Altered awareness of cognitive and neuropsychiatric symptoms in Parkinson's disease and Dementia with Lewy Bodies: a systematic review

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REVIEW ARTICLE

Altered awareness of cognitive and neuropsychiatric symptoms in Parkinson's disease and Dementia with Lewy Bodies: A systematic review

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Objectives: Altered awareness of cognitive and neuropsychiatric symptoms is a common feature of neurodegeneration, which can significantly impact on quality of life, medication concordance and personal safety. Elucidating how awareness is affected by common alpha-synucleinopathies therefore has significant clinical relevance. We performed a systematic review of the literature on awareness of cognitive and neuropsychiatric symptoms in Parkinson's disease and Dementia with Lewy Bodies.

Methods: Searches of PubMed and Web of Science were carried out, using keywords and MeSH subheadings, limited to papers in English dealing with humans. The terms "Parkinson's" or "Lewy body" were used to denote the disease of interest, combined with either "agnosia", "anosognosia", "insight", "metacognition", or "neuropsychology" to denote the neuropsychological area of interest.

Results: 21 publications investigating awareness of cognitive symptoms, and 18 publications on awareness of neuropsychiatric symptoms were identified. The large majority focused on Parkinson's disease rather than Dementia with Lewy Bodies. Cognitively intact people with Parkinson's disease may over-report cognitive symptoms, whilst those with cognitive impairment under-report symptoms. Awareness of neuropsychiatric symptoms is likely to decline over time, particularly in those with progressive cognitive impairment.

Conclusions: Altered awareness of cognitive and neuropsychiatric symptoms is common in Parkinson's disease. Symptom awareness varies significantly between individuals, and appears to be influenced by mood and global cognitive functioning, with executive functioning specifically implicated. There are gaps in our understanding of how dopaminergic medications influence symptom awareness, and a need for longitudinal studies of how awareness changes over time in Parkinson's disease and Dementia with Lewy Bodies.

KEYWORDS
anosognosia, Dementia with Lewy bodies, insight, metacognition, Parkinson's disease, Parkinson's disease dementia, symptom awareness

1 | INTRODUCTION

Cognitive and neuropsychiatric symptoms are common manifestations of Parkinson's disease (PD) and Dementia with Lewy Bodies (DLB).[1,2] Altered awareness of cognitive and neuropsychiatric symptoms can have a significant impact not only on quality of life for the affected individual, but also their significant others or carers.[3] Those affected are frequently not fully consciously aware of their symptoms. Patients may decline therapies, become distressed by hallucinations or delusions, or be driven to potentially dangerous actions such as searching for the source of a hallucination. They may continue to carry out high risk activities, such as driving or making financial transactions, despite being cognitively compromised.

Cognitive decline is a common consequence of PD, with mild cognitive impairment being already present in around 30% of patients at the time of their PD diagnosis. Parkinson's Disease with Mild Cognitive Impairment (PD-MCI) confers an increased risk of future PD dementia.[4,5] Longitudinal studies indicate that the majority of people with PD will ultimately develop dementia.[1] Typically a profile of executive dysfunction, attentional deficits and visuospatial difficulties is seen, with memory being affected to a lesser extent.[6] This is a close match to the profile seen in DLB.[2] Clinically and pathologically, Parkinson's disease dementia (PDD) is closely related to DLB, and the two conditions are viewed as part of the alpha synucleinopathy spectrum, with a clinical distinction being drawn according to the temporal order of onset of motor or cognitive symptoms.[2,7]

Neuropsychiatric manifestations of PD and DLB include depression, anxiety, apathy, psychotic symptoms, impulse control disorders and sleep disorders.[2,8] Strong associations are seen between neuropsychiatric symptoms and cognitive decline in PD, with higher prevalence in those with MCI or dementia.[8] Depression has been reported in over half of cognitively intact people with PD, and 70% of those with amnestic MCI.[9] Anxiety and apathy is also common, and apathy is reported in up to 50% of people with PDD.[10] Visual hallucinations are a key feature of DLB, and form part of the diagnostic criteria.[2] They are typically formed and feature animals or people. In PD, a range of psychotic experiences may occur.[11,12] PD psychosis is recognised to encompass a spectrum of symptoms, often with a pattern of increasing severity and complexity as PD progresses. Early features include the feeling of another being present (presence hallucinations) and passage hallucinations - the experience of someone or something moving across the edge of the visual field. Visual illusions and formed hallucinations may also occur, with preserved insight early on. Later in the disease course, insight is lost and other sensory modalities can be involved, with auditory, tactile and olfactory hallucinations reported. Delusions can arise, often with paranoid ideation, and rarely misidentification syndromes such as Capgras delusion.

Loss of symptom awareness is a prominent feature of many forms of neurodegeneration, and has been most widely studied in Alzheimer's disease (AD), where it increases in proportion to the severity of cognitive impairment.[13] How awareness is affected by common alpha-synucleinopathies is less well understood. We now recognise that cognitive impairment is very common in PD, with around 80% of people developing dementia at some point in the disease process.[14] A diagnosis of PD-MCI or PDD can utilise reports from patients, caregivers and clinicians. Knowing how reliable patient reports are, and understanding how cognitive symptom awareness may interact with cognitive impairment in PD, is crucial in order to make an accurate clinical assessment. Inaccurate symptom reporting may also affect medical management decisions and clinical trial outcomes. Just as there are multiple cognitive domains, we can regard symptom awareness as segregating across different domains - with awareness of memory functioning being distinct from awareness of visuospatial functioning, for example.[15,16] Impaired executive functioning is often postulated as a cause of reduced symptom awareness.[13] One of the aims of this review is to examine how symptom awareness interacts with different cognitive domains, both in terms of accuracy of self-knowledge, and links between cognitive impairment and the severity of altered awareness.

Key points

- Altered awareness of cognitive and neuropsychiatric symptoms is common in Parkinson's disease.
- Awareness of neuropsychiatric symptoms appears to decline over time in people with Parkinson's disease, particularly in those with cognitive impairment.
- Cognitively intact people with Parkinson's disease may over-report cognitive symptoms.
- There is a lack of research on symptom awareness in people with Dementia with Lewy Bodies.

Neuropsychiatric symptoms may also be affected by loss of awareness, and this is acknowledged in the current PD psychosis definitions.[11] Neuropsychiatric symptoms may only become obvious when the affected individual actively reports them - hallucinations will only be evident if the person describes them, or is observed to interact with them. Similarly mild depression, anxiety or apathy may be purely internal experiences for the patient, and not obvious to external observers. Therefore the accuracy of self-report becomes critical for evaluation, treatment and research into neuropsychiatric symptomatology.

The aims of this review are to synthesise the literature investigating awareness of cognitive and neuropsychiatric symptoms in PD and DLB. We will review the existing knowledge base of how symptom awareness varies across different cognitive domains and neuropsychiatric symptom types. We will discuss how global and domain specific cognitive functioning interacts with symptom awareness, and the potential impact of dopaminergic therapies. We will evaluate the methodologies used in the field of symptom awareness, and highlight gaps in our understanding of symptom awareness in common alpha synucleinopathies.

2 | METHODS

A systemic review was undertaken of the literature investigating altered symptom awareness in PD and DLB across motor symptoms,

TABLE 1 Studies of awareness of cognitive symptoms in PD and DLB

Study	Study groups	Age (Years[a])	Disease duration (Months[a])	Cognitive status (Mean MMSE unless otherwise stated)	Recruitment source
Baran et al.[21]	18 people with PD-MCI 18 controls	PD-MCI: 65.30 Controls: 63.72	Not reported	PD-MCI: 26.72 Controls: 29.61	Specialist clinic
Basic et al.[19]	58 people with PD & carers 58 controls	PD: 66.09 Controls: 66.85	PD: 77.76	PD: 26.1 Controls: 27.1	Not reported
Castro et al.[50]	31 PD cognitively intact 21 PD & cognitive symptoms 25 controls	PD cognitively intact: 60.74 PD & cognitive symptoms: 60.62 Controls: 55.76	PD cognitively intact: 116.88 PD & cognitive symptoms: 128.76	PD cognitively intact: Mean SCOPA-Cog 20.38 PD & cognitive symptoms: Mean SCOPA-COG 18.35	Specialist clinic
Ivory et al.[47]	20 people with PD 20 controls	PD: 71.75 Controls: 70.80	PD: 61.80	PD: 27.45 Controls: 26.80	Community
Johnson et al.[48]	79 people with PD 40 controls	PD: 66.04 Controls: 62.55	PD: 85.80	Cognitive status not reported	Not reported
Koerts et al.[56]	39 people with PD 24 controls	PD: 63.5 Controls: 63.0	PD: 55.2	PD: 27.5 Controls: 27.5	Specialist clinic
Kudlicka & Hindle[51]	65 people with PD 43 controls	PD: 70.11 Controls: 72.02	PD: 71.97	PD: 29.48 Controls: 28.63	Specialist clinic
Lehrner et al.[52]	211 controls 280 SCD 28 cognitive intact PD 58 non-amnestic PD-MCI 10 amnestic PD-MCI 43 AD 137 amnestic MCI 181 non-amnestic MCI	Controls: 66.0 SCD: 64.0 Cognitively intact PD: 67.0 Non-amnestic PD-MCI: 69.0 Amnestic PD-MCI: 69.0 AD: 74.0 Amnestic MCI: 70.0 Non-amnestic MCI: 67.0	Not reported	Median MMSE - Controls: 29.0 SCD: 29.0 Cognitively intact PD: 29.0 Non-amnestic PD-MCI: 28.0 Amnestic PD-MCI: 27.0 AD: 25.0 Amnestic MCI: 27.0 Non-amnestic MCI: 28.0	Specialist clinics
Mack et al.[27]	17 PD with ICD 17 PD without ICD	PD with ICD: 61.1 PD without ICD: 63.8	PD with ICD: 157.2 PD without ICD: 122.4	PD & ICD: 28.7 PD without ICD: 28.5	Specialist clinics
Marino et al.[58]	58 people with PD	PD: 68.3	PD: 82.56	Cognitive status not reported	Not reported
McNamara et al.[60]	22 PD 15 AD 141 controls.	PD: 61.3 AD: 65.1 Controls: 56.4	Not reported	PD: excluded if dementia present AD: mild to moderate dementia	Not reported
Oh-Lee et al.[20]	22 people with PD 22 age matched controls 46 young controls	PD: 71.50 Age matched controls: 68.41 Young controls: 20.65	PD: 102.24	PD: 25.36 Age matched controls: 26.55 Young controls: 27.87	Community
Orfei et al.[22]	197 PD cognitively intact 136 multi-domain PD-MCI 5 single-domain PD-MCI 47 mild PDD	PD cognitively intact: 62.6 PD multi-domain MCI: 68.7 PD single-domain MCI: 65.4 Mild PDD: 73.4	PD cognitively intact: 49.2 PD multi-domain MCI: 67.2 PD single-domain MCI: 50.4 Mild PDD: 108	PD cognitively intact: 29.2 Single-domain PD-MCI: 28.0 Multi-domain PD-MCI: 27.1 Mild PDD: 20.7	Specialist clinic
Seltzer[59]	32 people with PD 31 people with AD	PD: 72.2 AD: 76.6	PD: 64.8 AD: 32.4	PD: 23.4 AD: 18.5	Specialist clinic
Sitek et al.[55]	45 PD	PD: 64.98	PD: 96	PD: 27.72	Specialist clinic

(Continues)

TABLE 1 (Continued)

Study	Study groups	Age (Years[a])	Disease duration (Months[a])	Cognitive status (Mean MMSE unless otherwise stated)	Recruitment source
Sitek et al.[57]	21 mild PD 25 advanced PD 23 HD 20 cervical dystonia	Mild PD: 64.67 Advanced PD: 65.68 HD: 49.83 Cervical dystonia: 51.75	Mild PD: 52.0 Advanced PD: 145.9 HD: 88.2 Cervical dystonia: 100.2	Mild PD: 27.8 Advanced PD: 26.5 HD: 26.1 Cervical dystonia: 28.0	Specialist clinic
Smith et al.[54]	16 PD 16 controls	PD: 72.56 Controls: 74.12	PD: 96.72	PD: 28.25 Controls: 28.56	Not reported
Souchay et al.[53]	16 PD 16 controls.	PD: 67.25 Controls: 69.62	PD: 60.6	PD: 26.62 Controls: 28.01	Specialist clinic
Trenado et al.[42]	10 PD 10 controls.	PD: 82.8 Controls: 64.3	PD: 93.6	PD: mean Mattis dementia rating scale 139.3	Not reported
Yu et al.[49]	25 tremor-predominant PD 30 akinetic-type PD 30 controls	PD-tremor: 62.64 PD-akinetic: 62.33 Controls: 64.2	PD-tremor: 48.48 PD-akinetic: 39.24	PD-tremor: 28.36 PD-akinetic: 28.23 Controls: 28.43	Specialist clinic
Zec et al.[24]	27 PD-MCI 18 AD 28 controls	Not reported	Not reported	PD-MCI: MMSE > = 26 AD: MMSE <15 Controls: cognitively intact	Not reported

Abbreviations: ICD, Impulse Control Disorder.
[a]Age and disease duration are given as mean values unless otherwise stated. The recruitment avenue for patients (not healthy controls) is shown.

cognitive and neuropsychiatric symptoms. The findings for awareness of cognitive and neuropsychiatric symptoms are reported in the current publication. In order to capture the extent of the literature a range of search terms were used. Either "Parkinson's" or "Lewy body" were used as a term to denote the disease of interest, combined with either "insight", "anosognosia" or "metacognition" to denote the neuropsychological area of interest. PubMed and Web of Science searches were conducted between January and March 2019. An additional MeSH term search was carried out in June 2020, using search terms of "Parkinson's disease" or "Lewy body" to denote the disease of interest, combined with either "metacognition" or "agnosia" or "awareness" or "neuropsychology". Studies published in English and including human participants with PD (of any cognitive status) and/or DLB, and reporting a measure of symptom awareness were deemed eligible. Duplicates were excluded. 1747 publications were identified, with 366 retained after title screening, and 97 after abstract screening. 26 relevant publications detailing investigations of symptom awareness in people with PD or DLB were then selected after a full review of the text. An additional 28 studies were identified by manual screening of references from the selected publications. A PRISMA systematic review flow diagram[17] and a PRISMA checklist are available in supplemental data.

Conference abstracts were included if these were indexed for MEDLINE and included essential study data such as group sizes and results data. Data was then extracted following a standardised format, capturing group sizes and diagnosis, baseline group age and cognitive functioning, the nature of the measure of symptom awareness, and study findings. The medication status during assessment was dichotomised to being ON medication, referring to participants being within the therapeutic window of their dopaminergic medication, or OFF medication, referring to participants being out with the therapeutic phase of their dopaminergic medication.

3 | RESULTS

3.1 | Literature search

A total of 1747 publications were identified from the search. 366 were retained following title screening, 97 after abstract screening and 26 after full text review. A further 28 relevant publications were identified from references in screened literature. These were then sub-divided according to whether their primary objective was investigation of awareness of cognitive symptoms (Table 1; 21 publications), neuropsychiatric symptoms (Table 4; 18 publications), or motor symptoms (reported elsewhere[18]; 15 publications). Key findings of studies of awareness of cognitive symptoms are shown in Table 2, and of awareness of neuropsychiatric symptoms in Table 5.

3.2 | Participant groups

Most studies of cognitive symptom awareness actively excluded participants with dementia, and no studies including participants with DLB were found (Table 1). Only 1 study recruited people with PDD, whilst 4 included a specific PD-MCI group. Review of the mean global cognitive scores of studies of cognitive symptom awareness shows that many are likely to have included individuals with PD-MCI or mild PDD. Basic et al.[19] reported a mean Mini-Mental State Examination (MMSE) of 26.1 (SD 2.76) in their PD group, and acknowledged that

TABLE 2 Awareness of cognitive symptoms: key findings

Study	Key findings
Baran et al.[21]	FoK judgements at chance level in PD-MCI group, with evidence of over-confidence. FoK linked to executive functioning in controls but not in people with PD-MCI.
Basic et al.[19]	PD group self-rated their memory for new information as more impaired than their carers did, suggesting that subtle memory issues may not be noticed by carers.
Castro et al.[50]	Subjects who self-reported cognitive problems had intact cognition but more depressive & neuropsychiatric symptoms. Those without subjective problems had objective cognitive impairment, suggesting altered awareness in both groups.
Ivory et al.[47]	PD subjects had intact memory awareness & FoK judgements, despite mild memory impairments.
Johnson et al.[48]	Those with PD self-reported intact meta-memory but made less use of strategies to aid memory (especially external strategies for example, diaries, environmental cues).
Koerts et al.[56]	People with PD showed variable insight into executive functioning problems: some over-estimated & others under-estimated their performance.
Kudlicka & Hindle[51]	People with PD & executive dysfunction showed impaired performance monitoring, with over confidence. People with PD & intact executive function rated themselves as being more impaired than informants did.
Lehrner et al.[52]	Those with amnestic MCI (with or without PD) had worse awareness of cognitive functioning than controls, but not as severe as AD subjects. Over-estimation of memory function correlated with reduced cognition (particularly memory), increasing age, and lower levels of depression.
Mack et al.[27]	Those with ICD reported more executive dysfunction, despite performing at same level as those without ICD. Those with ICD showed greater insight & a trend towards greater self-reflectiveness.
Marino et al.[58]	Measures of perceived cognition from the PDQ-39 moderately associated with Geriatric Depression Scale (GDS) scores. No correlations between performance on tests of memory or frontal lobe function and GDS scores or self-perceived cognition ratings on the PDQ-39. MMSE scores correlated with the perceived cognition item of PDQ-39, but not with scores on the Mattis DRS. Self-perceived cognition appears to be more influenced by mood than cognitive ability.
McNamara et al.[60]	People with PD showed impaired monitoring of speech errors compared to healthy controls, although this was not as severe as in AD subjects. Naming ability negatively correlated with the number of uncorrected errors.
Oh-Lee et al.[20]	"Tip of the tongue" metamemory ability was not impaired in those with PD.
Orfei et al.[22]	Loss of awareness for cognitive impairment was associated with lower depressive symptoms in cognitively intact PD & multi-domain PD-MCI. A diagnosis of anosognosia on the Anosognosia Questionnaire-Dementia was made in 36% of PDD and 16% of the multi-domain PD-MCI group.
Seltzer[59]	PD group unaware of social and self-care symptoms, but retained insight into cognition. Level of unawareness in PD patients correlated with cognitive functioning.
Sitek et al.[55]	No discrepancy between patient & carer rating of overall memory functioning. Patient & carer ratings of overall memory functioning moderately consistent with objective memory performance. Patients over-estimated memory dysfunction on individual memory items; associated with poorer objective memory function and cognitive control, greater PD severity & depression.
Sitek et al.[57]	Those with mild PD rated their executive function as better than their informants did, whereas the converse was true for people with advanced PD. People with advanced PD underperformed on a test of executive function, whereas those with mild PD performed normally.
Smith et al.[54]	People with PD were inaccurate when predicting how they would perform on a prospective memory task, but accurate at rating their performance after the task. This suggests impaired knowledge of memory ability, but intact monitoring ability.
Souchay et al.[53]	Both people with PD & controls slightly under-estimated episodic memory ability. People with PD had reduced FoK accuracy. This suggests that memory knowledge is intact in PD but monitoring of memory performance is impaired.
Trenado et al.[42]	On a reversal learning task, accuracy and confidence correlated in controls and people with PD when OFF medication. When OFF medication participants had reduced memory awareness.
Yu et al.[49]	FoK on an episodic memory task reduced in people with akinetic/rigid PD, but intact in those with tremor predominant PD. FoK related to memory performance in the akinetic/ rigid group, and executive function in the tremor predominant group.
Zec et al.[24]	AD participants self-rated themselves as being less impaired than control, but were actually more impaired. PD group tended to rate themselves as more impaired than independent raters did.

Abbreviations: FoK, Feeling of Known; Mattis DRS, Marris Dementia Rating Scale, PDQ-39, Parkinson's Disease Questionnaire-39.

some participants likely had mild dementia. Oh-Lee et al.[20] recruited a PD group with a mean MMSE of 25.36 (SD 2.80), a value which suggests the group may include people with PD-MCI and PDD.

Definitions of PD-MCI varied; Baran et al.[21] recruited subjects with performance in 2 cognitive domains 1.5 standard deviations below age and education predicted norms, Cambridge Dementia

TABLE 3 Awareness of cognitive symptoms: study quality summary

Study	Measure of awareness	Study quality summary
Baran et al.[21]	Feeling of Knowing judgements	Case-control study. Small group sizes, well characterised. Participants excluded if MMSE<24. Medication status during assessment not stated.
Basic et al.[19]	Brief questions about subjective changes to cognition	Case-control study. Good group sizes. Limited, subjective assessment of awareness. Medication status during assessment not stated.
Castro et al.[50]	Self-report of cognitive symptomatology	Case-control study. Small group sizes, well characterised. Medication status during assessment not stated.
Ivory et al.[47]	Feeling of Knowing judgements	Case-control study. Small group sizes; PD severity not reported. PD participants assessed ON medication.
Johnson et al.[48]	Metamemory In Adulthood questionnaire (self-report)	Case-control study. Good group sizes but limited characterisation. Medication status during assessment not reported.
Koerts et al.[56]	Discrepancy scores: subject vs informant ratings on the Dysexecutive questionnaire	Case-control study. Good group sizes; well characterised; excluded if MMSE<24. PD participants assessed ON medication.
Kudlicka & Hindle[51]	Discrepancy scores: subject vs informant ratings on the BRIEF-A executive function questionnaire	Case-control study. Good group sizes, well characterised, excluded if MMSE<24. PD participants assessed when ON medication.
Lehrner et al.[52]	Memory self-rating vs objective performance on the Neuropsychological Test Battery Vienna	Case-control study. Good group sizes; PD severity not stated. Medication status of PD subjects during assessment not stated.
Mack et al.[27]	Discrepancy scores: subject vs informant ratings on DEX & Everyday Memory Questionnaire-Revised	Case-control study; small group sizes. Well characterised; excluded if MMSE<24. PD participants assessed whilst ON medication.
Marino et al.[58]	Comparison of self-rated cognition on PDQ-39 items and GDS score with performance on: MMSE, Mattis DRS, COWA, Animal naming, Stroop, TMT & HVLT.	Cross-sectional study with no control group. Cognitive status & recruitment source not stated. Medication status and PD severity not stated. Those with clinical depression excluded.
McNamara et al.[60]	Boston Cookie Theft task with measurement of corrected & uncorrected speech errors.	Case-control study; small group sizes. Limited characterisation; those with dementia excluded. Medication status during assessment not stated.
Oh-Lee et al.[20]	Tip of the Tongue metamemory task & motor timing task.	Case-control study; small group sizes; limited group characterisation. PD subjects assessed ON medication.
Orfei et al.[22]	Discrepancy scores: subject vs informant ratings on the Anosognosia Questionniare - dementia	Case-control study; good group sizes, well characterised. Medication status of PD subjects during assessment not stated.
Seltzer[59]	Discrepancy scores: subject vs informant ratings on Patient Competency Rating Scale	Case-control study; medium group sizes; well characterised groups. Medication status of PD subjects during assessment not stated.
Sitek et al.[55]	Discrepancy scores: subject vs informant ratings on Self-Rating Scale of Memory Functions	Cross-sectional study; medium group size; well characterised. Those with MMSE<23

TABLE 3 (Continued)

Study	Measure of awareness	Study quality summary
		excluded. Medication status of PD subjects during assessment not stated.
Sitek et al.[57]	Discrepancy scores: subject vs informants on Dysexecutive function questionnaire	Case-control study; small group sizes; well characterised. Medication status of PD subjects during assessment not stated.
Smith et al.[54]	Prospective time or event-based memory tasks & self-ratings of prospective & retrospective memory.	Case-control study; small group sizes; well characterised. Medication status of PD subjects during assessment not stated.
Souchay et al.[53]	Prediction of performance on a word pair association task, & Feeling of Knowing judgements.	Case-control study; small groups; limited characterisation; excluded if MMSE<24. PD participants assessed ON medication
Trenado et al.[42]	Reversal learning task with confidence ratings after each trial.	Case-control study; small groups; well characterised; excluded if Mattis Dementia Rating score < =132 or Beck Depression Inventory > = 18. PD participants assessed ON and OFF medication.
Yu et al.[49]	Feeling of Knowing episodic memory task.	Case-control study; well characterised; small group sizes; excluded if MMSE<24. Medication status of PD participants during assessment not stated.
Zec et al.[24]	Discrepancy scores: subject vs informant on Dysexecutive Self-Rating & Dysexecutive Independent Rating	Case control study; small group sizes; PD subjects & controls excluded if MMSE<26. Medication status of PD participants during assessment not stated.

Abbreviations: BRIEF-A, Behaviour Rating Inventory of Executive Function, Adult version; COWA, Controlled Oral Word Association Test; DEX, Dysexecutive Questionnaire; HVLT, Hopkins Verbal Learning Test; TMT, Trail Making Task.

Rating Scale (CDR) of 0.5 and MMSE of 24 or greater; Orfei et al.[22] applied the Movement Disorder Society criteria for PD-MCI and PDD. Martínez-Martín et al.[23] used the Unified Parkinson's Disease Rating Scale (UPDRS), Pfeiffer's Short Portable Mental Status Questionnaire, and neurologist and care-giver ratings, whilst Zec et al.[24] reported PD-MCI subjects as having an MMSE of > = 26, without providing further details of their definition of PD-MCI.

Studies of neuropsychiatric symptoms included participants with PDD (2 studies), DLB (2 studies), cognitively intact people with PD (7 studies) and groups containing people with PD with varied levels of cognitive functioning (8 studies). The mean MMSE (or similar global cognitive measure) is shown in Table 4. Various global cognitive measures were used; the MMSE (9 studies[1,25-32]), the Mattis Dementia Rating Scale (Mattis-DRS; 2 studies[33,34]), and in one study each the: CDR,[35] mini-mental Parkinson's,[36] National Adult Reading Test IQ (NART IQ),[37] Telephone Interview for Cognitive Status (TICS),[38] Functional Dementia Scale (FDS),[39] Dementia Rating Scale-2 (DRS-2).[40] 1 study described 15% of participants as having dementia, but did not give further details.[41]

Study group sizes were highly variable: in the cognitive studies: the smallest group numbered 10 participants[42] and the largest 197,[22] whilst in neuropsychiatric studies this ranged from 15[35] to 250[43] participants.

3.3 | Techniques for assessing symptom awareness

Various methods were used to quantify symptom awareness (Tables 3 and 6). Techniques used to evaluate awareness of cognitive functioning included discrepancy scores between self and informant or clinician ratings, comparison of self-ratings and objective performance on cognitive tasks, Feeling of Knowing tasks or Judgement of Learning tasks, and self-report questionnaires. When evaluating insight into neuropsychiatric symptoms, direct self-report was often used (ie, directly enquiring if the person thought their hallucinations were real or not; 9 out 18 studies), followed by discrepancy scores (where the participant is asked to rate their own ability, symptom or performance, and this score compared to that given by a knowledgeable informant or expert rater; 8 studies) on a variety of questionnaires evaluating different neuropsychiatric symptoms. 1 study relied on informant reports.

TABLE 4 Studies of altered awareness of neuropsychiatric symptoms in PD and DLB

Study	Study participants	Age (Years[a])	Disease duration (Months[a])	Cognitive status (Mean MMSE unless otherwise stated)	Recruitment source
Aarsland et al.[65]	235 people with PD	73.6	109.2	24.5	Community & Specialist clinics
Ballard et al.[25]	42 DLB 30 AD	LBD group: 73.6 AD group: 81.7	Not stated	DLB group: 14.9 AD group: 13.9	Specialist clinics
Fenelon et al.[36]	52 PD with feeling of presence	67.0	138.0	Mean Mini Mental Parkinson's cognitive 25.4 (maximum score 32)	Specialist clinic & inpatients
Goetz et al. 2006[41]	48 PD & hallucinations with insight	69.0	144.0	15% had dementia at baseline (no other cognitive data reported)	Specialist clinics
Goetz et al. 2010[26]	89 PD	67.7	123.6	26.9	Specialist clinics
Graham et al.[37]	97 PD no hallucinations 32 PD with hallucinations	PD no hallucinations: 55.2 PD with hallucinations: 56.6	PD no hallucinations: 79.2 PD with hallucinations: 112.8	Mean NART IQ PD no hallucinations: 111.7 PD with hallucinations: 108.6	Specialist clinics
Holroyd et al.[38]	98 PD	67.5	116.4	Mean TICS (normal range 31 to 41) PD with hallucinations: 29.9 PD without hallucinations: 33.0	Specialist clinics
Llebaria et al.[33]	28 PD no hallucinations 11 PD & minor hallucinations 10 PD, major hallucinations with insight 8 PD, major hallucinations, no insight	No hallucinations: 72.7 Minor hallucinations: 71.1 Major hallucinations with insight: 75.8 Major hallucinations, no insight: 79.2	No hallucinations: 94.8 Minor hallucinations: 91.2 Major hallucinations with insight: 102.0 Major hallucinations, no insight: 122.4	Mean Mattis DRS No hallucinations: 130.1 Minor hallucinations: 128.4 Major hallucinations with insight: 121.2 Major hallucinations, no insight: 110.2	Specialist clinics
Mack et al.[43]	250 PD	66.3	93.6	28.3	Specialist clinics
Mathias[39]	30 PD 30 controls	63.7	64.8	FDS mean score 24.5 (little or no functional decline due to dementia)	Community & Specialist clinics
McKinlay et al.[28]	43 PD	66.5	72.0	28.5	Community
Mikos et al.[40]	37 PD 12 controls	PD group: 68.97 Controls: 66.90	77.88	DRS-2 scores: PD group: 138.89 Controls: 140.62	Specialist clinics
Noe et al.[35]	16 DLB 15 PDD 16 AD	PDD: 73.5 DLB: 72.7 AD: 71.5	PDD: 64.4 DLB: 35.25 AD: 30.75	77% of participants CDR 1 17% of participants CDR 2 No between group differences on CDR	Specialist clinics
Papapetropoulos et al.[29]	70 PD	64.3	110.4	25.6	Specialist clinics
Serrano & Serrano[30]	70 PD	70.5	90.0	26.7	Specialist clinics
Schiehser et al.[34]	51 PD plus carer dyads	PD: 69.7 Carers: 66.9	PD: 80.88	No evidence of dementia & Mattis DRS score > =130	Specialist clinics
Starkstein et al.[31]	33 PDD 33 AD	PDD group: 70.3 AD group: 71.00	Not stated	PDD group: 20.3 AD group: 20.3	Specialist clinics
Valentino et al.[32]	48 PD	72.21	72.24	22.83	Specialist clinic

[a]Age and disease duration are given as mean values unless otherwise stated.

TABLE 5 Awareness of neuropsychiatric symptoms: key findings

Study	Key findings
Aarsland et al.[65]	9.8% had hallucinations with insight; 6.0% had hallucinations/delusions without insight. Loss of insight was associated with cognitive decline, PD severity, and depression. Altered insight best explained by MMSE score.
Ballard et al.[25]	Hallucinations were commoner in DLB (93% of subjects vs 27% of those with AD). 63% of people with DLB and hallucinations had no insight into these. No correlations between MMSE, dopaminergic or anticholinergic medication use and hallucinations.
Fenelon et al.[36]	77% of those reporting feeling of presence retained insight, whilst insight was lost in 23%. Those without insight had significantly higher mean BDI-II scores (19.8 vs 25.4) & lower MMP (26.2 vs 22.8).
Goetz et al. 2006[41]	81% of those with PD and hallucinations lost insight over a median of 2 years. The presence of dementia at baseline did not increase the likelihood of conversion to hallucinations without insight. Most of those who retained insight had a reduction in PD medication prescription.
Goetz et al. 2010[26]	22.5% had hallucinations with insight at baseline, whilst 10.1% had hallucinations without insight. Baseline PSQI score did not correlate with MMSE or UPDRS motor score. Higher severity scores on the Rush Hallucination Inventory were associated with lower MMSE scores and higher UPDRS motor scores at baseline. Hallucination prevalence and severity increased over time. Only 4 subjects were hallucination free at final 10 year follow up.
Graham et al.[37]	70.4% of those with hallucinations retained insight. Other data not broken down by insight status.
Holroyd et al.[38]	26.5% of the group had visual hallucinations (N = 26). Of those with hallucinations, 46.2% had full insight, 38.5% had partial or variable insight, and 15.4% had no insight. Cognitive scores did not vary between these sub-groups.
Llebaria et al.[33]	No cognitive differences emerged between those without hallucinations & those with minor hallucinations. Action verbal fluency was significantly worse in those with major hallucinations (with insight) & those with minor hallucinations. Posterior cortical score & clock copying were significantly worse in those with major hallucinations without insight than people with major hallucinations & retained insight.
Mack et al.[43]	20.4% of subjects with PD had hallucinations or illusions with insight 5.6% had hallucinations or illusions with no insight.
Mathias[39]	Good agreement on reporting of neuropsychiatric symptoms between people with PD and informants. The only significant discrepancy was on reporting of depressive symptoms. No evidence of loss of awareness.
McKinlay et al.[28]	Low level of agreement between subjects with PD and informants: 45.8% for depression, 45% for apathy, 28.6% on hallucinations, 26.9% on sleep issues, 6.7% for anxiety. Most hallucinations were not reported by carers.
Mikos et al.[40]	No group difference in self & informant ratings, but variable discrepancies in individuals. Higher levels of apathy in those with PD who under-reported reduced expressivity.
Noe et al.[35]	Psychosis (delusions or hallucinations) were commoner in DLB (31.3%) than PDD.
Papapetropoulos et al.[29]	31 participants had hallucinations. 35.5% retained insight, 25.5% had intermittent insight (sometimes thinking hallucinations were real), 32.3% did not have any insight, and 9.7% were unsure if hallucinations were real or not. There was no difference in MMSE scores between those with different levels of insight.
Serrano & Serrano[30]	Low correlation between ratings by people with PD and their informants on the Neuropsychiatric Inventory: Questionnaire.
Schiehser et al.[34]	Moderate agreement was seen on the Frontal Systems Behavior Scale (FrSBe) for prediagnosis ratings of apathy and executive dysfunction (ICC between 0.4 and 0.59) but poor agreement on disinhibition prediagnosis (ICC <0.4). Patients reported greater symptoms prediagnosis than informants did. Higher Ldopa usage was associated with greater discrepancy on apathy scores, for both prediagnosis and current apathy. No other associations found between patient factors (mood, cognition, motor symptoms, Ldopa usage) & discrepancy scores for disinhibition, executive function or apathy. Higher Ldopa usage associated with higher apathy ratings. Higher levels of medication usage associated with carer ratings of executive dysfunction. Higher carer burden & depression levels were significantly associated with greater discrepancies in prediagnosis & current apathy ratings. Higher levels of carer burden associated with greater discrepancy in prediagnosis & current disinhibition ratings. No associations between care burden or depression and executive function ratings.
Starkstein et al.[31]	More severe anosognosia & disinhibition in AD compared to PDD. Higher prevalence of major depression & worse visual reasoning in PDD.
Valentino et al.[32]	Patients' self-rated apathy scores were higher than carer ratings - both for the total AES score & the emotional apathy sub-score. No differences were found between self & carer ratings on the cognitive & behavioural apathy sub-scores. Self & carer apathy ratings positively correlated with severity scores on the UPDRS-3, H&Y & NPI-P. An inverse correlation was found between self-rated apathy scores & MMSE & FAB scores (but no correlations emerged with carer scores). Neither self-ratings or carer ratings correlated with HDRS scores.

Abbreviations: AES, Apathy Evaluation Scale; FAB, Frontal Assessment Battery; HDRS, Hamilton Depression Rating Scale; H&Y, Hoehn and Yahr scale; NPI-P, Neuropsychiatric Inventory - patient version; PSQI, Pittsburgh Sleep Quality Index.

TABLE 6 Awareness of neuropsychiatric symptoms: study quality summary

Study	Mesure of awareness	Study quality summary
Aarsland et al.[65]	Self-reports on the UPDRS Thought Disorder scale	Cross-sectional study. No comparator group. Large, well characterised population; MMSE reported. Medication status at time of assessment not stated.
Ballard et al.[25]	Discrepancy scores: subject vs informant on a study specific questionnaire	Case control study: DLB and AD groups. Well characterised groups. MMSE reported. Medication status at time of assessment not stated.
Fenelon et al.[36]	Structured questionnaire for feeling of presence & insight. BDI-II & MMP.	Cross-sectional study. Good group sizes, well characterised. Insight only report in terms of BDI-II scores & not analysed in reference to other variables.
Goetz et al.[41]	Self-reports on the UPDRS Thought Disorder scale	3 year longitudinal study. No comparator group. Cognitive evaluation performed, but only presence or absence of dementia reported. Medication status at time of assessment not stated.
Goetz et al. 2010[26]	UPDRS modified thought disorder stratum	10 year longitudinal study. No comparator group. Good group size. MMSE reported, Medication status at time of assessment not stated.
Graham et al.[37]	Psychosis questionnaire (self-report), UPDRS, neuropsychological assessment.	Longitudinal study but only very limited baseline data on insight presented. Good group size, well characterised. Motor assessments carried out ON medication. Medication status during other assessments not stated.
Holroyd et al.[38]	Self-report of hallucinations & insight into these. TICS, UPDRS, GDS.	Cross-sectional study. Moderate group size, well characterised but only limited data reported specifically in relation to insight. Those with delirium or non-PD related psychosis excluded. Medication status during assessment not stated.
Llebaria et al.[33]	Self-report on the MDS-UPDRS. Mattis DRS, Neuropsychological assessment.	Cross-sectional study. Small sub-group sizes. Well characterised groups. Assessed ON medication (if motor fluctuations present).
Mack et al.[43]	Self-reports on a study specific questionnaire	Cross-sectional study. No comparator group. Large, well characterised population; MMSE reported. Medication status at time of assessment not stated.
Mathias[39]	Discrepancy scores: subject vs informant on the NBAP, DEX & NRS-Revised	Case-control study. Control group matched for age, education and IQ. Small group sizes. PD group described as not having dementia, but cognitive results not reported. Medication status at time of assessment not stated.
McKinlay et al.[28]	Discrepancy scores: subject vs informant on the Frontal Systems Behaviour Scale	Cross-sectional study. No comparator group. Well characterised but small group: MMSE reported. PD group assessed ON medication.
Mikos et al.[40]	Discrepancy scores: subject vs informant on the Berkeley Expressivity Questionnaire	Case control study. Controls matched for age, cognition & education but more depressive symptoms in PD group. Small group sizes. PD group assessed ON medication.

TABLE 6 (Continued)

Study	Mesure of awareness	Study quality summary
Noe et al.[35]	Informant reports	Case control study: DLB, PDD and AD groups. Small group sizes. Neuropsychology results reported. Medication status at time of assessment not stated.
Papapetropoulos et al.[29]	University of Miami PD Hallucinations Questionnaire	Questionnaire validation study. No comparator group. Good group size, well characterised. Medication status at time of assessment not stated.
Serrano & Serrano[30]	Discrepancy scores: subject vs informant on the Neuropsychiatric Inventory	Cross-sectional study. Good group size but limited participant characterisation. PD group assessed ON medication.
Schiehser et al.[34]	Discrepancy scores: subject vs informant on prediagnosis & current ratings using FrSBe. Neuropsychological evaluation & carer ZBI.	Cross-sectional study. Good group size & participant characterisation. Medication status during assessment not stated.
Starkstein et al.[31]	Discrepancy scores: subject vs informant on the Anosognosia questionnaire-dementia	Case control study (PDD and AD groups). Neuropsychology results reported. PD group assessed ON medication.
Valentino et al.[32]	Discrepancy scores: Apathy Evaluation Scale carer and participant reports. Neuropsychological evaluation	Cross-sectional study. Moderate group sizes. Those with "overt cognitive impairment" excluded, but mean MMSE 22.83 (SD 4.71), indicating the presence of participants with significant cognitive impairment. Medication status of participants during testing not stated.

Abbreviations: BDI-II, Beck Depression Inventory version 2; MMP, Mini-mental Parkinson's; NBAP, Neuropsychology Behavior and Affect Profile; NRS-R, Neurobehavioural Functioning Scale- Revised version.

3.4 | Timing of assessments

Dopaminergic therapies may impact on cognitive[44] and neuropsychiatric symptoms,[45] but most studies did not report whether participants were assessed in the ON or OFF medication state (Tables 3 and 6). Of the studies of awareness of cognitive symptoms, 14 did not report the participants' medication state at the time of assessment, 6 assessed whilst ON medication, and 1 whilst ON and OFF medication. Studies of awareness of neuropsychiatric symptoms did not state medication timing in 13 instances, whilst 5 studies assessed participants ON medication.

4 | DISCUSSION

4.1 | Awareness of cognitive symptoms

Cognitive decline is often seen in people with PD, with older individuals being most vulnerable.[46] Studies of awareness of cognitive symptoms have variable results, with some showing intact awareness,[20,47-49] and others finding under or over-estimation of cognitive functioning.[19,24,50,51] The majority of studies have focused on different aspects of memory.[19-21,42,47-49,52-55] Only five studies were found which focused on awareness of executive function,[24,27,51,56,57] four used a global measure of cognition[22,50,58,59] and one investigated awareness of spoken language functioning.[60]

It is probable that awareness of memory functioning varies across the disease course. Lehrner et al.[52] examined a range of groups across the spectrum of cognitive decline, including Subjective Cognitive Decline (SCD), PD with non-amnestic MCI or amnestic MCI, non-PD related non-amnestic or amnestic MCI and AD. A gradient of worsening awareness for cognition performance was found, with the SCD group under-estimating their performance, non-amnestic MCI persons with or without PD having a level of awareness indistinguishable from controls, whilst those with amnestic MCI or AD showed reduced awareness, with the AD group being most severely affected. Orfei et al.[22] made similar findings, with significant loss of awareness of cognition being seen in 36% of those with PDD and 16% of those with multi-domain MCI-PD, whilst awareness was retained by cognitively intact PD participants and those with single-domain PD-MCI.

Awareness of cognitive functioning can be sub-divided into a number of processes: real-time performance monitoring; registration of positive and negative external feedback; comparison of current with past ability; integration and updating of an internal model of one's own abilities, and using this model to consciously report current and predict future performance.[61,62] How these individual aspects of

metacognition are affected by PD and other forms of neurodegeneration remains an area of considerable debate. Participants in two studies were found to have intact global knowledge of their memory ability, but to be inaccurate when estimating performance after a task.[53,55] This suggests impaired memory performance monitoring. A further study compared awareness of memory function on time and event-based memory tasks.[54] Both performance and self-ratings on the event-based memory task were at control level. However, people with PD were inaccurate on the time-based memory task and showed impaired pre-task performance predictions, although post-task performance ratings were intact. This implies that awareness of memory ability is task specific and linked to task performance. Impaired self-monitoring was found in a study investigating self-awareness of speech errors, and this correlated with naming ability.[60] People with PD also make less use of "*aide-memoire*" strategies such as diaries or environmental cues,[48] which may reflect a lack of awareness of memory difficulties.

Different aspects of cognitive awareness can be explored using dedicated tasks, such as those probing feeling of knowing (FoK) - an individual's judgement of how likely they are to recognise an item they failed to spontaneously recall. The accuracy of FoK judgements appears to be linked to general cognitive status in PD, with cognitively intact individuals performing normally,[47] in contrast to those with mild cognitive decline, who show significant deficits.[21,53] The PD phenotype may also influence cognitive awareness. Yu et al.[49] explored FoK in tremor predominant PD vs those with an akinetic-rigid phenotype. In the akinetic-rigid group FoK decisions were at chance level, but the tremor predominant group were unimpaired. FoK correlated with memory ability in the akinetic-rigid group, whereas executive function was implicated in those with tremor predominant PD.

As with awareness of mnemonic ability, there is a spectrum of insight into executive functioning. Both over-estimation and under-estimation of executive functioning has been seen.[24,43,51,57] Altered symptom awareness is classically attributed to dysfunction in the fronto-striatal circuits, implying that executive function and working memory are involved. Kudlicka et al. found those with PD and executive dysfunction to have impaired monitoring of their performance on an executive function task.[51] Baran et al. identified significant correlations between executive functioning and accuracy of self-ratings of episodic memory performance in healthy controls, but only modest links in those with PD.[21] This implies that executive function-dependent mechanisms for self-monitoring may be dysfunctional in PD.

Imaging studies of healthy adults implicate the prefrontal cortex (PFC) in awareness of cognitive ability. The medial PFC is postulated to support prospective judgements of performance, whilst the lateral PFC is involved in retrospective evaluations.[63] The anterior cingulate cortex and insula also appear to have a role in cognitive control. As PD progresses the level of dopamine within the corticostriatal and mesocortical loops is progressively reduced. With dopaminergic treatments, the level is boosted, at times potentially to a supra-physiological level. Both hypo- and hyperdopaminergic functioning have been suggested as mechanisms for cognitive and metacognitive dysfunction in PD.

Trenado et al. investigated memory awareness on a reversal learning task in a small group of people with PD, both ON and OFF dopaminergic medication.[42] When OFF medication participants showed reduced confidence in their correct responses, although performance accuracy was unimpaired. Mattay et al. utilised fMRI to investigate the impact of dopaminergic medication on working memory in people with PD.[44] Activity was seen in the prefrontal cortex, pericingulate cortex, anterior cingulate cortex and parietal cortex during the task, with significantly more activation in the OFF state. However, task accuracy was also worse during the OFF state, suggesting that dopaminergic stimulation improved efficiency within the mesocortical dopaminergic system.

The role of dopamine in self-assessment of cognitive function in healthy adults has been investigated using haloperidol, an antagonist at the D2 autoreceptors which regulate dopamine release (and which theoretically results in a subsequent increase in dopaminergic transmission). During a recognition memory task, low dose haloperidol improved memory accuracy in healthy adults but decreased confidence.[64] This was associated with more disorganised activity in the frontostriatal networks, and a failure to modulate neural activity according to the level of confidence in the decision. This mixture of findings suggests that loss of cognitive awareness is not uniformly affected across the PD spectrum, and that different aspects of awareness may be differentially affected at different stages of the disease. It may be that both low and high dopamine levels have an impact on cognition and cognitive awareness, with suboptimal functioning in different dopaminergic circuits contributing to over and under-confidence in cognition at different disease stages.

Factors such as low mood or adjustment reaction may also negatively affect an individual's perception of their cognition. Castro et al.[50] investigated subjective cognitive symptoms in PD. Those self-reporting cognitive difficulties performed normally on neuropsychological assessments but had higher levels of depressive symptomatology. In contrast, the self-reported cognitively asymptomatic group had impairments on objective cognitive tests. Sitek et al.[55] also found subjective cognitive decline to correlate with depressive symptoms. These findings suggest that self-reported cognitive symptoms may more accurately reflect lower mood than objective cognitive functioning.

4.2 | Awareness of neuropsychiatric symptoms

Hallucinations, depression, apathy and sleep disturbances are all common features of PD, PDD and DLB. Hallucinations (typically visual) are a core diagnostic feature of DLB and are common in PDD; they were the most frequently studied type of neuropsychiatric symptom. Other modalities of hallucinations can occur, and a major limitation of the literature is a frequent failure to delineate the type of hallucination under study, or the grouping together of people experiencing hallucinations or delusions. Of 12 studies including people with hallucinations, 8 included hallucinations in any domain, 3 were specifically of visual hallucinations, and 1 feeling of presence.

Studies of hallucinations were often not directly comparable due to differences in study populations. Mack et al.[43] interviewed 250 people with PD and normal cognition, and found 26% to have hallucinations or illusions, the majority with retained insight. Only 15.8% of people reported hallucinations in a cohort of people with PD examined by Aarsland et al.[1] Within the Aarsland et al. study cognitive status clearly played a role, with the group as a whole having a mean MMSE of 24.5, compared to 18.1 in the subgroup of people with hallucinations and retained insight, and falling further to 13.5 of those with psychosis.[65] A longitudinal study of the clinical course of hallucinations found that the majority of people with PD lose insight over time,[41] although counterintuitively, an individual's baseline cognitive status did not appear to predict the loss of awareness. The large majority of those with feeling of presence hallucinations (a phenomenon considered to be one of the earliest types of psychotic symptoms in PD) retained insight.[36] Altered awareness has been association with greater depressive symptoms,[36] global cognitive dysfunction and higher severity of motor symptoms,[33,41] although several studies did not find an association between insight into hallucinations and cognition.[29,38]

In those with DLB hallucinations are very common (unsurprisingly, as their presence forms part of the DLB diagnostic criteria), with estimates for the presence of psychosis varying from 31.3%[35] to 63%.[25]

A link between dopaminergic medication load and hallucinations without awareness has not emerged experimentally. Rather, the presence and severity of cognitive impairment and depression is positively associated with psychotic symptoms. However there are a number of issues which can potentially confound study results. Firstly, physicians may reduce or avoid dopaminergic agents in response to the emergence of psychotic symptoms; Goetz et al.[41] reported an association between retained awareness and a reduction in medication, and such a pre-emptive medical approach may confound observational study results. Secondly, ascertaining whether or not an individual has neuropsychiatric symptoms (with or without awareness) depends upon reports from the subject and a knowledgeable informant. Caregiver burden may artificially inflate report of neuropsychiatric symptoms (due to caregiver distress heightening the impact of neuropsychiatric symptoms, and hence greater reporting), whilst subjects may choose to conceal unusual experiences or behaviours due to perceived social stigma, or simply not recall them due to coexistent memory impairment. On direct questionning, McKinlay et al.[28] found patients to report a greater number of hallucinatory experiences than caregivers.

Studies looking at a broader range of neuropsychiatric symptoms have yielded conflicting results. Mathias et al. found good agreement between patient and informant ratings on a number of symptom scales, with the exception of depression, which informants rated as being more severe than the participant themselves did.[39] However other studies have shown significant discrepancies between participant and informant ratings for a range of neuropsychiatric symptoms.[28,30,32,34] Self-ratings of apathy were higher than informant ratings in two studies,[32,34] suggesting that external observers may not be able to fully report changes to internal motivations. Valentino et al.[32] found self-rated apathy severity to correlate with reductions in global cognition, executive functioning and motor ability, whilst Schiehser et al.[34] found higher levels of levodopa use to predict greater discrepancies between self and informant apathy ratings. Greater levels of carer burden and carer depression associated with greater discrepancies on apathy and disinhibition ratings.[34] Whether this means that higher carer burden causes over-rating of symptoms by carers, or that loss of symptom awareness by patients results in greater carer burden (or both) is unclear.

A study of emotional expressivity (the ability to demonstrate feelings, for example, by facial expression) revealed reduced expressivity in those with PD, but with no evidence of loss of awareness of this at the group level.[40] In those individuals who did under-report symptoms, this was found to be associated with a higher level of apathy. This suggests that loss of awareness for some psychiatric phenomena may be inter-linked with wider cognitive functioning. Loss of awareness for neuropsychiatric symptoms appears to be less severe in DLB and PDD when compared to AD.[25,31] However there is a paucity of studies of awareness of neuropsychiatric symptoms other than hallucinations in PD, and the DLB literature is extremely small.

4.3 | Methodological challenges

There are potential methodological issues with the study of cognitive and neuropsychiatric symptom awareness. The commonest measure of awareness used is a discrepancy score, where symptoms are rated by the subject and an informant and the difference between the two taken as a measure of the subject's awareness. This assumes the informant to have perfect insight into the subject's symptoms. Potential confounders include informant bias, either positively by a desire to protect the subject, or negatively by the stress of being a carer. In addition, a subject may experience subtle changes which are not evident to an informant. We found there to be considerable heterogeneity in the cognitive assessment tools used. Not all studies reported the global cognitive status of their participants, and those that did predominantly used a brief screening tool (typically the MMSE). Whilst valuable, the MMSE and similar tools are insufficient to identify subtle cognitive changes, PD-MCI, or domain specific impairments. The Movement Disorders Society has developed detailed testing recommendations for the diagnosis of MCI due to PD, and future studies would benefit from using these more robust criteria.[4,66] Interpreting study findings becomes very challenging when a mixed population of cognitive normal, mildly impaired people and those with dementia are included. This issue also works to obscure the influence of cognitive dysfunction on symptom awareness. A number of studies found links between lower global cognitive scores and poorer awareness of cognitive or neuropsychiatric symptoms, and it is widely accepted that insight into psychotic symptoms tends to worsen in tandem with cognitive decline. Frontal and temporal cortex is heavily implicated in loss of symptom awareness in other neurodegenerative and psychiatric disorders,[13,67] and in loss of awareness of cognitive symptoms in PD. However there is little known about how functioning in specific

cognitive domains influences and interacts with neuropsychiatric symptom awareness. Our understanding of how symptom awareness evolves over time in PD and DLB would be greatly enhanced by work utilising detailed, domain specific neuropsychological assessment, and advanced techniques such as functional neuroimaging.

5 | CONCLUSIONS

The findings from the literature investigating awareness of cognitive and neuropsychiatric symptoms in people with PD are mixed and limited by methodological heterogeneity, but altered symptom awareness is clearly a common phenomenon. A far smaller body of research into awareness in PD exists than in AD. We did not identify any studies of cognitive symptom awareness in people with DLB, and only two investigating neuropsychiatric symptom awareness. Minimal information is available regarding the influence of dopaminergic medications on neuropsychiatric symptom awareness.

Altered awareness in neurodegenerative conditions can greatly exacerbate the impact of the primary condition itself. Those affected may not seek appropriate help and support or refuse essential care. Understanding how symptom awareness changes in PD and DLB, and the impact this has on those affected and their significant others and caregivers is a vital step in the pathway towards developing effective management and therapeutic techniques. Given the chronic nature of these conditions and the high incidence of progressive cognitive decline, future longitudinal studies are needed to accurately establish the neural underpinnings of symptom awareness, the most effective assessment techniques and to ultimately generate novel tools to support those affected.

CONFLICT OF INTEREST

None declared.

ETHICS IN PUBLISHING

The present study does not require ethical approval.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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