

Intact emotion recognition and experience but dysfunctional emotion regulation in idiopathic Parkinson's disease



Rottraut Ille^{a,*}, Albert Wabnegger^a, Petra Schwingenschuh^b, Petra Katschnig-Winter^b, Mariella Kögl-Wallner^b, Karoline Wenzel^b, Anne Schienle^a

^a Clinical Psychology, University of Graz, Austria

^b Department of Neurology, Medical University of Graz, Auenbruggerplatz 22A, 8036 Graz, Austria

ARTICLE INFO

Article history:

Received 23 June 2015

Received in revised form 15 October 2015

Accepted 4 December 2015

Available online 8 December 2015

Keywords:

Affective traits

Emotion experience

Facial emotion recognition

Emotion regulation

Disease severity

Compensatory mechanisms

Parkinson's disease

ABSTRACT

Background: A specific non-motor impairment in Parkinson's disease (PD) concerns difficulties to accurately identify facial emotions. Findings are numerous but very inconsistent, ranging from general discrimination deficits to problems for specific emotions up to no impairment at all. By contrast, only a few studies exist about emotion experience, altered affective traits and states in PD.

Objective: To investigate the decoding capacity for affective facial expressions, affective experience of emotion-eliciting images and affective personality traits in PD.

Methods: The study sample included 25 patients with mild to moderate symptom intensity and 25 healthy controls (HC) of both sexes. The participants were shown pictures of facial expressions depicting disgust, fear, and anger as well as disgusting and fear-relevant scenes. Additionally, they answered self-report scales for the assessment of affective traits.

Results: PD patients had more problems in controlling anger and disgust feelings than HC. Higher disgust sensitivity in PD was associated with lower functioning in everyday life and lower capacity to recognize angry faces. Furthermore, patients reported less disgust towards poor hygiene and spoiled food and they stated elevated anxiety. However, the clinical group displayed intact facial emotion decoding and emotion experience. Everyday life functionality was lowered in PD and decreased with stronger motor impairment. Furthermore, disease duration was negatively associated to correct classification of angry faces.

Conclusions: Our data indicate that problems with emotion regulation may appear already in earlier disease stages of PD. By contrast, PD patients showed appropriate emotion recognition and experience. However, data also point to a deterioration of emotion recognition capacity with the course of the disease. Compensatory mechanisms in PD patients with less advanced disease are discussed.

© 2015 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

1. Introduction

A clinical diagnosis of Parkinson's disease (PD) is based upon motor symptoms, such as bradykinesia, rigidity, and resting tremor, which usually arise unilaterally, and spread to the opposite side of the body with progression of this neurodegenerative disease. Typical PD symptoms result from cell death in multiple brain regions, particularly brain stem nuclei and dopamine-generating cells in the substantia nigra [1,2]. In addition to classic motor dysfunction PD is characterized by a progressive pattern of neuropsychological impairment including abnormal emotion processing [3].

Numerous studies have been conducted to analyze PD-related capacity to accurately identify emotions in others' facial expressions, but

findings are very inconsistent. General discrimination deficits [4–6], problems for specific emotions [7–12] as well as no impairment at all [13–17] have been reported.

Differences in facial emotion recognition in PD at least partially are a result of moderating factors such as emotion assessment, stage of disease, medication status, or comorbid depression [7,18]. Symptom severity of patients greatly varies between studies, which is correlated with different visual and executive dysfunctions [10]. In the majority of studies patients received dopamine replacement therapy [18]. Only few studies included non-medicated patients, who were in early stages of PD [4,6,12] or withdrawn from their medication during the study [11]. A study by Péron et al. [12] using vocal stimuli have found worse recognition of emotional prosody in PD patients who were 'on' dopamine replacement therapy than those who were 'off'. Generally, comorbid disorders such as depression could have a confounding effect on the decoding of facial affect. In some studies, authors did not exclude patients with mild depressive symptoms, which makes it difficult to

* Corresponding author at: Karl-Franzens-University of Graz, Department of Psychology, Universitätsplatz 2/III, 8010 Graz, Austria.
E-mail address: rottraut.ille@uni-graz.at (R. Ille).

interpret these results. Therefore, when individuals with PD show a deficit in emotion recognition abilities, this may be related to depression.

Impaired decoding of affective facial expressions of disgust, anger and fear has been reported repeatedly for PD [4,8,12] and for these emotions specific recognition deficits were pointed out in several studies [11,19]. Specific emotion recognition deficits were considered as a consequence of dysfunctions in brain regions that are involved in decoding of these facial emotions (e.g., basal ganglia and insula for the decoding of facial disgust) [19].

In contrast to the multitude of studies on facial emotion recognition only a few studies exist on emotional experience and mood patterns in PD. Findings of those studies which combined self-report with electrocortical and startle measures point to a lowered intensity of aversive feelings in PD patients [20,21,22]. In contrast, Dietz et al. [23] observed normal sympathetic arousal in PD patients who viewed affective pictures. Additionally, Vicente et al. [24] found that patients even with advanced PD did not differ from healthy controls in their emotional reactivity to emotion-inducing film excerpts.

Furthermore, information about alterations of affective traits in PD patients is limited. In a review, Poletti and Bonuccelli [25] concluded that PD patients present a personality profile characterized by increasing inflexibility and apathy (e.g., lowered novelty seeking, increased harm avoidance) on the one side and diminished inhibitory control on the other side (see also Weintraub et al. [26]).

The aim of this study was to assess the decoding capacity for the affective facial expressions of fear, disgust and anger, related affective personality traits and affective states of fear and disgust in response to emotion-eliciting images in patients suffering from idiopathic PD. As it is known that IAPS scenes which should induce anger usually produce mixed emotions we did not include investigation of anger state as a target emotion.

We expected lowered recognition capacity for the facial emotions of disgust, fear and anger, lowered emotion experience (disgust, and fear) and decreased trait disgust, anxiety and anger in PD patients. This assumption is based on previous findings regarding emotion recognition deficits and related dysfunctions of the neuronal substrate for disgust, fear and anger processing in PD [11,19,27]. Furthermore, flattened emotional responses and decreased affective traits has been reported in PD [19,20,24]. Findings by Macías et al. [28] displayed lower levels of external expression of anger in PD patients compared to healthy controls. Whereas, disgust proneness (the tendency of a person to experience disgust across different situations) and disgust sensitivity (the tendency of a person to experience one's own disgust feelings as aversive and uncontrollable) in PD has only been investigated by our working group thus far [29].

Additionally, we were interested in the relationship between affective traits (the disposition to respond to specific classes of stimuli in a predetermined, affect-based manner) and affective states (subjective intensity evaluation of the target emotion of an emotion category) in PD. Findings of previous studies have revealed associations between the experience of specific emotions and affective traits in different clinical groups and healthy controls [30,31].

Finally, we planned to assess if the capacity in emotion recognition, emotion experience and affective traits are associated with measures of PD symptom severity and duration. Previous results about connections between disease severity and impaired emotion recognition are inconsistent but have been rather weak [7].

2. Methods and materials

2.1. Participants

Twenty-five symptomatic patients with idiopathic PD (15 males, 10 females) and 25 healthy controls (HC; 15 males, 10 females) participated in the study. All patients were recruited and diagnosed with idiopathic PD by neurologists of the University Hospital in Graz (Austria).

Written informed consent was obtained from each individual. The study was carried out in accordance with the Declaration of Helsinki and had been approved by the ethics committee of the Medical University of Graz.

The groups did not differ in mean age ($M_{PD} = 56.1$ years ($SD = 8.5$), $M_{HC} = 55.4$ ($SD = 8.4$); $t(48) = 0.28$, $p = .778$) and years of education ($M_{PD} = 12.9$ years ($SD = 3.4$), $M_{HC} = 13.2$ ($SD = 3.3$); $t(48) = 0.30$, $p = .769$).

Cognitive performance was assessed by the Test for Early Detection of Dementia (TEDD) [32]. The scores of this scale range between 0 and 50. A score below 35 indicates a tentative dementia diagnosis and was set as an exclusion criterion. The Cronbach's alpha of the TEDD is 0.88. None of the patients showed signs of cognitive impairment ($M_{PD} = 45.1$ ($SD = 3.1$), $M_{HC} = 44.4$ ($SD = 3.2$); $t(48) = 0.67$, $p = .508$). Additionally, PD patients and HC did not differ in their Beck Depression Inventory scores (BDI; $M_{PD} = 6.7$ ($SD = 7.0$), $M_{HC} = 9.1$ ($SD = 6.4$); $t(48) = 0.32$ ($p = .752$) [33].

Daily life functioning was assessed by the 'Functional Assessment Scale' of the Unified Huntington's Disease Rating Scale (UHDRS), asking for (paid) occupation, management of financial affairs, independent housekeeping, body care and mobility [34]. The scale ranges up to a score of 25, with higher scores revealing better functioning. Cronbach's alpha for the functional checklist is 0.95. PD patients showed lower daily life functioning than healthy controls ($M_{PD} = 23.28$ ($SD = 2.44$), $M_{HC} = 24.72$ ($SD = 0.46$); $t(25.69) = 2.90$ ($p = .008$).

Patients' mean motor score assessed by the Unified Parkinson's Disease Rating Scale (UPDRS) was 34.2 ($SD = 8.5$) [35]. Eighteen patients had right body side onset of motor symptoms and 7 had left-side onset. Motor symptoms became manifest at a mean age of 50.1 years ($SD = 8.9$), and at the time of testing the symptom duration was $M = 6.0$ years ($SD = 3.0$) on average. The stage of disease was checked with the Hoehn and Yahr rating score [36]. Twenty-two patients showed stage 2.0, one patient showed stage 2.5, and two patients showed stage 3.0. This indicates mild to moderate symptom intensity. With one exception all patients were treated with L-Dopa and/or a dopamine agonist (pramipexole, ropinirole). The mean total L-Dopa equivalent daily dosage was $M = 459$ mg ($SD = 295.5$) on average. However, medication was discontinued for 10 to 12 h prior to the experiment and was continued only afterwards.

2.2. Questionnaires

All participants answered the following scales:

The Questionnaire for the Assessment of Disgust Proneness (QADP) measures disgust propensity and describes 37 situations, which have to be judged on 5-point scales with regard to the experienced disgust (0, 'not disgusting'; 4, 'very disgusting'). The five subscales are a) death/deformation, b) body secretions, c) spoilage/decay, d) poor hygiene, and e) oral rejection [37]. The Cronbach's alpha of the total scale is .90.

The scale for the Assessment of Disgust Sensitivity (SADS) consists of seven items addressing the appraisal of one's own disgust feelings (e.g., 'Experiencing disgust is stressful for me.') and its control (e.g., 'It embarrasses me when I feel disgusted') [38]. The Cronbach's alpha of the scale is 0.85.

The Beck Depression Inventory (BDI, German version) consists of 21 items rated on 4-point scales [33]. A sum score of 18 or higher indicates clinical relevance. The Cronbach's alpha is 0.88.

The trait scale of the State-Trait Anger Expression Inventory (STAXI; German version) assesses the tendency of a person to experience, express and control anger [39]. The questionnaire consists of four subscales labeled 'trait anger', 'anger suppression' (anger in), 'anger expression' (anger out), and 'anger control'. The 24 items are answered on 4-point scales (1 = almost never, 4 = almost ever). The Cronbach's alpha ranges between .76 und .87.

The trait scale of the State-Trait Anxiety Inventory (STAI) measures the frequency of anxious feelings [40]. The questionnaire consists of 20 items which are answered on a 4-point scale (1 = almost never, 4 = almost ever). The Cronbach's alpha of the scale is .88.

2.3. Stimuli and design (Fig. 1)

The stimulus material consisted of 30 pictures with emotional facial expressions depicting fear (10), anger (10), and disgust (10) from the Karolinska set [41]. Half of the posers were female, and half were male. Also, the participants were presented with a total of 20 affective scenes for the elicitation of fear (10) and disgust (10). Scenes were

taken from the International Affective Picture System (IAPS) [42] or developed by Schienle et al. [43]. Disgust-eliciting pictures included scenes with animals (maggots, burying beetle on carrion, lollipop with cricket and mealworm, an opened mussel, a slug, and earthworms), a dirty toilet, unappetizing food, an eczematous face, and an eczematous bum. The fear-inducing pictures showed threatening situations either through attacks of animals ('dog with its teeth bared', IAPS 1300; gorilla, IAPS 1660; a threatening bear, a tiger with its muzzle opened, and a bird of prey with its catches spread, developed by the authors) or human attacks ('man threatening a woman with a knife', IAPS 6350; 'men with pistol', IAPS 6230; 'war scene', IAPS 6940; 'masked robber', IAPS 6370, and a pursuit scene by night, developed by the authors). Since the

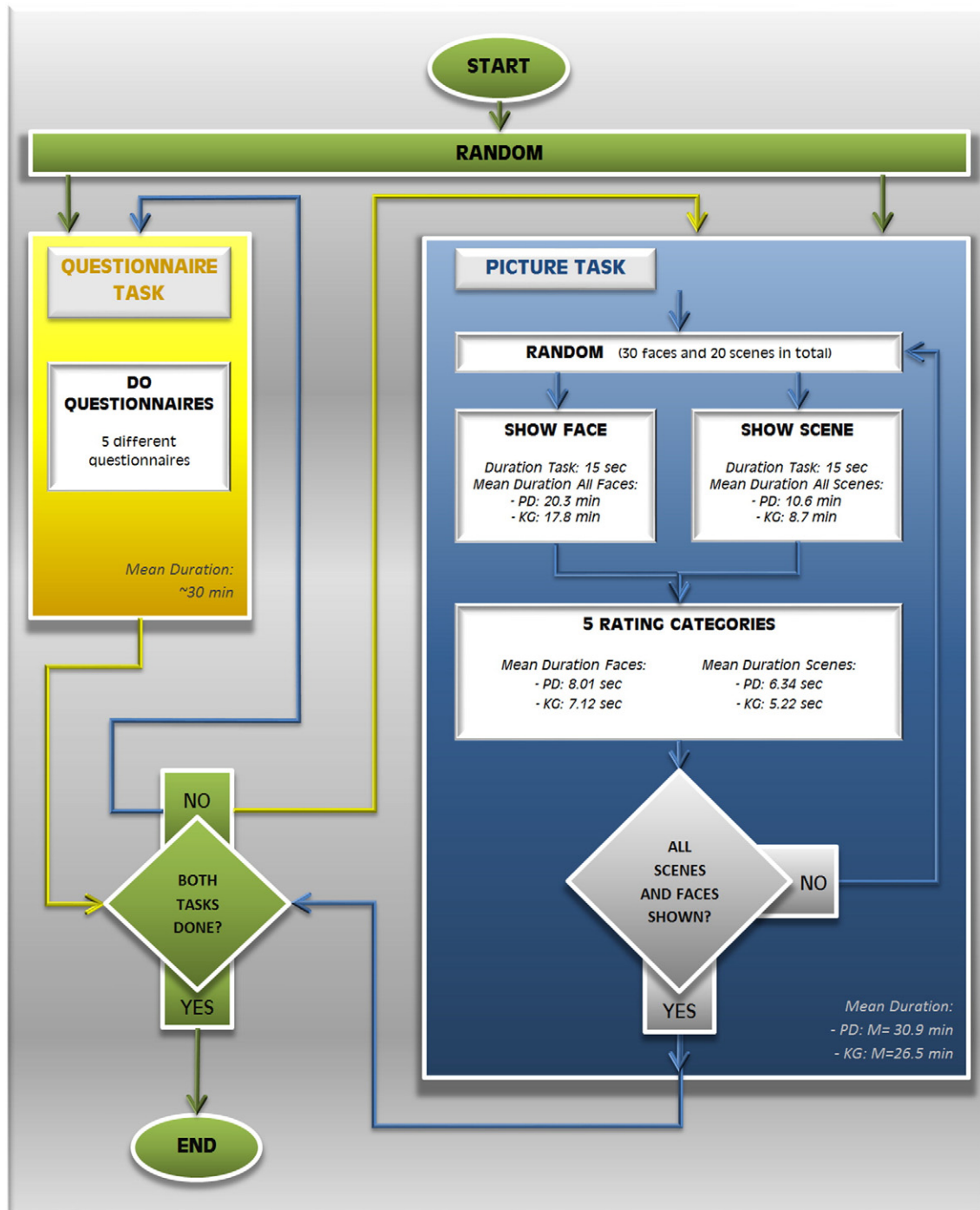


Fig. 1. Design and course of the experiment.

IAPS does not include pictures which reliably induce anger, this category had to be omitted.

Each picture was presented on a computer screen (15 in. diameter) for a maximum of 15 s. The subjects were asked to rate each picture on a 9-point scale within 15 s but could terminate the presentation earlier in order to proceed with the rating. After the rating of a picture the next picture was presented without any delay. For each facial expression (fear, disgust, anger) subjects rated how intense the depicted person had experienced the five basic emotions (happiness, fear, sadness, anger, and disgust; e.g. 'Please indicate how intense the depicted person experienced disgust': 1 = very little; 9 = very intense). For each affective scene (fear, and disgust) subjects rated how intense they had experienced the five basic emotions (happiness, fear, sadness, anger, and disgust; e.g. 'Please indicate how intense you experienced disgust while viewing the picture': 1 = very little, 9 = very intense). To avoid position effects, the order of the two picture tasks (recognition vs. experience), the order of pictures, and the order of basic emotions that had to be rated were randomized.

Summarizing, the features extracted for PD and HC were: disgust propensity, disgust sensitivity, anxiety, anger expression, intensity and recognition accuracy for fearful, disgusted and angry faces, intensity experience for fear- and disgust-eliciting scenes, and anger experience for fear- and disgust-eliciting scenes.

2.4. Statistical analysis

All statistical analyses were carried out using SPSS 22.0 for Windows. We computed Student t-tests in order to compare questionnaire scores between groups (PD, HC).

For the facial emotion recognition task we computed 2×3 ANOVAs with the factors group (PD, HC) and emotion condition (fear, disgust, and anger) in order to compare the target emotion ratings (e.g., intensity of disgust for disgust faces, intensity of anger for anger faces...).

Additionally, 2×5 ANOVAs were conducted separately for each target emotion rating (e.g., disgust ratings across all conditions), with the between-subject factors group (PD, HC) and the repeated measure factor emotion condition (intensity rating of happiness, fear, sadness, disgust, and anger).

Moreover, we conducted Student t-tests in order to compare recognition accuracy between groups. Recognition accuracy was defined as the difference between the perceived intensity of a target emotion and the mean intensity of all non-target emotions for a specific facial expression (e.g., classification accuracy of disgust recognition for a disgust expression = disgust intensity minus mean intensity of non-target emotions [happiness, fear, sadness, anger]).

For the emotion experience task we computed 2×5 ANOVAs separately for fear and disgust ratings with the factors group (PD, HC) and emotion condition (intensity rating of happiness, fear, sadness, disgust, and anger).

Significant effects of ANOVAs were followed up with post-hoc t-tests. As we formulated investigated directional hypotheses, one-sided t-tests were calculated.

Associations between disease severity measures and affective parameters were analyzed with Pearson's correlations.

Alpha level significance was set at .05 for all statistical tests. Cohen's d was used as effect size measure.

3. Results

3.1. Questionnaires (Table 1)

Compared to the HC group PD patients displayed higher disgust sensitivity (SADS; Cohen's $d = 0.49$) and lower anger control (STAXI; $d = 0.78$). Further, PD patients reported lower disgust for 'poor hygiene'

Table 1
Self-report data on affective traits.

	PD	HC	t value	P value
QADP total	1.98 (0.49)	2.07 (0.49)	−0.60	.276
Death	1.29 (0.98)	0.94 (0.91)	1.30	.100
Body secretions	2.38 (0.64)	2.55 (0.71)	−0.92	.180
Spoilage/decay	1.60 (0.69)	1.95 (0.63)	−1.86	.035
Poor hygiene	2.05 (0.64)	2.46 (0.65)	−2.27	.014
Oral rejection	2.60 (0.57)	2.38 (0.76)	1.16	.126
SADS	8.00 (4.51)	5.56 (5.34)	1.75	.043
STAI	39.52 (10.88)	34.60 (8.03)	1.82	.038
STAXI trait	17.40 (3.78)	18.08 (3.98)	−0.62	.269
Anger in	15.20 (4.20)	16.36 (5.63)	−0.83	.207
Anger out	11.28 (2.92)	11.80 (2.75)	−0.65	.260
Anger control	21.84 (5.13)	25.32 (3.65)	−2.76	.004

Notes. QADP: Questionnaire for the Assessment of Disgust Proneness; SADS: Scale for the Assessment of Disgust Sensitivity; STAI: State Trait Anxiety Inventory (trait scale); STAXI: State Trait Anger Expression Inventory (trait scales).

($d = 0.64$) and for 'spoilage/decay' of the QADP ($d = 0.53$). Patients also reported higher trait anxiety than HC (STAI; $d = 0.51$).

3.2. Emotion recognition (Table 2)

3.2.1. Intensity ratings

PD patients rated higher fear intensity for fearful faces than HC ($d = 0.52$). However, groups did not differ in intensities of target emotions for disgusted and angry faces.

3.2.2. Recognition accuracy

PD patients and HC did not differ in classification accuracy for facial disgust, fear and anger.

3.3. Emotion experience (Table 2)

PD patients perceived fear in fear-eliciting scenes slightly more intense than HC ($d = 0.45$) but there was no group difference for disgust.

As no reliable anger-eliciting scenes are provided by IAPS we have compared anger experience in fear- and disgust-eliciting scenes. The PD group reported more intense anger for disgusting scenes compared to HC ($d = 0.58$) (Table 2).

3.4. Correlations (Figs. 2–5)

In PD patients disgust sensitivity (SADS) was negatively correlated with daily life functioning ($r = -0.55$, $p = .006$) and recognition accuracy of angry faces ($r = -0.46$, $p = .022$), and marginally correlated with fear intensity when presented with fear-eliciting scenes ($r = 0.38$, $p = .065$) (Fig. 2).

Table 2
Ratings of affective faces and scenes.

	PD	HC	t value	P value
<i>Affective ratings of facial expressions: intensity</i>				
Fear	6.21 (1.51)	5.31 (1.93)	1.83	.037
Disgust	5.74 (1.70)	5.71 (1.65)	0.06	.476
Anger	7.53 (1.06)	7.59 (0.90)	−0.19	.425
<i>Affective ratings of facial expressions: accuracy</i>				
Fear	3.17 (1.80)	2.42 (1.75)	1.50	.071
Disgust	2.59 (1.80)	2.90 (1.96)	−0.58	.283
Anger	5.03 (1.50)	5.51 (1.45)	−1.14	.130
<i>Affective ratings of scenes: intensity</i>				
Fear	6.64 (1.53)	5.74 (2.39)	1.60	.059
Disgust	6.02 (1.49)	5.41 (1.73)	1.34	.094
Anger (fear-inducing scenes)	3.74 (1.68)	3.54 (1.89)	0.38	.352
Anger (disgusting scenes)	2.85 (1.46)	2.08 (1.17)	2.07	.022

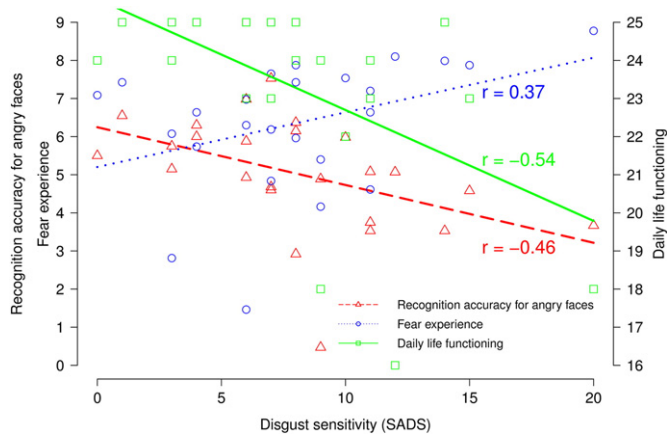


Fig. 2. Correlation between disgust sensitivity (SADS), daily life functioning, recognition accuracy for angry faces and fear experience in PD patients.

Trait disgust (QADP, SADS) was not significantly correlated with disgust experience and disgust recognition in PD patients (all $p > .157$). However, trait anxiety (STAI) was positively correlated to fear intensity ratings for fear-eliciting scenes ($r = 0.49$, $p = .014$) and intensity estimation of fearful faces ($r = 0.52$, $p = .008$). Furthermore, intensity of fear experience for fear-eliciting scenes and fear intensity estimation for fearful faces were positively correlated ($r = 0.61$, $p = .001$) (Fig. 3). Additionally, we found a positive correlation between anger perception in fear-eliciting scenes and trait anger (STAXI; $r = 0.53$, $p = .006$) as well as 'anger in' ($r = 0.42$, $p = .038$) in PD patients.

Concerning associations to disease severity, we found a negative correlation between disease duration and estimated anger intensity for angry faces ($r = -0.43$, $p = .031$) and a slightly negative correlation to STAXI anger control ($r = -0.37$, $p = .069$) (Fig. 4). Motor impairment (UPDRS) was negatively associated with daily life functioning ($r = -0.41$, $p = .042$). Cognitive capacity (TEDD) was positively correlated with recognition accuracy of fearful faces in PD patients ($r = 0.52$, $p = .008$) (Fig. 5).

For controls we found a positive correlation between disgust propensity (mean QADP) and disgust intensity for disgusting scenes ($r = 0.54$, $p = .006$). Anxiety (STAI) was negatively correlated to recognition accuracy of disgusted faces ($r = -0.49$, $p = .013$).

4. Discussion

This study investigated different facets of affectivity and their mutual relations in mild to moderate PD. The patient group reported more problems in controlling anger and disgust feelings than healthy

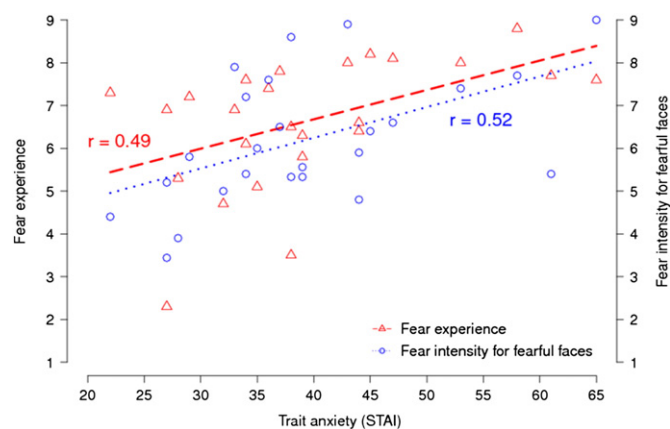


Fig. 3. Correlation between trait anxiety (STAI), intensity of fear experience and fear intensity for fearful faces in PD patients.

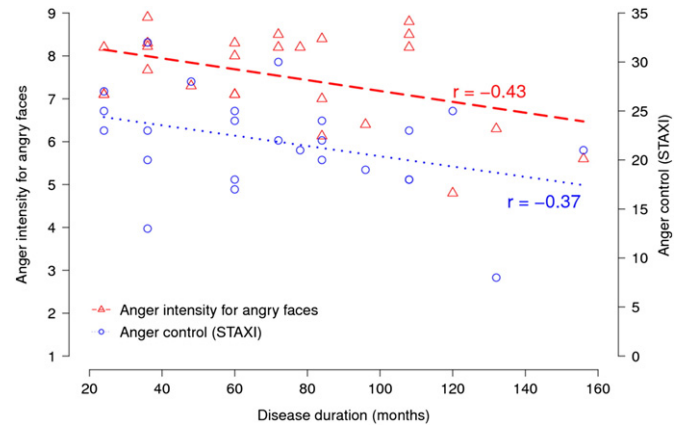


Fig. 4. Correlation between disease duration, anger intensity perception for angry faces and anger control (STAXI) in PD patients.

controls, pointing to deficits in the regulation of negative emotions. Patients with lower control of disgust feelings displayed worse functioning in everyday life, had more problems to accurately recognize angry faces and additionally experienced slightly more fear when presented with fear-eliciting scenes. In a recent study by Ille et al. [29] we could show that PD patients at an earlier disease stage showed reduction of the gray matter volume in the OFC, a brain region which is relevant for emotion control (e.g., Hooker and Knight [44]).

Further, PD patients reported less disgust feelings towards poor hygiene and spoiled food than healthy controls. We did not measure olfactory function of PD patients in this study but olfactory dysfunction is known as one of the early signs in PD showing high prevalence [45,46,47]. Therefore, it seems reasonable that olfactory impairment leads to decreased disgust towards aversive smells like bodily odors or spoiled food. Findings by Hummel et al. [45] showed that PD patients rated unpleasant stimuli smelling like rotten eggs less intense and perceived it as more pleasant than healthy controls. Furthermore, findings by Ille et al. [29] revealed GMV reduction in the central olfactory system (orbitofrontal and piriform cortex) in PD associated with olfactory-related disgust propensity. Also findings in patients with Huntington's disease, which is also associated with olfactory dysfunction, revealed less disgust than healthy controls when presented with disgusting smells [48].

PD patients displayed elevated trait anxiety that was associated with more intense fear experience, and higher trait anger was associated with more intense anger ratings. This finding met our expectations for a positive relationship between affective traits and affective states. However, disgust propensity and disgust sensitivity

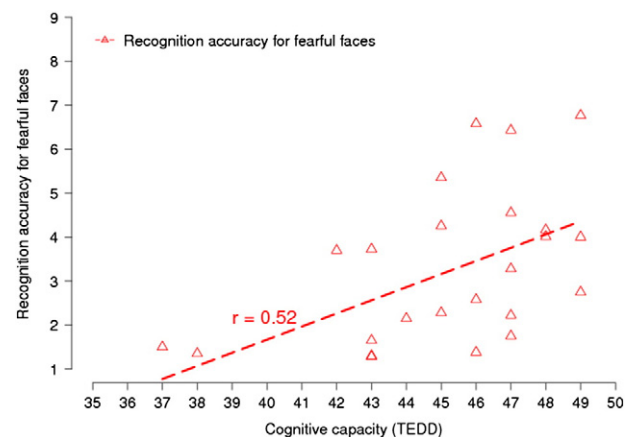


Fig. 5. Correlation between cognitive capacity (TEDD) and recognition accuracy of fearful faces in PD patients.

were not related to disgust experience. We assume that in PD the associations between different aspects of disgust could be altered by olfactory dysfunction and patients' problems in controlling disgust feelings.

The intensity and accuracy ratings for the displayed negative facial emotions indicated no perception deficit in the clinical group. Furthermore, PD patients and HC gave comparable intensity ratings for disgusting and fear-eliciting scenes. However, PD patients showed an anger bias when presented with disgusting scenes which was associated with their trait anger and could be another indication of problems with anger regulation.

Our findings replicate previous reports on intact facial emotion decoding in PD by several authors [13–17]. Cohen et al. [14] who investigated a nondemented and nondepressed patient group could not detect any impairment in recognition accuracy for emotional faces but PD patients showed delayed responses. However, contrary to our study, there was no limitation in response time.

It must be mentioned that our findings are most likely not due to low statistical power since PD patients even were better in recognizing facial fear, an emotion that is difficult to decode.

One argument for intact emotion decoding would be that our patient group was not afflicted by depressive symptoms or cognitive impairment. However, several studies investigating non-depressed and non-demented PD patients have reported impaired facial emotion recognition [5,8].

Furthermore, the medication had been discontinued for 12 h prior to the investigation. It might be expected that interrupting dopatherapy is a disadvantage for emotion processing, but Péron, et al. [12] assumed a potentially adverse effect of dopaminergic therapy on affective abilities in earlier stages of the disease (see also Delaveau et al. [49]).

One further explanation for unimpaired facial emotion recognition in PD could be due to compensatory mechanisms as long as the disease is less advanced. Findings by Anders et al. [50] who investigated positive facial expressions in Parkin mutation carriers revealed that compensatory (increased) activity in the ventrolateral premotor cortex during emotion processing can diminish recognition impairments. Furthermore, Wieser et al. [16] reported that early visual discrimination of facial expressions was diminished on electrocortical level in PD, whereas patients showed no impairments in emotion recognition as measured by affective ratings. The authors suggest that the absence of behavioral impairment may point to compensatory strategies by intact pathways, e.g., premotor, somatosensory and prefrontal areas.

However, it is not known until now, which factors will promote compensatory mechanisms. Previous findings reveal no strong association between disease severity and emotion decoding capacity. In our study disease duration was negatively associated to correct classification of angry faces which could indicate that compensatory mechanisms work less well with disease progression.

Compensatory mechanisms could also be adjusted to olfactory deficits in PD. Recent findings by Lemogne et al. [51] revealed decreased error rates for facial fear and disgust in patients with long-lasting anosmia. The authors suggested that anosmic patients may compensate their inability to detect environmental hazards through olfaction by an increased ability to detect fear or disgust as facially expressed by others.

Simulation theories could also provide an explanation for intact emotion capacity of facial emotion decoding in PD. Simulation approaches assume that the ability to recognize the emotions expressed by other individuals relies, at least in part, on processes that internally simulate the same emotional state in themselves [52,53]. Thus, if emotion experience is unimpaired in PD this could be the substrate for appropriate decoding of facial emotions.

However, emotion recognition and experience were correlated only for fear in PD patients, while for disgust and anger there was no relationship. This may indicate that emotion experience and recognition of facial emotions are processed differently. Presumably, also further

information processing mechanisms will be used for emotion decoding, e.g., cognitive processing of facial features [54].

Intact emotion recognition and experience in PD patients are also in concordance with results by Wabnegger et al. [17] and Ille et al. [29], as PD patients in this study displayed no alterations of regions that are typically involved in emotion experience (e.g., amygdala). Therefore, degenerations of these regions can appear at a more progressive disease stage.

Our patient group revealed lowered daily life functionality that was negatively correlated to motor impairment. Moreover, the capacity to accurately recognize angry faces was negatively related to disease duration, and anger control slightly worsened with course of the disease. However, there was no relationship between motor impairment in PD patients and their emotion recognition capacity. Previous findings on the role of motor impairment in emotion recognition are inconsistent, but effects seem to be rather slight [7,15]. Additionally, in the patient group a lower cognitive capacity was associated with poorer decoding of fearful faces. This indicates that with disease progression not only a worsening of everyday life functions but also of social capacity must be considered.

In conclusion, PD patients reported more problems with control of disgust and anger feelings. Lowered control of disgust feelings was associated with inferior daily life and social functionality, and anger control slightly worsened with disease duration. However, it must be mentioned that further studies will be necessary for estimating emotion regulation in PD which should include experimental and imaging methods.

Additionally, the PD group displayed lower disgust towards aversive smells like bodily odors or spoiled food. This could be associated with olfactory impairment, one of the early signs in PD. However, PD patients displayed unimpaired facial emotion decoding and emotion experience. Nevertheless, data also suggest a deterioration of emotion recognition capacity with the course of the disease. Therefore, it can be assumed that PD patients use compensatory mechanisms as long as the disease is less advanced. Thus, PD patients without dementia and depression may yield appropriate emotion recognition and experience in earlier disease stages. By contrast, problems with emotion regulation can appear already earlier in the course of the disease.

Acknowledgments

This study was supported by the Austrian Science Fund (FWF), project number P 23258-B18.

References

- [1] A.B. Nelson, A.C. Kreitzer, Reassessing models of basal ganglia function and dysfunction, *Annu. Rev. Neurosci.* 37 (2014) 117–135.
- [2] T. Wu, M. Hallett, The cerebellum in Parkinson's disease, *Brain* 136 (2013) 696–709.
- [3] J. Péron, T. Dondaine, F. Le Jeune, D. Grandjean, M. Vérin, Emotional processing in Parkinson's disease: a systematic review, *Mov. Disord.* 27 (2012) 186–199.
- [4] K. Dujardin, S. Blairy, L. Defebvre, S. Duhem, Y. Noël, U. Hess, et al., Deficits in decoding emotional facial expressions in Parkinson's disease, *Neuropsychologia* 42 (2004) 239–250.
- [5] N. Ibarretxe-Bilbao, C. Junque, E. Tolosa, M.J. Martí, F. Valldeoriola, N. Bargallo, M. Zarei, Neuroanatomical correlates of impaired decision-making and facial emotion recognition in early Parkinson's disease, *Eur. J. Neurosci.* 30 (2009) 1162–1171.
- [6] R. Sprengelmeyer, A.W. Young, K. Mahn, U. Schroeder, D. Woitalla, T. Büttner, Facial expression recognition in people with medicated and unmedicated Parkinson's disease, *Neuropsychologia* 41 (2003) 1047–1057.
- [7] F. Assogna, F.E. Pontieri, C. Caltagirone, G. Spalletta, The recognition of facial emotion expressions in Parkinson's disease, *Eur. Neuropsychopharmacol.* 18 (2008) 835–848.
- [8] H.C. Baggio, B. Segura, N. Ibarretxe-Bilbao, F. Valldeoriola, M.J. Martí, Y. Compta, et al., Structural correlates of facial emotion recognition deficits in Parkinson's disease patients, *Neuropsychologia* 50 (2012) 2121–2128.
- [9] U.S. Clark, S. Neargard, A. Cronin-Golomb, Specific impairments in the recognition of emotional facial expressions in Parkinson's disease, *Neuropsychologia* 46 (2008) 2300–2309.
- [10] G. Hipp, N. Diederich, V. Piera, M. Vaillant, Primary vision and facial emotion recognition in early Parkinson's disease, *J. Neurol. Sci.* 338 (2014) 178–182.

- [11] A.D. Lawrence, I.K. Goerendt, D.J. Brooks, Impaired recognition of facial expressions of anger in Parkinson's disease patients acutely withdrawn from dopamine replacement therapy, *Neuropsychologia* 45 (2007) 65–74.
- [12] J. Péron, D. Grandjean, S. Drapier, M. Vérin, Effect of dopamine therapy on nonverbal affect burst recognition in Parkinson's disease, *PLoS One* 9 (2014), e90092.
- [13] R.A. Adolphs, R. Schul, D. Tranel, Intact recognition of facial emotion in Parkinson's disease, *Neuropsychology* 12 (1998) 253–258.
- [14] H. Cohen, M.H. Gagné, U. Hess, E. Pourcher, Emotion and object processing in Parkinson's disease, *Brain Cogn.* 72 (2010) 457–463.
- [15] M.D. Pell, C.L. Leonard, Facial expression decoding in early Parkinson's disease, *Cogn. Brain Res.* 23 (2005) 327–340.
- [16] M.J. Wieser, E. Klupp, P. Weyers, P. Pauli, D. Weise, D. Zeller, et al., Reduced early visual emotion discrimination as an index of diminished emotion processing in Parkinson's disease? — evidence from event-related brain potentials, *Cortex* 48 (2012) 1207–1217.
- [17] A. Wabnegger, R. Ille, P. Schwingenschuh, P. Katschnig-Winter, M. Kögl-Wallner, K. Wenzel, A. Schienle, Facial emotion recognition in Parkinson's disease: an fMRI investigation, *PLoS One* 8 (2015), e0136110.
- [18] H.M. Gray, L. Tickle-Degnen, A meta-analysis of performance on emotion recognition tasks in Parkinson's disease, *Neuropsychology* 24 (2010) 176–191.
- [19] A. Suzuki, T. Hoshino, K. Shigemasa, M. Kawamura, Disgust-specific impairment of facial expression recognition in Parkinson's disease, *Brain* 129 (2006) 707–717.
- [20] D. Bowers, K. Miller, A. Mikos, L. Kirsch-Darrow, U. Springer, H. Fernandez, et al., Startling facts about emotion in Parkinson's disease: blunted reactivity to aversive stimuli, *Brain* 129 (2006) 3356–3365.
- [21] J. Dietz, M.M. Bradley, J. Jones, M.S. Okun, W.M. Perlstein, D. Bowers, The late positive potential, emotion and apathy in Parkinson's disease, *Neuropsychologia* 51 (2013) 960–966.
- [22] M.J. Wieser, A. Mühlberger, G.W. Alpers, M. Macht, H. Ellgring, P. Pauli, Emotion processing in Parkinson's disease: dissociation between early neuronal processing and explicit ratings, *J. Clin. Neurophysiol.* 117 (2006) 94–102.
- [23] J. Dietz, M.M. Bradley, M.S. Okun, D. Bowers, Emotion and ocular responses in Parkinson's disease, *Neuropsychologia* 49 (2011) 3247–3253.
- [24] S. Vicente, J. Péron, I. Biseul, S. Ory, P. Philippot, S. Drapier, et al., Subjective emotional experience at different stages of Parkinson's disease, *J. Neurol. Sci.* 310 (2011) 241–247.
- [25] M. Poletti, U. Bonuccelli, Personality traits in patients with Parkinson's disease: assessment and clinical implications, *Neurology* 259 (2012) 1029–1038.
- [26] D. Weintraub, A.S. David, A.H. Evans, J.E. Grant, M. Stacy, Clinical spectrum of impulse control disorders in Parkinson's disease, *Mov. Disord.* 30 (2015) 121–127.
- [27] T.P. Bouchard, N. Malykhin, W.R.W. Martin, C.C. Hanstock, D.J. Emery, N.J. Fisher, et al., Age and dementia-associated atrophy predominates in the hippocampal head and amygdala in Parkinson's disease, *Neurobiol. Aging* 29 (2008) 1027–1039.
- [28] Y. Macías, J. Benito-León, E.D. Louis, A. Cano-Vindel, Anger in Parkinson's disease: a case-control study, *Mov. Disord.* 23 (2008) 195–199.
- [29] R. Ille, A. Wabnegger, P. Schwingenschuh, P. Katschnig-Winter, M. Kögl-Wallner, K. Wenzel, A. Schienle, Role of disgust proneness in Parkinson's disease: a voxel-based morphometry study, *J. Int. Neuropsychol. Soc.* 24 (2015) 1–4.
- [30] R. Ille, A.K. Holl, H.P. Kapfhammer, K. Reisinger, A. Schäfer, A. Schienle, Emotion recognition and experience in Huntington's disease: is there a differential impairment? *Psychiatry Res.* 188 (2011) 377–382.
- [31] A. Schienle, A. Haas-Krammer, H. Schögl, H.P. Kapfhammer, R. Ille, Altered state and trait disgust in borderline personality disorder, *J. Nerv. Ment. Dis.* 201 (2013) 105–108.
- [32] R. Ihl, B. Grass-Kapanke, P. Lahrem, J. Brinkmeyer, S. Fischer, N. Gaab, Development and validation of a test for early diagnosis of dementia with differentiation from depression TEDD, *Fortschr. Neurol. Psychiatr.* 68 (2000) 413–422.
- [33] M. Hautzinger, M. Bailer, H. Worall, F. Keller, Beck-Depressionsinventar BDI. Bearbeitung der deutschen Ausgabe. Testhandbuch. Huber, Bern, 1994 (1994).
- [34] Huntington Study Group, Unified Huntington's disease rating scale: reliability and consistency, *Mov. Disord.* 11 (1996) 136–142.
- [35] S. Fahn, R.L. Elton, members of the UPDRS Development Committee, C.D. Marsden, D.B. Calne, M. Goldstein, The unified parkinson's disease rating scale, in: S. Fahn (Ed.), *Recent Developments in Parkinson's Disease*, Macmillan Healthcare Information, New York, Florham Park, 1987.
- [36] M. Hoehn, M. Yahr, Parkinsonism: onset, progression and mortality, *Neurology* 17 (1967) 427–442.
- [37] A. Schienle, B. Walter, R. Stark, D. Vaitl, A questionnaire for the assessment of disgust sensitivity, *Z. Klin. Psychol. Psychother.* 31 (2002) 110–120.
- [38] A. Schienle, G. Dietmaier, R. Ille, V. Leutgeb, A scale for assessing disgust sensitivity (SADS), *Z. Klin. Psychol. Psychother.* 39 (2010) 80–86.
- [39] P. Schwenkmezger, V. Hodapp, C.D. Spielberger, State-Trait Anger Expression Inventory (STAXI), Huber, Bern, 1992.
- [40] L. Laux, P. Glanzmann, C.D. Spielberger, State-Trait Anxiety Inventory (STAI), Beltz Testgesellschaft, Weinheim, 1981.
- [41] D. Lundquist, A. Flykt, A. Öhman, The Karolinska Directed Emotional Faces of Emotion and Attention, Department of Neurosciences, Karolinska Hospital, Lawrence, Stockholm, 1998.
- [42] P.J. Lang, M. Bradley, B. Cuthbert, International Affective Picture System, Center for Research in Psychophysiology, University of Florida, Gainesville, Florida, 2001.
- [43] A. Schienle, R. Stark, B. Walter, C. Blecker, U. Ott, G. Sammer, The insula is not specifically involved in disgust processing: an fMRI study, *Neuroreport* 13 (2002) 2023–2026.
- [44] C.I. Hooker, R.T. Knight, The role of lateral orbitofrontal cortex in the inhibitory control of emotion, in: D. Zald, S.L. Rauch (Eds.), *The Orbitofrontal Cortex*, OUP, Oxford 2006, pp. 1–22.
- [45] T. Hummel, K. Fließbach, M. Abele, T. Okulla, J. Reden, H. Reichmann, et al., Olfactory fMRI in patients with Parkinson's disease, *Front. Integr. Neurosci.* 28 (2010) 125.
- [46] H. Braak, E. Ghebremedhin, U. Rüb, H. Bratzke, K. Del Tredici, Stages in the development of Parkinson's disease-related pathology, *Cell Tissue Res.* 318 (1) (2004) 121–134.
- [47] A. Haehner, T. Hummel, H. Reichmann, Olfactory loss in Parkinson's disease, *Parkinsons Dis.* 2011 (2011) 450939.
- [48] I.J. Mitchell, H. Heims, E.A. Neville, H. Rickards, Huntington's disease patients show impaired perception of disgust in the gustatory and olfactory modalities, *J. Neuropsychiatr. Clin. Neurosci.* 17 (1) (2005) 119–121.
- [49] P. Delaveau, P. Salgado-Pineda, T. Witjas, J. Micallef-Roll, E. Fakra, J.P. Azulay, et al., Dopaminergic modulation of amygdala activity during emotion recognition in patients with Parkinson disease, *J. Clin. Psychopharmacol.* 29 (2009) 548–554.
- [50] S. Anders, B. Sack, A. Pohl, T. Münte, P. Pramstaller, C. Klein, et al., Compensatory premotor activity during affective face processing in subclinical carriers of a single mutant Parkin allele, *Brain* 135 (2012) 1128–1140.
- [51] C. Lemogne, J. Smadja, e.-H. Zerdazi, Y. Soudry, M. Robin, S. Berthoz, et al., Congenital anosmia and emotion recognition: a case-control study, *Neuropsychologia* 72 (2015) 52–58.
- [52] A.S. Heberlein, A.P. Atkinson, Neuroscientific evidence for simulation and shared substrates in emotion recognition: beyond faces, *Emot. Rev.* 1 (2009) 162–177.
- [53] A.I. Goldman, C.S. Sripada, Simulationist models of facebased emotion recognition, *Cognition* 94 (2005) 193–213.
- [54] D.V. Becker, D.T. Kenrick, S.L. Neuberg, K.C. Blackwell, D.M. Smith, The confounded nature of angry men and happy women, *J. Pers. Soc. Psychol.* 92 (2007) 179–190.