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Social Cognition and Mild Cognitive Impairment in Mid-Stage Parkinson's Disease

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Abstract: Mild cognitive impairment (MCI) is a relevant non-motor feature in Parkinson's disease (PD). Social cognition (SC) is a cognitive domain that refers to the ability to decode others' intentions and to guide behavior in social contexts. We aimed to compare SC performance in mid-stage PD patients compared to a healthy population and according to their cognitive state. Fifty-two PD patients were classified as being cognitively normal (PD-CN) or having mild cognitive impairment (PD-MCI) following the Movement Disorder Society (MDS) Level II criteria. SC assessment included facial emotion recognition (FER), affective and cognitive theory of mind (ToM), and self-monitoring (RSMS test). Twenty-seven age-matched healthy controls (HC) were enrolled. PD-MCI patients scored worse than HC on affective and cognitive ToM task scores. Only cognitive ToM scores were significantly lower when compared with the PD-MCI and PD-CN groups. We found no differences in FER or self-monitoring performance. There were significant correlations between cognitive ToM and executive functions, memory, language, and attention, whereas FER and affective ToM correlated with memory. Our findings indicates that SC is normal in cognitively unimpaired and non-depressed mid-stage PD patients, whereas a decline in affective and cognitive ToM is linked to the presence of MCI.

Keywords: theory of mind; social cognition; Parkinson's disease; mild cognitive impairment

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1. Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disorder after Alzheimer disease (AD). In 2017, the Global Burden of Disease (GBD) study documented 1.02 million incident cases of Parkinson's disease [1]. The cardinal motor features are bradykinesia, tremor, and rigidity with a typical response to dopaminergic treatment [2]. In some cases, patients may develop motor fluctuations, with the clinical effect of medication diminishing over time, leading to an OFF state and a decline in motor function. Non-motor manifestations such as cognition may also fluctuate, emphasizing dopamine's crucial role in cognitive functions, as reported in the literature [3]. However, PD also comprises a heterogeneous spectrum of non-motor symptoms that contribute greatly to overall disease burden [4]. Cognitive decline is one of the most studied non-motor aspects of PD. Prevalence studies have found that mild cognitive impairment (MCI) is present even in newly

diagnosed PD patients [5,6], and dementia is common, especially in late stages. It has been found that 83% of patients are affected after 20 years of disease [7]. This makes dementia one of the most important non-motor manifestations of PD, with an important impact on the quality of life [8,9].

MCI has been recognized as an intermediate phase between normal cognition and dementia in the general population [10] as well as in PD [11,12]. Its relevance is such that the Movement Disorder Society (MDS) appointed a task force to outline the diagnostic criteria for MCI in Parkinson's disease, which may impact the following cognitive domains: attention and working memory, executive functions, visuospatial skills, memory, and language [11]. Its cognitive correlates have been extensively examined as a risk factor for PD dementia (PDD) [13,14] and have shown a moderate but robust effect on conversion of PD-MCI to PDD [15].

Social cognition (SC) is a domain that can also be affected in PD patients but has been less frequently studied [16]. It refers to the ability to decode others' intentions and behaviors and use those social cues to guide behavior in social contexts. This cognitive domain mainly comprises three clusters: facial emotion recognition (FER), theory of mind (ToM, also known as social understanding), and social decision making [17]. ToM refers to our ability to comprehend our own and others' cognitive states, such as thoughts, beliefs, and intentions (referred to as "cognitive ToM"), as well as affective states like emotions or feelings ("affective ToM"), and to anticipate others' actions based on these mental representations [18]. In addition, we are interested in other aspects of SC, such as self-monitoring, the ability to work out social and emotional cues, whose impairment can be observed in other neurodegenerative diseases such as frontotemporal dementia [19]. Self-monitoring in PD has been investigated in only one study, regardless of cognitive state [20]. SC impairment has been described in the early stages of PD in numerous studies [21–23].

Most of the studies about SC in PD have been focused on non-demented patients; therefore, they do not differentiate among cognitively normal individuals and those with MCI. Two recent studies in patients with PD-MCI showed poorer scores in SC tasks, such as FER tasks and ToM, than in PD patients with preserved cognition [24,25]. By contrast, a recent study showed FER and ToM impairment often occurred in the absence of impairment in any other cognitive domain [26].

Moreover, depression has been related to social cognitive deficits in the general population [27,28] and in PD patients [29]. In addition, higher levels of depression have been associated with MCI in PD [30,31]. However, previous studies on PD-MCI patients did not consider it as a potential confounder of SC performance.

In this study, we aimed to compare SC performance in mid-stage PD patients compared to healthy population and according to their cognitive state and to further study the relationship between SC and other cognitive domains.

2. Materials and Methods

2.1. Subjects

We performed an observational, case–control, cross-sectional study. Fifty-two patients with PD diagnosed according to the United Kingdom Parkinson's Disease Society Brain Bank Criteria [32] were recruited by convenience sampling at HM CINAC, Hospital Universitario HM Puerta del Sur, from May 2017 to April 2021 to undergo social, neuropsychological, and neuropsychiatric evaluation. Patients had a disease duration of less than 10 years and an age of disease onset greater than 50 years. Twenty-seven age- and gender-matched healthy controls (HC) were also enrolled for statistical comparison. Exclusion criteria for all participants were coexistence of severe cerebrovascular disease, metabolic disease, active oncologic process, severe depression (score over 20) at baseline measured by Geriatric Depression Scale (GDS-30) [33], presence of alterations in magnetic resonance imaging that indicate other causes of cognitive impairment, and any previous neurosurgical intervention. Subjects with dementia were excluded following the Movement Disorder Society criteria [11]. The study received the HM Puerta del Sur Ethics Committee

approval (16.09.0942E1-GH), and all subjects provided signed informed-consent prior to investigation.

2.2. Clinical Assessment

Demographic and clinical data included age, gender, disease duration, educational level, and disease severity evaluated using the MDS-Unified Parkinson's Disease Rating Scale (MDS-UPDRS) part III (motor score) and levodopa equivalent daily dose (LEDD). The LEDD condenses the antiparkinsonian medication into a single number according to a standardized method [34]. The GDS and the Starkstein apathy scale (SAS) were performed in all patients. The entire evaluation was performed during the on-medication state so that we avoid clinical fluctuations that could affect comparisons between patients. The battery of tests was performed in one session, or two in case of signs of fatigue, and the order of the tests was randomized.

2.3. Neuropsychological Evaluation

In addition to SC assessment, a neuropsychological battery was performed, including the following tests: attention/working memory (digit span forward and backward [35], a test that requires the patient to repeat numbers in either identical or reverse sequence as read by the evaluator; Trail Making Test A (TMTA) [36], which requires rapid linkage of fifteen sequentially numbered circles); executive function (Stroop test inhibition time (TMTB) [36], a similar test that requires alternating between connecting numbers and letters in order; and phonemic fluency [37], where the subject is asked to name as many items as he or she can recall for a specific phoneme (i.e., /p/)); language (Boston Naming Test [38], a test in which the examiner presents a series of black and white line drawings, enabling the participant to identify and name each item; semantic fluency [37], which asks the subject to name as many items as he or she can recall in a category fluency (i.e., animals)); memory (Consortium to Establish a Registry for Alzheimer's Disease (CERAD) [39], delayed recall/recognition for verbal memory, a test that consists of three learning trials, a delayed recall trial and a recognition trial; and Wechsler Memory Scale for visual memory [40], designed to assess the capacity for recalling designs from memory and accurately reproducing and recognizing them); visuospatial function (Judgment of Line Orientation [JLO] [41], a test that measures accuracy of angular orientation through assessments of a pair of angled lines, based on their visual congruence; Visual Object and Space Perception Battery for visuospatial domain (VOSP) [42], which evaluates visuospatial function focusing on one component of visual perception, while minimizing other cognitive functions; and a copy of the Rey–Osterrieth complex figure (ROCF) [43], which evaluates visuospatial constructional ability, wherein the patient is instructed to copy a complex geometric figure onto a blank sheet of paper.). Patients were classified as cognitively normal (PD-CN) or as having mild cognitive impairment (PD-MCI) following the MDS Level II criteria [11], which implies comprehensive neuropsychological testing with two tests for each of the five cognitive domains. Impairment was required to be present on at least two tests in one or different cognitive domains [11].

2.4. Social Cognition Assessment

SC was evaluated during the on-medication state and included measures of FER, affective ToM, cognitive ToM, and a self-monitoring test. In order to rule out difficulties in face perception, we first used the Benton Facial Recognition Test (BFRT) as a potential prerequisite for the successful interpretation of emotional expressions. In this test, the participant was required to match a target face with either one face from the same viewpoint (6 items) or three out of six faces with varying viewpoints and lighting conditions (16 items). The maximum score on the task is 54, and a score less than or equal to 38 implies impairment of individual face-matching ability [44]. For FER assessment, we used the Karolinska Directed Emotional Faces (KDEF) test [45], which comprised 64 chosen photographs, 8 for each emotion (anger, disgust, fear, happiness, sadness, surprise), and 16 depicting neutral

faces (no expression) from the electronic database [46,47]. Participants had to identify which emotion was expressed in each photograph, and each correct response scored one point, with a maximum of 64 points. Affective ToM was assessed with the revised version of the Reading the Mind in The Eyes Test (RMET) [48–50] that consists of 36 photographs of the eye region of male and female subjects. The participants were asked to choose which of the four options better described the intentions and emotions of the subjects in the photographs. As in the previous test, each photograph scored one point, with a maximum of 36. For exploring cognitive ToM we used the Theory of Mind Picture Stories Task (ToM stories) [51] that uses a cartoon picture story consisting of four pictures that comprise a first-order false belief, a second-order false belief, and a tactical deception. The participants had to order the four pictures in the correct chronological sequence. If they failed, a correction was made, and the story was correctly presented before ToM testing. Participants were then asked about the story with questions about the ability to infer the mental states of the characters in the story. Scores were classified into a maximum global rate of 59 points, a correct sequencing rate (maximum score of 36), and a correct questionnaire score (maximum score of 23); two points were given to the first and last cards and 1 point to the rest and for each question. Finally, the Revised Self-Monitoring Scale (RSMS) [52] consists of 13 items to be answered by the informant, covering the capacity to regulate one's (patient) behavior in a social context. The answers are on a 6-point Likert-type scale, and the patients are more likely to adapt to a social context when the score is higher.

2.5. Statistical Analysis

Clinical and neuropsychological variables were compared between the three groups: HC, PD-CN, and PD-MCI. Normally distributed variables were compared by ANOVA followed by Bonferroni post hoc correction; variables with non-parametric distribution were studied by the Kruskal–Wallis test followed by Dwass–Steel–Critchlow–Fligner pairwise comparisons post hoc test. In the case of categorical variables, the Chi-square test was applied.

Finally, Pearson correlation test or Spearman rank correlation coefficients (according to normality of distribution) and corresponding 95% confidence intervals (CIs) were performed, followed by Bonferroni correction, to assess the correlation between SC variables and neuropsychiatric and neuropsychological measures. Statistical analyses were performed with STATA software version 17 (STATA Corporation, College Station, TX, USA).

3. Results

3.1. Demographic, Clinical, and Neuropsychiatric Assessment

As a remainder, fifty-two non-demented PD patients and twenty-seven HC were enrolled in this study. Six patients (2 PD-CN and 4 PD-MCI) were removed for FER analysis because they scored $\leq$38 on the BFRT. Following the MDS PD-MCI diagnosis Level II criteria, thirty-three patients were classified as PD-CN, while nineteen were classified as PD-MCI. The mean age of the participants was 67.2 years (SD = 5.04), with no significant differences between groups. PD-MCI patients only differed from HC subjects in apathy, showing higher scores. PD-MCI patients also scored worse than PD-CN patients in apathy and in the MDS-UPDRS motor score ($p < 0.05$). There were no other significant differences in demographic or neuropsychiatric variables between groups. The demographic results are shown in Table 1.

Table 1. Demographic, clinical, and neuropsychiatric variables.

	PD-CN M (SD) n = 33	PD-DCL M (SD) n = 19	HC M (SD) n = 27	X^2, F or t	p Value
Age, years [c]	68.0 (5.4)	67.4 (5.1)	66.4 (4.6)	2.65	0.27
Females, *n* (%) [e]	12 (36.4)	3 (15.8)	11 (40.7)	3.45	0.17
Education level, years [c]	14.7 (4.8)	12.2 (4.9)	14.7 (3.9)	4.48	0.11
Disease duration, years [d]	5.2 (2.1)	6.4 (1.9)	n/a	3.92	0.053
LEDD [d]	573 (316)	687 (184)	n/a	1.87	0.11
MDS-UPDRS III [d]	17.9 (7.6)	24.6 (7.5)	n/a	8.73	0.005 [a]
GDS-30 [c]	7.2 (5.9)	8.9 (5.1)	5.8 (4.4)	3.63	0.16
SAS [c]	11.0 (6.4)	16.1 (7.7)	6.3 (4.4)	12.9	0.002 [a,b]

Note: [a] Significant differences between PD_MCI and PD_CN in post hoc test. [b] Significant differences between PD_MCI and HC in post hoc test. [c] ANOVA test used. [d] *t*-test used. [e] Chi-square used. PD-CN = Parkinson's disease cognitively normal; PD-MCI = Parkinson's disease with mild cognitive impairment; HC = healthy control; MDS-UPDRS: Movement Disorder Society-Sponsored Revision of the Unified Parkinson's Disease Rating Scale; LEDD: Levodopa equivalent daily dose; GDS = Geriatric Depression Scale; SAS = Starkstein Apathy Scale.

3.2. Social Cognition Assessment According to Classification Groups

A Kruskal–Wallis test was performed to determine if there were statistically significant differences among diagnostic groups on SC tests (Table 2). In case of cognitive ToM, we found significant differences in ToM stories total scores (F = 6.85, $p < 0.05$) between PD-MCI groups (M = 44.5, SD = 10.76) compared to PD-CN (M = 50.5, SD = 9.07) and HC (M = 51.73, SD = 6.12). When we analyzed the ToM stories sequencing and questionnaire scores separately, only questionnaire scores remained statistically significant (F = 11.14, $p < 0.05$) for the same groups.

Table 2. Social cognition assessment according to classification groups.

	PD-CN M (SD) n = 33	PD-MCI M (SD) n = 19	HC M (SD) n = 27	X^2	p Value
RMET	20.2 (4.7)	18.4 (3.2)	22.6 (4.6)	8.43	0.02 [b]
ToM Stories, total	50.5 (9.1)	43.5 (10.8)	51.4 (6.1)	6.85	0.03 [a,b]
ToM Stories, sequencing	29.5 (6.8)	25.4 (7.6)	30.0 (4.9)	5.11	0.07
ToM Stories, questionnaire	20.9 (2.8)	18.1 (3.9)	21.4 (1.7)	11.14	0.004 [a,b]
RSMS	44.2 (12.6)	41.8 (6.9)	47.0 (8.4)	2.54	0.28
KDEF (total score)	46.7 (9.3)	44.1 (7.1)	49.6 (3.9)	2.23	0.32
Neutral	12.6 (4.3)	11.5 (3.7)	14.4 (2.4)	5.03	0.08
Fear	2.66 (1.8)	2.8 (1.1)	2.2 (1.5)	1.28	0.52
Happiness	7.8 (0.4)	7.7 (0.6)	7.8 (0.7)	4.30	0.11
Disgust	5.4 (2.2)	5.4 (1.5)	5.5 (1.7)	0.32	0.85
Sadness	4.7 (2.3)	3.9 (2.6)	5.6 (1.7)	2.02	0.36
Surprise	7.2 (1.5)	7.0 (1.3)	7.5 (0.5)	0.19	0.91
Anger	6.3 (1.3)	5.8 (2.2)	6.6 (1.3)	0.98	0.61

Note: Non-parametric variables. Kruskal–Wallis was applied. [a] Significant differences between PD-MCI and PD-CN in DSCF analysis. [b] Significant differences between PD-MCI and HC in DSCF analysis. PD-CN = Parkinson's disease, Cognitively Normal; PD-MCI = Parkinson's disease, mild cognitive impairment; HC = healthy control; RMET = Reading the Mind in The Eyes Test; ToM Stories = Theory of Mind Picture Stories Task; RSMS = Revised Self-Monitoring Scale; KDEF = Karolinska Directed Emotional Faces; DSCF = Dwass–Steel–Critchlow–Fligner pairwise comparisons post hoc test.

However, in the case of affective ToM and FER, we observed significant differences in RMET test (F = 8.43, $p < 0.05$), but only between PD-MCI (M = 18.4, SD = 3.24) and HC (M = 22.4, SD = 4.59). The analysis of the total KDEF score and analysis of each emotion individually showed no significant results (F = 2.23, p = 0.32).

3.3. Correlations between Neuropsychological and Neuropsychiatric Evaluation and Social Cognition Assessment

We calculated the correlation coefficient of the relationship between neuropsychological and neuropsychiatric variables with SC.

After Bonferroni correction, the following statistically significant correlations were found: (1) ToM task total score correlated with TMT-A (r = −0.52; $p < 0.05$) (attention), TMT-B (r = −0.59; $p < 0.05$) and phonemic fluency (r = 0.54; $p < 0.05$) (executive function), Boston Naming Test (r = 0.63; $p < 0.05$) (language), and WMS-IV (r = 0.56; $p < 0.05$) (visual memory); (2) affective ToM correlated with CERAD (r = 0.52; $p < 0.05$) (verbal memory); and (3) FER correlated with Stroop test inhibition (r = −0.50; $p < 0.05$) (executive function) and CERAD (r = 0.58; $p < 0.05$) (verbal memory) (Table 3).

Table 3. Spearman rank correlations between neuropsychological evaluation and social cognition assessment.

		The Karolinska Directed Emotional Faces	Reading the Mind in the Eyes Test	Theory of Mind Picture Stories Task			Revised Self-Monitoring Scale
				Total	Sequencing	Questionnaire	
Apathy	SAS	−0.39	−0.47	−0.29	−0.30	−0.28	−0.32
Attention	Digit Span Forward and Backward	0.28 0.34	0.29 0.31	0.35 0.34	0.35 0.38	0.31 0.22	0.26 0.20
	TMTA	−0.30	−0.30	−0.52 [a]	−0.48 [a]	−0.54 [a]	−0.15
Executive function	Stroop test inhibition	−0.50 [a]	−0.30	−0.47	−0.47 [a]	−0.41	0.11
	TMTB	−0.41	−0.25	−0.59 [a]	−0.55 [a]	−0.57 [a]	−0.10
	Phonemic fluency	0.34	0.30	0.54 [a]	0.51 [a]	0.54 [a]	0.01
Language	Boston Naming Test	0.44	0.34	0.63 [a]	0.66 [a]	0.48 [a]	0.07
	Semantic fluency [b]	0.24	0.33	0.39	0.34	0.42	0.01
Memory	CERAD	0.58 [a]	0.52 [a]	0.40	0.41	0.37	0.06
	WMS	0.47	0.39	0.56 [a]	0.57 [a]	0.48 [a]	0.02
Visuospatial function	JLO	0.29	0.23	0.36	0.35	0.40	−0.03
	VOSP	0.17	0.25	0.33	0.35	0.33	−0.08
	ROCF	0.32	0.25	0.37	0.37	0.32	0.16

Note: [a] Statistically significant results after Bonferroni adjustment. [b] Parametric variable. Pearson correlation coefficient was used. TMTA = Trail Making Test A; TMTB = Trail Making Test B; CERAD = Consortium to Establish a Registry for Alzheimer's Disease; WMS = Wechsler Memory Scale; JLO = Judgment of Line Orientation; VOSP = Visual Object and Space Perception Battery; ROCF = Rey–Osterrieth complex figure; SAS = Starkstein Apathy Scale.

4. Discussion

The primary purpose of this study was to examine SC abilities in non-demented mid-stage PD patients with and without cognitive impairment (PD-CN and PD-MCI). Our results revealed that PD-MCI patients show worse global (affective and cognitive) ToM performance than HC. In contrast, when we compared PD-MCI and PD-CN patients, only ToM task scores (more specifically in the questionnaire part) showed differences, with no significant results for affective ToM. We found no differences when comparing FER and RSMS performance among groups. There was a moderate negative correlation between cognitive ToM and executive functions, as well as positive with memory (WMS),

language and attention (TMT-A) cognitive tests, whereas FER and affective ToM, only were moderately positively correlated with memory function.

Our results support previous data that reported both cognitive and affective ToM are impaired in PD-MCI patients [24,25]. In our study, patients with severe clinical depression were excluded, given its association with SC impairment in general population [27] and PD patients [29]. In comparison with previous studies, disease duration was relatively homogeneous in our sample (mean disease duration of 5.56 ± 2.09 years) that allows the study of SC in a well-established population of late-onset mid-stage PD patients. Finally, in our study, we have applied the MDS Level II criteria for the diagnosis of PD-MCI [11], which seem to have the best balance of sensitivity, specificity, and diagnostic accuracy, as well as a better predictive value for the development of PDD for PD-MCI patients compared to patients with normal cognition [53].

It has been proposed that cognitive ToM impairment could be explained by dopaminergic loss [54]. This statement is supported by recent neuroimaging data that show a significant positive correlation between ToM stories and Fluorodopa uptake in the right thalamus and the left putamen in PD patients [55]. Some previous studies in PD have suggested that ToM deficits may emerge at more advanced stages of the disease, specifically in those patients in whom the degenerative process has extended beyond the dopaminergic pathways, but not in early-stage patients where neuronal loss appears to be confined to the nigrostriatal and mesolimbic dopaminergic systems [56,57]. In any case, to date, there are no neuropathological studies confirming this hypothesis.

On the other hand, cognitively normal PD patients did not show differences in SC performance in comparison with HC. Previous studies have shown that SC appears to be preserved in de novo cognitively normal PD patients [55], so it seems that SC is preserved as long as cognition is unimpaired. In this regard, it has been shown that deficits in cognitive functioning can predict future SC deficits [58], and SC severity is significantly associated with cognitive impairment [59].

As in previous research, cognitive ToM mainly correlated with language [60], attention [61], and executive functions [62,63]. Cognitive ToM and executive functions have been related to the associative circuit of the basal ganglia in PD involving the dorsal anterior cingulate cortex, the dorsomedial prefrontal cortex, and the dorsal striatum/caudate. Impairment of this circuit would lead to cognitive ToM dysfunction, whereas impairment of the affective ToM component would appear to be due to limbic network impairment, including the ventromedial prefrontal cortex and orbitofrontal cortex, the ventral anterior cingulate cortex, the amygdala, and the ventral striatum [16,64].

Even though we have shown that both cognitive and affective ToM are impaired in PD-MCI, the fact that PD-MCI patients only show impairment in affective ToM when comparing with HC and not with PD-CN, suggests initial cognitive ToM impairment followed by the addition of affective ToM worsening.

In comparison to previous studies in non-demented PD [24,26], we did not find impairment in FER. This can be explained by the use of BFRT as a potential prerequisite to analyze FER [65,66], which may have functioned as a confounder in previous studies. Additionally, the exclusion of patients with severe clinical depression may explain this result. Depression has been described as a possible confounder when analyzing FER [67] and its severity increases FER deficits [68]. Likewise, our PD patient sample did not show self-monitoring difficulties. This aspect probably shares neural correlates with affective ToM and FER, since both are associated with insula and orbitofrontal cortex disconnection from structures of the salience network [69,70].

Finally, gender and educational level have been identified as having an impact in SC in the general population [71] and a weak effect on PD [60,72]. Although our sample has been matched for both variables, it is noteworthy that the inclusion in the analysis of these variables as potential confounders did not modify our results.

The main strength of this study lies in the administration of a comprehensive neuropsychological assessment and a broad SC evaluation to patients with homogeneous

age and disease duration, the exclusion of severe depression, and the comparison with a healthy control sample. Nevertheless, our study limitation is that the small sample size could limit the generalizability of the results. In addition, we did not perform a sample size analysis, the number of subjects was chosen on the basis of previous studies.

5. Conclusions

Our findings indicate that SC is normal in cognitively unimpaired and mid-stage PD patients without clinically relevant depression, whereas a decline in affective and cognitive ToM seems to be linked to the presence of MCI. Overall, our results increase the evidence of SC as an important aspect of PD likely associated with the onset of cognitive impairment and emphasize the importance of evaluating these functions in this population.

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Data Availability Statement: The data that support the findings of this study are available from the corresponding author, [C.G.S.], upon reasonable request. The data are not publicly available due to their containing information that could compromise the privacy of research participants.

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References

1. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: A systematic analysis for the Global Burden of Disease Study 2017. *Lancet* **2018**, *392*, 1789–1858. [CrossRef]
2. Postuma, R.B.; Berg, D.; Stern, M.; Poewe, W.; Olanow, C.W.; Oertel, W.; Obeso, J.; Marek, K.; Litvan, I.; Lang, A.E.; et al. MDS clinical diagnostic criteria for Parkinson's disease. *Mov. Disord. Off. J. Mov. Disord. Soc.* **2015**, *30*, 1591–1601. [CrossRef]
3. Bloem, B.R.; Okun, M.S.; Klein, C. Parkinson's disease. *Lancet* **2021**, *397*, 2284–2303. [CrossRef]
4. Aarsland, D.; Batzu, L.; Halliday, G.M.; Geurtsen, G.J.; Ballard, C.; Ray Chaudhuri, K.; Weintraub, D. Parkinson disease-associated cognitive impairment. *Nat. Reviews. Dis. Primers* **2021**, *7*, 47. [CrossRef]
5. Monastero, R.; Cicero, C.E.; Baschi, R.; Davì, M.; Luca, A.; Restivo, V.; Zangara, C.; Fierro, B.; Zappia, M.; Nicoletti, A. Mild cognitive impairment in Parkinson's disease: The Parkinson's disease cognitive study (PACOS). *J. Neurol.* **2018**, *265*, 1050–1058. [CrossRef] [PubMed]
6. Santangelo, G.; Vitale, C.; Picillo, M.; Moccia, M.; Cuoco, S.; Longo, K.; Pezzella, D.; di Grazia, A.; Erro, R.; Pellecchia, M.T.; et al. Mild Cognitive Impairment in newly diagnosed Parkinson's disease: A longitudinal prospective study. *Park. Relat. Disord.* **2015**, *21*, 1219–1226. [CrossRef]
7. Hely, M.A.; Reid, W.G.; Adena, M.A.; Halliday, G.M.; Morris, J.G. The Sydney multicenter study of Parkinson's disease: The inevitability of dementia at 20 years. *Mov. Disord. Off. J. Mov. Disord. Soc.* **2008**, *23*, 837–844. [CrossRef] [PubMed]
8. Lawson, R.A.; Yarnall, A.J.; Duncan, G.W.; Breen, D.P.; Khoo, T.K.; Williams-Gray, C.H.; Barker, R.A.; Collerton, D.; Taylor, J.P.; Burn, D.J.; et al. Cognitive decline and quality of life in incident Parkinson's disease: The role of attention. *Park. Relat. Disord.* **2016**, *27*, 47–53. [CrossRef] [PubMed]
9. Tang, Y.; Liang, X.; Han, L.; Peng, F.; Shen, B.; Yu, H.; Shen, Y.; Shen, C.; Yu, J.; Wang, J. Cognitive Function and Quality of Life in Parkinson's Disease: A Cross-Sectional Study. *J. Park. Dis.* **2020**, *10*, 1209–1216. [CrossRef] [PubMed]
10. Petersen, R.C.; Smith, G.E.; Waring, S.C.; Ivnik, R.J.; Tangalos, E.G.; Kokmen, E. Mild cognitive impairment: Clinical characterization and outcome. *Arch. Neurol.* **1999**, *56*, 303–308. [CrossRef] [PubMed]

11. Litvan, I.; Goldman, J.G.; Tröster, A.I.; Schmand, B.A.; Weintraub, D.; Petersen, R.C.; Mollenhauer, B.; Adler, C.H.; Marder, K.; Williams-Gray, C.H.; et al. Diagnostic criteria for mild cognitive impairment in Parkinson's disease: Movement Disorder Society Task Force guidelines. *Mov. Disord. Off. J. Mov. Disord. Soc.* **2012**, *27*, 349–356. [CrossRef]
12. Pedersen, K.F.; Larsen, J.P.; Tysnes, O.B.; Alves, G. Natural course of mild cognitive impairment in Parkinson disease: A 5-year population-based study. *Neurology* **2017**, *88*, 767–774. [CrossRef]
13. Gasca-Salas, C.; Estanga, A.; Clavero, P.; Aguilar-Palacio, I.; González-Redondo, R.; Obeso, J.A.; Rodríguez-Oroz, M.C. Longitudinal assessment of the pattern of cognitive decline in non-demented patients with advanced Parkinson's disease. *J. Park. Dis.* **2014**, *4*, 677–686. [CrossRef]
14. Hoogland, J.; Boel, J.A.; de Bie, R.M.A.; Geskus, R.B.; Schmand, B.A.; Dalrymple-Alford, J.C.; Marras, C.; Adler, C.H.; Goldman, J.G.; Tröster, A.I.; et al. Validation of Mild Cognitive Impairment in Parkinson Disease" Mild cognitive impairment as a risk factor for Parkinson's disease dementia. *Mov. Disord. Off. J. Mov. Disord. Soc.* **2017**, *32*, 1056–1065. [CrossRef]
15. Wallace, E.R.; Segerstrom, S.C.; van Horne, C.G.; Schmitt, F.A.; Koehl, L.M. Meta-Analysis of Cognition in Parkinson's Disease Mild Cognitive Impairment and Dementia Progression. *Neuropsychol. Rev.* **2022**, *32*, 149–160. [CrossRef]
16. Trompeta, C.; Fernández Rodríguez, B.; Gasca-Salas, C. What Do We Know about Theory of Mind Impairment in Parkinson's Disease? *Behav. Sci.* **2021**, *11*, 130. [CrossRef]
17. Arioli, M.; Crespi, C.; Canessa, N. Social Cognition through the Lens of Cognitive and Clinical Neuroscience. *BioMed Res. Int.* **2018**, *2018*, 4283427. [CrossRef]
18. Rossetto, F.; Baglio, F.; Massaro, D.; Alberoni, M.; Nemni, R.; Marchetti, A.; Castelli, I. Social Cognition in Rehabilitation Context: Different Evolution of Affective and Cognitive Theory of Mind in Mild Cognitive Impairment. *Behav. Neurol.* **2020**, *2020*, 5204927. [CrossRef] [PubMed]
19. Franklin, H.D.; Russell, L.L.; Peakman, G.; Greaves, C.V.; Bocchetta, M.; Nicholas, J.; Poos, J.; Convery, R.S.; Cash, D.M.; van Swieten, J.; et al. Genetic FTD Initiative, GENFI The Revised Self-Monitoring Scale detects early impairment of social cognition in genetic frontotemporal dementia within the GENFI cohort. *Alzheimer's Res. Ther.* **2021**, *13*, 127. [CrossRef] [PubMed]
20. Multani, N.; Taghdiri, F.; Anor, C.J.; Varriano, B.; Misquitta, K.; Tang-Wai, D.F.; Keren, R.; Fox, S.; Lang, A.E.; Vijverman, A.C.; et al. Association Between Social Cognition Changes and Resting State Functional Connectivity in Frontotemporal Dementia, Alzheimer's Disease, Parkinson's Disease, and Healthy Controls. *Front. Neurosci.* **2019**, *13*, 1259. [CrossRef] [PubMed]
21. Roca, M.; Torralva, T.; Gleichgerrcht, E.; Chade, A.; Arévalo, G.G.; Gershanik, O.; Manes, F. Impairments in social cognition in early medicated and unmedicated Parkinson disease. *Cogn. Behav. Neurol. Off. J. Soc. Behav. Cogn. Neurol.* **2010**, *23*, 152–158. [CrossRef]
22. Palmeri, R.; Lo Buono, V.; Corallo, F.; Foti, M.; Di Lorenzo, G.; Bramanti, P.; Marino, S. Nonmotor Symptoms in Parkinson Disease: A Descriptive Review on Social Cognition Ability. *J. Geriatr. Psychiatry Neurol.* **2017**, *30*, 109–121. [CrossRef]
23. Seubert-Ravelo, A.N.; Yáñez-Téllez, M.G.; Lazo-Barriga, M.L.; Calderón Vallejo, A.; Martínez-Cortés, C.E.; Hernández-Galván, A. Social Cognition in Patients with Early-Onset Parkinson's Disease. *Park. Dis.* **2021**, *2021*, 8852087. [CrossRef]
24. Alonso-Recio, L.; Carvajal, F.; Merino, C.; Serrano, J.M. Social Cognition and Cognitive Decline in Patients with Parkinson's Disease. *J. Int. Neuropsychol. Soc. JINS* **2021**, *27*, 744–755. [CrossRef]
25. Dodich, A.; Funghi, G.; Meli, C.; Pennacchio, M.; Longo, C.; Malaguti, M.C.; Di Giacopo, R.; Zappini, F.; Turella, L.; Papagno, C. Deficits in Emotion Recognition and Theory of Mind in Parkinson's Disease Patients with and without Cognitive Impairments. *Front. Psychol.* **2022**, *13*, 866809. [CrossRef] [PubMed]
26. Czernecki, V.; Benchetrit, E.; Houot, M.; Pineau, F.; Mangone, G.; Corvol, J.C.; Vidailhet, M.; Levy, R. Social cognitive impairment in early Parkinson's disease: A novel "mild impairment"? *Park. Relat. Disord.* **2021**, *85*, 117–121. [CrossRef]
27. Ladegaard, N.; Larsen, E.R.; Videbech, P.; Lysaker, P.H. Higher-order social cognition in first-episode major depression. *Psychiatry Res.* **2014**, *216*, 37–43. [CrossRef] [PubMed]
28. Lee, S.; Jia, Y.; Snitz, B.E.; Chang, C.H.; Ganguli, M. Assessing Social Cognition in Older Adults: A Population-Based Study. *Alzheimer Dis. Assoc. Disord.* **2022**, *36*, 103–110. [CrossRef]
29. Mengelberg, A.; Siegert, R.J. Is theory-of-mind impaired in Parkinson's disease? *Cogn. Neuropsychiatry* **2003**, *8*, 191–209. [CrossRef]
30. Baiano, C.; Barone, P.; Trojano, L.; Santangelo, G. Prevalence and clinical aspects of mild cognitive impairment in Parkinson's disease: A meta-analysis. *Mov. Disord. Off. J. Mov. Disord. Soc.* **2020**, *35*, 45–54. [CrossRef]
31. Ng, A.; Chander, R.J.; Tan, L.C.; Kandiah, N. Influence of depression in mild Parkinson's disease on longitudinal motor and cognitive function. *Park. Relat. Disord.* **2015**, *21*, 1056–1060. [CrossRef]
32. Hughes, A.J.; Daniel, S.E.; Kilford, L.; Lees, A.J. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: A clinico-pathological study of 100 cases. *J. Neurol. Neurosurg. Psychiatry* **1992**, *55*, 181–184. [CrossRef]
33. Yesavage, J.A.; Brink, T.L.; Rose, T.L.; Lum, O.; Huang, V.; Adey, M.; Leirer, V.O. Development and validation of a geriatric depression screening scale: A preliminary report. *J. Psychiatr. Res.* **1982**, *17*, 37–49. [CrossRef]
34. Jost, S.T.; Kaldenbach, M.A.; Antonini, A.; Martinez-Martin, P.; Timmermann, L.; Odin, P.; Katzenschlager, R.; Borgohain, R.; Fasano, A.; Stocchi, F.; et al. International Parkinson and Movement Disorders Society Non-Motor Parkinson Disease Study Group Levodopa Dose Equivalency in Parkinson's Disease: Updated Systematic Review and Proposals. *Mov. Disord. Off. J. Mov. Disord. Soc.* **2023**, *38*, 1236–1252. [CrossRef]
35. Wechsler, D. *WAIS-IV. Escala de Inteligencia de Wechsler Para Adultos-IV. Manual de Aplicación y Corrección*; NCS Pearson, Inc.: Madrid, Spain, 2012.

36. Partington, J.; Leiter, R. Partington's Pathway Test. *Psychol. Serv. Cent. J.* **1949**, *1*, 11–20.
37. Isaacs, B.; Kennie, A.T. The Set test as an aid to the detection of dementia in old people. *Br. J. Psychiatry* **1973**, *123*, 467–470. [CrossRef]
38. Kaplan, E.F.; Goodglass, H.; Weintraub, S. *Boston Naming Test*; Lippincott Williams & Wilkins: Philadelphia, PA, USA, 1983.
39. Morris, J.C.; Heyman, A.; Mohs, R.C.; Hughes, J.P.; van Belle, G.; Fillenbaum, G.; Mellits, E.D.; Clark, C. The Consortium to Establish a Registry for Alzheimer's Disease (CERAD). Part I. Clinical and neuropsychological assessment of Alzheimer's disease. *Neurology* **1989**, *39*, 1159–1165.
40. Wechsler, D. *Escala de Memoria de Wechsler*; Pearson Educación, S.A.: Madrid, Spain, 2013.
41. Benton, A.L.; Sivan, A.B.; de Hamsher, K.S.; Varney, N.R.; Spreen, O. *Contributions to Neuropsychological Assessment*; Oxford University Press: New York, NY, USA, 1994.
42. Rapport, L.J.; Millis, S.R.; Bonello, P.J. Validation of the Warrington theory of visual processing and the Visual Object and Space Perception Battery. *J. Clin. Exp. Neuropsychol.* **1998**, *20*, 211–220. [CrossRef] [PubMed]
43. Berry DT, R.; Allen, R.S.; Schmitt, F.A. Rey-Osterrieth complex figure: Psychometric characteristics in a geriatric sample. *Clin. Neuropsychol.* **1991**, *5*, 143–153. [CrossRef]
44. Rossion, B.; Michel, C. Normative accuracy and response time data for the computerized Benton Facial Recognition Test (BFRT-c). *Behav. Res. Methods* **2018**, *50*, 2442–2460. [CrossRef] [PubMed]
45. Lundqvist, D.; Flykt, A.; Öhman, A. Karolinska directed emotional faces. *PsycTESTS Dataset* **1998**, *91*, 630.
46. Calvo, M.G.; Lundqvist, D. Facial expressions of emotion (KDEF): Identification under different display-duration conditions. *Behav. Res. Methods* **2008**, *40*, 109–115. [CrossRef]
47. McIntosh, L.G.; Park, S. Social trait judgment and affect recognition from static faces and video vignettes in schizophrenia. *Schizophr. Res.* **2014**, *158*, 170–175. [CrossRef] [PubMed]
48. Baron-Cohen, S.; Jolliffe, T.; Mortimore, C.; Robertson, M. Another advanced test of theory of mind: Evidence from very high functioning adults with autism or asperger syndrome. *J. Child Psychol. Psychiatry Allied Discip.* **1997**, *38*, 813–822. [CrossRef] [PubMed]
49. Baron-Cohen, S.; Wheelwright, S.; Hill, J.; Raste, Y.; Plumb, I. The "Reading the Mind in the Eyes" Test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *J. Child Psychol. Psychiatry Allied Discip.* **2001**, *42*, 241–251. [CrossRef]
50. Fernández-Abascal, E.G.; Cabello, R.; Fernández-Berrocal, P.; Baron-Cohen, S. Test-retest reliability of the 'Reading the Mind in the Eyes' test: A one-year follow-up study. *Mol. Autism* **2013**, *4*, 33. [CrossRef] [PubMed]
51. Brüne, M. Theory of mind and the role of IQ in chronic disorganized schizophrenia. *Schizophr. Res.* **2003**, *60*, 57–64. [CrossRef]
52. Lennox, R.D.; Wolfe, R.N. Revision of the self-monitoring scale. *J. Personal. Soc. Psychol.* **1984**, *46*, 1349–1364. [CrossRef]
53. Boel, J.A.; de Bie, R.M.A.; Schmand, B.A.; Dalrymple-Alford, J.C.; Marras, C.; Adler, C.H.; Goldman, J.G.; Tröster, A.I.; Burn, D.J.; Litvan, I.; et al. Mild Cognitive Impairment in Parkinson's Disease Level I PD-MCI Using Global Cognitive Tests and the Risk for Parkinson's Disease Dementia. *Mov. Disord. Clin. Pract.* **2022**, *9*, 479–483. [CrossRef]
54. Poletti, M.; Bonuccelli, U. Acute and chronic cognitive effects of levodopa and dopamine agonists on patients with Parkinson's disease: A review. *Ther. Adv. Psychopharmacol.* **2013**, *3*, 101–113. [CrossRef]
55. Trompeta, C.; Gasca-Salas, C.; Pineda-Pardo, J.A.; Guida, P.; Cohn, M.; Mata-Marín, D.; Monje, M.H.; López-Aguirre, M.; Obeso, I.; Sánchez Ferro, Á. Longitudinal assessment of social cognition in de novo Parkinson's disease patients and its relationship with dopaminergic innervation. *Behav. Brain Res.* **2023**, *454*, 114654. [CrossRef]
56. Péron, J.; Vicente, S.; Leray, E.; Drapier, S.; Drapier, D.; Cohen, R.; Biseul, I.; Rouaud, T.; Le Jeune, F.; Sauleau, P.; et al. Are dopaminergic pathways involved in theory of mind? A study in Parkinson's disease. *Neuropsychologia* **2009**, *47*, 406–414. [CrossRef]
57. Braak, H.; Del Tredici, K.; Rüb, U.; de Vos, R.A.; Jansen Steur, E.N.; Braak, E. Staging of brain pathology related to sporadic Parkinson's disease. *Neurobiol. Aging* **2003**, *24*, 197–211. [CrossRef]
58. Bora, E.; Walterfang, M.; Velakoulis, D. Theory of mind in Parkinson's disease: A meta-analysis. *Behav. Brain Res.* **2015**, *292*, 515–520. [CrossRef]
59. Romosan, A.M.; Dehelean, L.; Romosan, R.S.; Andor, M.; Bredicean, A.C.; Simu, M.A. Affective theory of mind in Parkinson's disease: The effect of cognitive performance. *Neuropsychiatr. Dis. Treat.* **2019**, *15*, 2521–2535. [CrossRef] [PubMed]
60. Maggi, G.; Cima Muñoz, A.M.; Obeso, I.; Santangelo, G. Neuropsychological, neuropsychiatric, and clinical correlates of affective and cognitive theory of mind in Parkinson's disease: A meta-analysis. *Neuropsychology* **2022**, *36*, 483–504. [CrossRef] [PubMed]
61. Kosutzka, Z.; Kralova, M.; Kusnirova, A.; Papayova, M.; Valkovic, P.; Csefalvay, Z.; Hajduk, M. Neurocognitive Predictors of Understanding of Intentions in Parkinson Disease. *J. Geriatr. Psychiatry Neurol.* **2019**, *32*, 178–185. [CrossRef] [PubMed]
62. Saltzman, J.; Strauss, E.; Hunter, M.; Archibald, S. Theory of mind and executive functions in normal human aging and Parkinson's disease. *J. Int. Neuropsychol. Soc. JINS* **2000**, *6*, 781–788. [CrossRef] [PubMed]
63. Costa, A.; Peppe, A.; Martini, M.; Coletta, K.; Oliveri, M.; Caltagirone, C.; Carlesimo, G.A. Parkinsonian patients with deficits in the dysexecutive spectrum are impaired on theory of mind tasks. *Behav. Neurol.* **2013**, *27*, 523–533. [CrossRef] [PubMed]
64. Abu-Akel, A. The neurochemical hypothesis of 'theory of mind'. *Med. Hypotheses* **2003**, *60*, 382–386. [CrossRef]
65. Biotti, F.; Cook, R. Impaired perception of facial emotion in developmental prosopagnosia. *Cortex A J. Devoted Study Nerv. Syst. Behav.* **2016**, *81*, 126–136. [CrossRef]

66. Djouab, S.; Albonico, A.; Yeung, S.C.; Malaspina, M.; Mogard, A.; Wahlberg, R.; Corrow, S.L.; Barton, J.J.S. Search for Face Identity or Expression: Set Size Effects in Developmental Prosopagnosia. *J. Cogn. Neurosci.* **2020**, *32*, 889–905. [CrossRef]
67. Siquier, A.; Andrés, P. Facial emotion recognition in Parkinson's disease: The role of executive and affective domains. *Neuropsychol.* **2022**, *36*, 384–393. [CrossRef]
68. Argaud, S.; Vérin, M.; Sauleau, P.; Grandjean, D. Facial emotion recognition in Parkinson's disease: A review and new hypotheses. *Mov. Disord. Off. J. Mov. Disord. Soc.* **2018**, *33*, 554–567. [CrossRef] [PubMed]
69. Parthimos, T.P.; Karavasilis, E.; Rankin, K.P.; Seimenis, I.; Leftherioti, K.; Papanicolaou, A.C.; Miller, B.; Papageorgiou, S.G.; Papatriantafyllou, J.D. The Neural Correlates of Impaired Self-Monitoring among Individuals with Neurodegenerative Dementias. *J. Neuropsychiatry Clin. Neurosci.* **2019**, *31*, 201–209. [CrossRef]
70. Baggio, H.C.; Segura, B.; Ibarretxe-Bilbao, N.; Valldeoriola, F.; Marti, M.J.; Compta, Y.; Tolosa, E.; Junqué, C. Structural correlates of facial emotion recognition deficits in Parkinson's disease patients. *Neuropsychologia* **2012**, *50*, 2121–2128. [CrossRef] [PubMed]
71. Quesque, F.; Coutrot, A.; Cox, S.; de Souza, L.C.; Baez, S.; Cardona, J.F.; Mulet-Perreault, H.; Flanagan, E.; Neely-Prado, A.; Clarens, M.F.; et al. Does culture shape our understanding of others' thoughts and emotions? An investigation across 12 countries. *Neuropsychology* **2022**, *36*, 664–682. [CrossRef] [PubMed]
72. Siripurapu, G.; Verma, B.; Biswas, D.; Reghu, A.; Vishnoi, A.; Radhakrishnan, D.M.; Elavarasi, A.; Gupta, A.; Vishnu, V.Y.; Singh, M.B.; et al. Social Cognition in Parkinson's Disease: A Case-Control Study. *Mov. Disord. Clin. Pract.* **2023**, *10*, 399–405. [CrossRef]