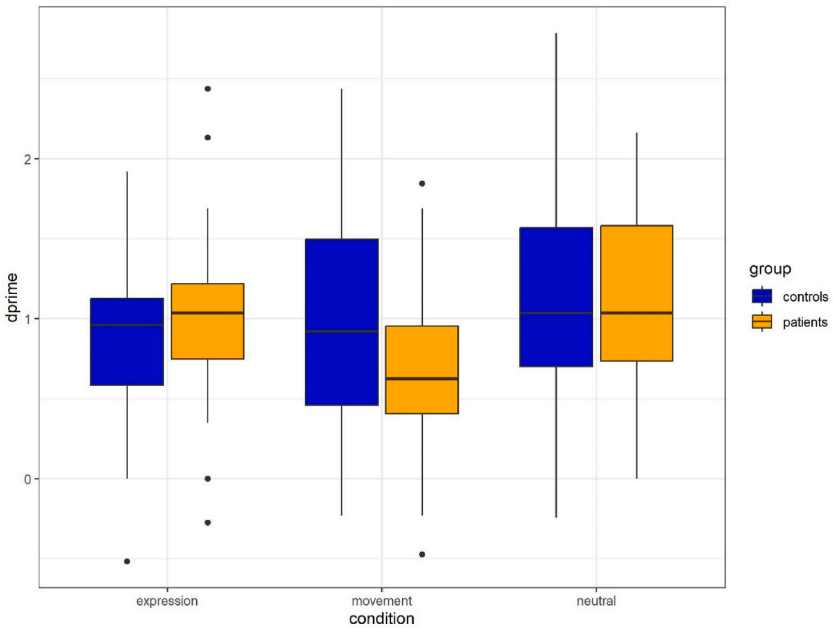

Fig. 8. Graphical representation of dprime scores in each experimental condition for each group. A significant effect of condition emerged: however, the effect of group was not significant.

dependent variable. Group (PD or control) and condition (non-emotional facial expression, rigid head movement, or neutral) were entered as fixed factors. Moreover, age, education, MoCa, and BFRT scores were entered as covariates. Single subjects were entered as random factors. Results reveal a main effect of condition ($F_{(2,92)} = 3.510$, $p = 0.0340$). Post-hoc tests revealed that there was a significant difference between rigid movement and neutral conditions (T ratio = −2.644, $p = 0.0289$). No other contrasts resulted as significant. No effect of group was observed ($F_{(1,42)} = 0.237$, $p = 0.6291$). A graphical representation of d'prime scores relative to each condition and group can be found in Fig. 8. In addition, none of the covariates resulted as significant. In order to consider hypomimia data for PD patients, we performed the same analysis only on the PD group. Particularly, we performed a linear mixed effects model where we entered d'prime scores as the dependent variable and the presentation condition as a fixed factor. Moreover, we added age, education, MoCa, and Benton as covariates. In addition, the score relative to the UPDRS hypomimia question was added. It must be noted that we could not obtain this score for all the participants as for some of them, it was impossible for the neurologist to perform the UPDRS due to time constraints. Thus, this analysis was performed on a subsample of 20 patients. Results reveal a main effect of condition ($F_{(2,36)} = 4.085$, $p = 0.0252$). Post hoc tests revealed that faces encoded with a rigid movement were recognized to a poorer extent both when compared to neutral faces (T ratio = −2.514, $p = 0.0299$) and non-emotional expressions (T ratio = 2.436, $p = 0.0299$). Interestingly, the UPDRS hypomimia score also significantly affected d'prime scores ($F_{(1,14)} = 4.73$, $p = 0.0473$). However, no interaction of UPDRS hypomimia with the condition of presentation of stimuli was found ($F_{(2,36)} = 2.11$, $p = 0.1361$). A graphical representation of this effect can be found in Fig. 9. None of the other covariates resulted as significant.

4. General discussion

Parkinson's Disease is a degenerative pathology with unknown aetiology and is the second most frequent neurodegenerative disorder for frequency after Alzheimer disease [59]. It is characterised by motor and non-motor symptoms, among which a difficulty in recognizing emotional facial expressions [11]. An explanation that has been proposed for such deficits postulates that PD affects neural substrates connected to basal ganglia, which in turn prevent patients from correctly recognizing emotional expressions [15,60].

Another explanation is the one given by the embodied cognition theory, which postulates that in order to understand an emotional expression, we simulate it [61].

Following this interpretation, PD patients might not be as good at simulating it due to the presence of hypomimia and facial bradykinesia [20], or to altered functioning of the mirror neuron system [23].

The use of non-emotional facial expressions could help in disentangling between the two main hypotheses, as they share with emotional facial expressions the motor component but not the emotional one. In order to further shed light on this matter, in Experiment 1, we tested non-emotional facial expression recognition in a sample of PD participants compared to healthy controls. By using non-emotional expressions, we aimed to disentangle the contribution of emotion recognition impairment and the contribution of facial simulation of expressions in facial expression recognition. Moreover, to make sure that eventual expression recognition deficits were not due to general cognitive decline or perceptual malfunctioning, we administered a similar test to our participants investigating facial identity recognition.

Deficits in recognizing face identity have been reported in a minority of studies on PD patients [14,17]. We know from face recognition models that we process faces using two separated yet interacting systems: the ventral one, involved in processing structural information, and the dorsal one, involved in processing information related to facial expressions and biological motion [39,40]. Those systems interact in the recognition of face identity. Thus, as PD patients were reported to have deficits in the recognition of facial expressions, we wanted to understand whether they also have difficulties in recognizing identities of faces encoded through an expression. In Experiment 2, we tested a group of PD patients and an HC group in the recognition of the identity of faces encoded through dynamic non-emotional facial expressions, with a rigid movement or as neutral and static.

Results of Experiment 1 revealed that our patients do not show difficulties in recognizing facial expressions or identities compared to the control group.

These results seem to be in line with the study from Derya and colleagues [25], who did not find difficulties with non-emotional expressions in PD compared to HC. It is also in line with a recent study from Kuehne and colleagues [62] finding no effect of facial feedback in emotion recognition in PD patients. This might indicate that PD patients have a stronger deficit in general emotion recognition than in the expressions themselves [12]. This would also be sustained by studies finding emotion processing difficulties also in auditory stimuli, such as deficits in the recognition of emotion from prosody [63,64]. It must be noted that the two explanations for the expression recognition deficit in PD are not mutually exclusive. It might be that a deficit in emotion recognition and problems in expression mimicry interact in causing the deficit of patients with emotional expressions, and the deficit of mimicry itself is not as big to emerge for non-emotional facial expression recognition.

However, it might also be that the stimuli used in the present experiment were not suitable for measuring facial expression recognition. This is because we used static and not dynamic facial stimuli, which have been demonstrated to be more effective [65]. In addition, it would be interesting to enlarge our sample and test for the laterality of the insurgence of the deficit. Indeed, some authors separated their patients based on the laterality of the insurgence of symptoms. They found difficulties with expressions only in the patients with LPD whose insurgence is in the right hemisphere [33]. The right hemisphere is linked to expression processing and biological motion [66], thus it might be that only considering these patients would reveal some interesting results.

Results from Experiment 2 revealed that faces presented through a rigid head movement were more difficult to recognize: however, no group differences were found in this.

These results are coherent with Experiment 1 as our participants did not show difficulties in recognizing non-emotional expressions (Experiment 1), and this did not reflect on identity recognition of faces encoded with non-emotional expressions (Experiment 2), even

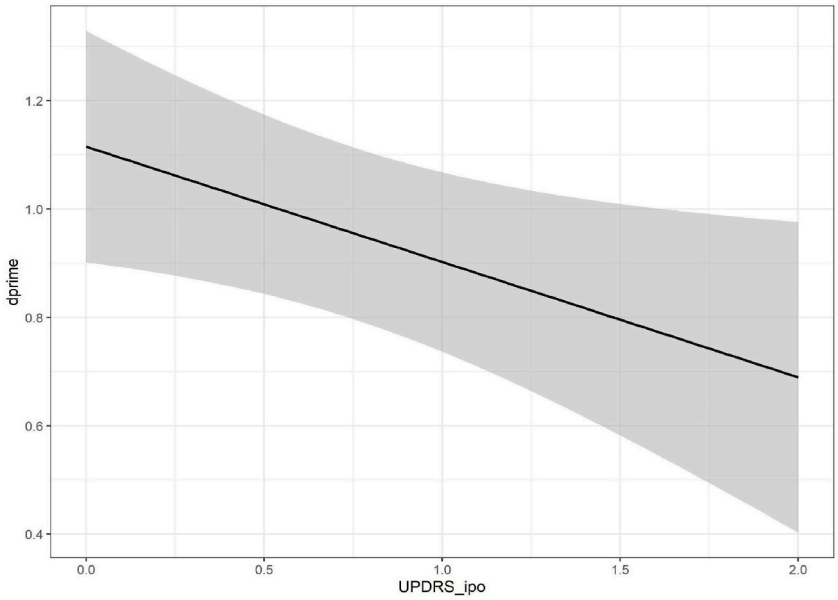

Fig. 9. Graphical representation of the relation between the UPDRS scores in the hypomimia scale and dprime scores for Parkinson's Disease patients. Dprime scores represent the mean dprime scores for all patients.

if in this case stimuli were dynamic. Once again, this result seems to point in the direction of impairment in general emotion processing rather than in the expression processing in PD. As stated above, it might be that there is an influence of simulation of expressions for their understanding, but not as strong for non-emotional expressions. It is interesting to note that in the PD group analyses, scores relative to hypomimia significantly affected d'prime scores relative to all conditions. The higher the hypomimia scores, the lower the d'prime scores. It might be that motor disturbances are an effect of disease severity, and they might be linked to cognitive decline [67, 68]. Thus, it might be that the effect of hypomimia scores that we observed is due to general cognitive impairment rather than specifically linked to hypomimia. In fact, we observed a general effect rather than an effect specific to the expression condition. This would also be coherent with the results obtained by authors finding perceptual impairment in PD [17] as it could be associated with disease severity too.

This study, although relevant, is not without limitations. Firstly, we did use static expressions in Experiment 1. Many other studies used static expressions: however, to be sure that an effect cannot be observed, we could have also used dynamic expressions. Nevertheless, in Experiment 2 we used dynamic stimuli and obtained the same results as Experiment 1. Moreover, in both studies 1 and 2, we did not compare our non-emotional expressions with emotional expressions. This would make our results more solid. Finally, as a face recognition test, we used the BFRT in its original version. A new version was published in 2022 [69]: however, it was published only after data collection began.

In conclusion, from these two studies, it emerged that Parkinson's Disease patients do not show difficulties in recognizing static non-emotional facial expressions compared to Healthy Controls. Moreover, they do not show deficits in the recognition of identities of faces encoded through a dynamic non-emotional expression. These results point into an explanation of the deficit reported for PD in recognizing emotional expressions which is predominantly tied to emotion processing rather than to expression simulation.

Ethics statement

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of University of Milan-Bicocca (protocol number for Experiment 1: 0126263/21; protocol number for Experiment 2: 0041468/20). All participants provided informed consent before participating to the studies.

Data availability statement

Data will be made available on request.

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CRediT authorship contribution statement

Silvia Gobbo: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Elisa Urso:** Writing – review & editing, Data curation, Conceptualization. **Aurora Colombo:** Writing – review & editing, Data curation, Conceptualization. **Matilde Menghini:** Data curation. **Cecilia Perin:** Writing – review & editing, Supervision, Conceptualization. **Ioannis Ugo Isaias:** Writing – review & editing. **Roberta Daini:** Writing – review & editing, Supervision, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e26860.

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