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Psychological and neuropsychological services for people with Parkinson's disease

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Contents

Executive summary

- Parkinson's disease (Parkinson's, or PD) is one of the fastest growing neurological conditions, with a range of both motor and non-motor symptoms, which significantly impact upon the lives of those living with the condition.
- Given the complex and multifaceted nature of the condition, we have proposed a matched care framework, recognising the need for multiple psychological support services to work together to support the individual and their family across the course of the condition. Practitioner psychologists will have a key role in providing specialist assessment and intervention, as well as overseeing the management of pathways, training and supervision.
- We have reviewed the current evidence base and practice in assessment, formulation and intervention with people with Parkinson's, and suggestions are made here to support specialist assessment and intervention.
- Despite significant numbers of people with Parkinson's and the need for specialist assessment, there is limited dedicated provision, and suggested service evaluation standards are indicated to benchmark service data in the future.

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Aim and audience

This guidance has been written for practitioner psychologists, mental health professionals and clinical staff working with people with Parkinson's disease to support psychological assessment, formulation and intervention. It is also hoped that this may be of use to NHS service managers, commissioners and policy makers to consider psychological support pathways and models of care. It is suggested that this guidance is read in conjunction with wider guidance provided by the British Psychological Society (BPS) such as *Guidelines for Commissioning NHS Neuropsychological Services* (2024) and the supplementary guidelines on *Workforce, Leadership & Governance in Clinical Neuropsychology Services* (2024). For consistency, these papers outline terminology around 'practitioner psychologist' and 'clinical neuropsychologist' which will also be used within this document.

EQUALITY, DIVERSITY AND INCLUSION

Many people with Parkinson's feel marginalisation, stigma and lack of understanding from wider society, compounded by lack of timely access to services. Within the population of those living with Parkinson's, there are further marginalised communities, and inequalities in access, experience and outcome within the Parkinson's healthcare system. Efforts have been made to consider aspects of inclusive psychological/neuropsychological practice throughout the document and embed this conversation within each section rather than having one stand-alone section to explore the myriad of ways in which marginalised communities can be impacted.

INTRODUCTION

Neurological conditions are the leading source of disability globally, and Parkinson's disease is the fastest growing neurological condition in the world (Dorsey et al., 2018). Currently affecting 145,000 people in the UK and over 10 million people worldwide (Parkinson's Foundation, 2021), these numbers are projected to double over the next 30 years (Rocca, 2018). First described by James Parkinson in 1817 as 'the shaking palsy' (Parkinson, 2002), Parkinson's is a neurodegenerative condition that causes motor symptoms of tremor, rigidity, slowness and loss of balance. Its neuropathological hallmarks are neuronal loss in the substantia nigra and α-synuclein accumulation in intraneuronal inclusions, termed Lewy bodies. There remains no cure, and treatments provide only symptomatic relief. Parkinson's progresses relentlessly to cause increasing disability.

In addition to motor symptoms, people with Parkinson's (pwP) can experience a range of neuropsychological difficulties including cognitive changes, psychosis and impulse control behaviours. These changes can be evident at multiple points during the course of the disease and cause significant distress for pwP, exacerbating disability and reducing quality of life, whilst placing greater burden upon family and support networks. These neuropsychological factors are influenced by both the neurobiological changes that occur in Parkinson's disease and the emotional and systemic impact of living with a progressive condition.

In 2009, the All-Party Parliamentary Group for Parkinson's disease (APPG) wrote that 'mental health symptoms are given less of a priority than physical symptoms of Parkinson's by health and social care professionals' (APPG, 2009). The same year, the BPS Professional Practice Board highlighted inconsistent access to psychological assessment and intervention for pwP across the UK, and the need for further research into the 'psychological management of emotional and cognitive problems associated with PD, with a view to enhancing the provision of clinical

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psychology and clinical neuropsychology input to specialist PD services' (BPS Professional Practice Board, 2009, p.2). Yet in 2018, the APPG reported that the situation had not changed: pwP and health professionals remain 'united in their dissatisfaction with the current state of mental health services for people with the condition'.

One of the main barriers remains provision of services suitably tailored to the needs of pwP (APPG, 2018). Much mental health funding has been focused on the establishment of NHS England's Talking Therapies, an anxiety and depression programme previously known as Improving Access to Psychological Therapies. Although there were hopes of expanding the NHS Talking Therapies programme to include treatment for people with neurological conditions as part of the long-term conditions pathway, this has not been realised.

The paucity of appropriate mental health services leads to poorer outcomes for pwP and greater demand for costly emergency care (Foley & Willis, 2023). These poor outcomes are likely magnified in those from minoritised communities, who are even less likely to be able to access appropriate physical and mental health support and more likely to end up in crisis (Bignall et al., 2019). Indeed, it is increasingly understood that there are racial and ethnic disparities and sociocultural implications of Parkinson's in terms of epidemiology, aetiology and access to treatment (Aamodt et al., 2023). This further compounds the stigma experienced by pwP from minoritised backgrounds and the social deprivation that pwP experience even in the prodromal phase of the condition (Heimrich et al., 2023). Throughout this review, we would ask the reader to hold in mind the limitations in our understanding of the neuropsychological issues and their optimal treatment in pwP from minoritised communities. This is in part because of limitations in representation in published research. A recent review highlighted the lack of recruitment to clinical trials for those from minoritised communities, and lack of accurate reporting on racial distribution and diversity or consideration of sociocultural factors when developing interventions for pwP (Lau et al., 2022).

Another barrier is appropriate guidance. Over the last decade, there have been considerable advances in our knowledge of the impact of Parkinson's neuropsychological issues, their underlying neuropathological mechanisms and individual risk factors, and how they are best assessed and managed. However, these advances have yet to be translated into clinical guidelines and recommendations.

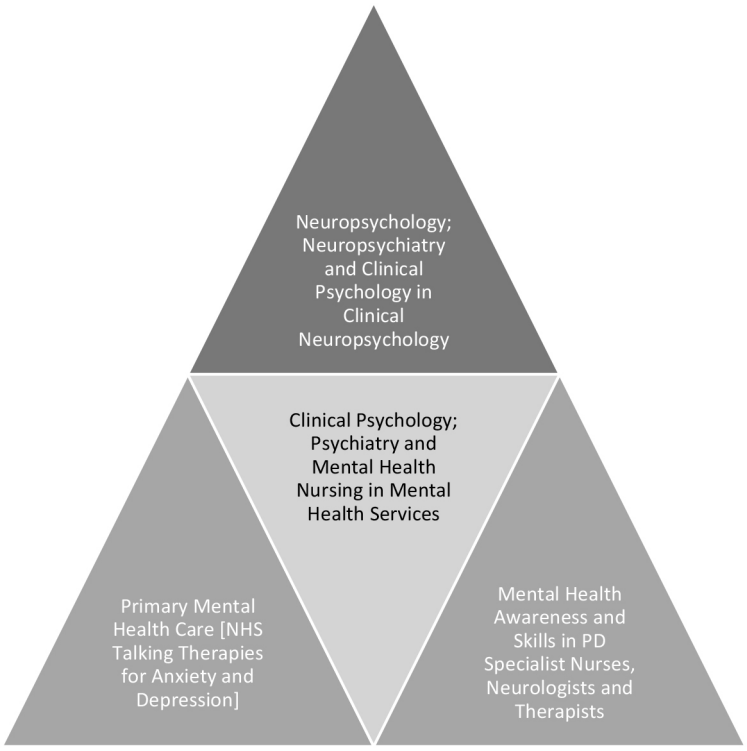
There is an urgent and critical need for appropriate guidance to enable the improvement of psychological services for pwP. Here, we provide an updated summary on each of these important psychological and neuropsychological factors, alongside considerations for their assessment, formulation and intervention. To bring these services together, a matched care model is proposed for how psychological services can be tailored to provide appropriate support to all those affected by Parkinson's.

Matched care models of psychological support for people with Parkinson's

The psychological needs of pwP are broad and complex, and they fluctuate across the course of the condition. Therefore, a matched care model, in which the psychological services provided are matched to the pwP's individual characteristics and current needs, is recommended to provide rapid, clinically effective and cost-efficient care. This model differs from 'stepped care', in which all pwP start with the least specialist service, and if they do not respond sufficiently, are 'stepped up' to more specialist services.

The matched care model proposed in Figure 1 below provides an overview of the ways of meeting psychological and neuropsychological needs of pwP. The feature of this model is that all these services are required and interdependent on one another.

FIGURE 1: MATCHED CARE MODEL FOR PWP



Primary care mental health services (NHS Talking Therapies) typically have a long-term conditions workstream, in which therapists with expertise in long-term conditions provide psychological therapy for people with mild-to-moderate anxiety and depression, which can include pwP. Often, therapists will be experienced in supporting people who have had life-changing health conditions, including consideration of change in identity and social roles. However, intervention is typically limited in time or number of sessions. Although therapists will have core training in a psychological therapy such as cognitive behavioural therapy (CBT) and additional training in long-term conditions generally, there is limited additional training on the impact of neurological conditions, and Parkinson's specifically. This can lead to some challenges in the interplay of cognitive, emotional and systemic factors for pwP. Sessions may need to be adapted to help manage fatigue and cognitive changes, and this may be difficult to facilitate within some of the structures of NHS Talking Therapies. For example, homework is frequently a strong feature of CBT work and requires higher-level cognitive planning. Similarly, there can be structural barriers;

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outcome measurement is standardised and might not reflect or capture change for pwP. Likewise, there may be requirement for joint working directly with the specialist nursing and medical teams, which can be difficult to facilitate. As such, NHS Talking Therapies are most suitable for those who are at an early stage of PD, with minimal cognitive or neuropsychiatric features, who are motivated or supported by family members to engage with the therapeutic work.

In order to provide this within a matched care model, the staff within NHS Talking Therapies should have access to additional training in understanding the physical, cognitive and emotional impact of living with Parkinson's. In addition, it is important to embed the learning associated with this training. Other components of the matched care model, such as local neuropsychology services, would need to provide both the training and the necessary ongoing support and supervision to embed it.

Medics, specialist nurses and allied health professionals who work routinely with pwP also have a fundamental role in supporting neuropsychological needs. These clinicians have detailed knowledge of PD and its range of symptoms and medication. They are also typically involved with pwP over a longer period, which enables connection with individuals and their families on a different level. This facilitates an understanding of the change for the individual and their sociocultural context, and the meaning of PD for them and their family. These clinicians can be in a strong position to understand the cause and nature of the distress associated with Parkinson's, and the likely response to different therapeutic interventions. Some clinicians can also have additional training in therapeutic methods, such as motivational interviewing or CBT. In this setting, there is the possibility of integrating therapeutic approaches into medical, nursing and functional therapy, which has benefits of building on existing relationships and extensive knowledge of PD. Due to their regular contact with pwP and their families, PD specialist clinicians are also in a good position to offer routine screening for neuropsychological aspects of Parkinson's, such as cognition and psychological distress. To deliver this work safely and effectively, PD specialist clinicians need access to suitable training, support and supervision.

Secondary mental health services and ***memory services*** are frequently involved in supporting pwP. This may include dementia assessments from memory services. It is important that memory services have strong links with the specialist PD services to address specific questions about optimal delivery of services. For example, when should a pwP be referred to memory assessment services? When should cognition be monitored? When should a diagnosis be offered by the PD specialist services rather than requiring a separate referral?

When mental health difficulties and levels of distress increase, then further input is required from mental health teams. This may include support around medication management, management of risk and admission avoidance or, when required, admission to inpatient mental health facilities. As with the memory service, strong links between services is essential to support pwP effectively. Within both these service lines, as well as within medical or health psychology settings, psychotherapeutic input may be requested from practitioner psychologists/clinical neuropsychologists.

The importance of strong pathways between services is essential to avoid unnecessary barriers in accessing services, and to ensure staff have the training and ongoing supervision and support from those with expertise in PD. As with NHS Talking Therapies, it is important to note that pwP may need adaptation of the provision to meet their needs, such as adaptation of session lengths and consideration of motivation and engagement.

Specialist neuropsychology services can provide work directly with individuals to aid in diagnosis, and with specialist intervention or systemic family support. These services combine knowledge

of mental health presentations and psychotherapeutic techniques, with expertise and training in neurological conditions, including Parkinson's. They also have the benefit of typically being able to work closely and jointly with the medical teams. However, these services are typically less well-resourced compared to the other components of the matched care model. As a result, the amount of direct work is likely to be guided by the referral criteria of the service, and levels of consultation and indirect work need to be included within job plans. This role includes provision of training and supervision required by other aspects of the matched care model and supporting the members of the multidisciplinary team (MDT). Although neuropsychology training typically covers Parkinson's, it may be beneficial for those working in this area to seek additional training and supervision to further develop their knowledge and skills.

Areas for neuropsychological and psychological focus

Psychological assessment and intervention may be particularly useful in a range of areas impacted by Parkinson's, and these areas are outlined below. For each area, an overview of the impact of Parkinson's is presented, alongside suggestions to support assessment, formulation and intervention. Within each section, consideration is also given to diversity and inclusion, as well as the needs of the individual's family members and support networks.

COGNITIVE IMPACT

In contrast to Parkinson's own initial assertion in 1817 of 'the senses and intellects being uninjured' (Parkinson, 2002), cross-sectional and longitudinal studies have now consistently shown that cognition is affected in many pwP. Cognitive impairment in pwP has significant impact, diminishing quality of life, intensifying distress for family and carers, and hastening mortality. It also has marked financial implications for the care system: people with Parkinson's and dementia are over three times more expensive than those without (Vossius et al., 2010). Treatment with cholinesterase inhibitors offers symptomatic benefits, but there remains no disease-modifying treatment, and cognition continues to deteriorate.

Currently, neuropsychological assessment is used for supporting differential diagnosis, grading cognitive impairment, determining suitability for advanced therapies, and evaluating impact of medical interventions, as follows:

SUPPORTING DIFFERENTIAL DIAGNOSIS

Motor symptoms of Parkinson's disease can be heterogenous, with significant variation in phenotype between individuals. Variations in phenotypes for pwP may be related to genetic factors that determine disease progression, as well as age, gender and ethnicity. As a diagnosis of Parkinson's is made based upon clinical features, and confirmation is only possible post-mortem, the identification of relevant red flags is critical to accurate diagnosis overall (Bhidayasiri et al., 2019). Identification of cognitive red flags may help refine diagnosis to that of one of the atypical parkinsonian syndromes, which have different treatment options, disease courses and survival rates from those of Parkinson's.

Recently, great effort has been invested in trying to diagnose Parkinson's early, so as to enable intervention before neuronal loss is advanced and slow disease progression. Parkinson's neuropathology starts up to a decade before motor symptoms emerge, with up to 50% of dopaminergic neurons lost by the point of diagnosis (Greffard et al., 2006; Rees et al., 2018). During this pre-motor state, a host of non-motor features may emerge (O'Sullivan et al., 2008; Schrag et al., 2015), including subjective cognitive decline (O'Sullivan et al., 2008) and objectively lower cognitive performance (Bougea et al., 2019). As a result, the International Parkinson and Movement Disorder Society (MDS) research criteria for prodromal Parkinson's now include 'global cognitive deficit' (Heinzel et al., 2019). Although a diagnosis of 'prodromal Parkinson's' is still met with some concern about false positive rates (Marsili et al., 2018), it is increasingly likely that neuropsychological assessment will be used in attempts to support diagnosis of both manifest and prodromal disease (Getz & Levin, 2017).

Comprehensive neuropsychological assessment has revealed a characteristic pattern of cognitive decline, starting with initial changes in fronto-subcortical cognitive functions of processing speed,

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attention and executive functions. These cognitive changes insidiously evolve to encompass cortically mediated functions of language, memory and visuospatial processing (Moustafa & Poletti, 2013). The progression is thought to reflect diffuse cortical Lewy-body deposition and widespread cortical grey-matter atrophy (Hwang et al., 2013). However, it is important to note that non-motor symptoms for pwP have largely focused on white western populations, and research has not typically included ethnically diverse populations of pwP. In a recent study considering prediagnostic presentation in ethnically diverse and deprived populations, it was noted that memory difficulties were particularly prominent and indicated early cognitive involvement in these populations (Simonet et al., 2022).

This characteristic cognitive profile can be contrasted with that of atypical parkinsonian syndromes. Multiple System Atrophy (MSA), Progressive Supranuclear Palsy (PSP) and Corticobasal Syndrome (CBS) all mimic Parkinson's motor symptoms, but they have different cognitive profiles. Similar to Parkinson's, MSA is also an α-synucleinopathy. It manifests with parkinsonism, autonomic symptoms and ataxia in any combination (Stanković et al., 2019), and tends to progress more rapidly than Parkinson's, with a median survival time of 8.5 years. It is much rarer, and its cognitive profile less well researched. Although many studies have suggested that cognition is relatively preserved (Greenland & Barker, 2018), mild cognitive impairment has been identified in up to 30% of people with MSA, mostly characterised by executive dysfunction (Stanković et al., 2014, 2019), with supportive evidence of frontotemporal degeneration noted upon neuroimaging (Paviour et al., 2006). Of the limited number of reports, very few have described the involvement of cortically mediated cognitive functions (see Gerstenecker, 2017; Stanković et al., 2014 for reviews).

In contrast to Parkinson's and MSA, PSP and CBS almost always feature early cognitive involvement (Burrell et al., 2014). PSP is a tauopathy; tau protein aggregates abnormally in the subcortex and midbrain, leading to rapid atrophy. It has a poorer prognosis, with a median survival time of only 5.6 years. Classically characterised by parkinsonism with early postural instability and falls and vertical supranuclear gaze palsy, cognitive impairment is also a striking feature and supportive of diagnosis. The cognitive profile is mostly one of marked frontal executive dysfunction and slowed speed of processing (Rittman et al., 2016), but there can also be significant language impairments affecting speech production, with the most recent diagnostic guidelines identifying a PSP-speech and language phenotype (Höglinger et al., 2017).

CBS is a clinical syndrome characterised by parkinsonism, progressive asymmetrical limb apraxia, dystonia and cognitive impairment. Previously thought to be underpinned by tauopathy, post-mortem evaluation has in fact revealed heterogeneous neuropathological features, including that of PSP, Alzheimer's disease and TAR DNA-binding protein 43 (Ling et al., 2010). Median survival time is 7.9 years, but can range considerably, and is shorter in those with greater parkinsonism and/or frontal lobe involvement (Wenning et al., 1998). Its clinical manifestation is similarly diverse, overlapping with PSP and frontotemporal dementia clinical phenotypes (Kertesz et al., 2005), with differential involvement of frontal executive functions and cortically mediated cognitive functions, including language and visuospatial abilities (Greenland & Barker, 2018).

In both PSP and CBS, language difficulties may manifest as non-fluent progressive aphasia or apraxia of speech (Peterson et al., 2021). In both, there can be dramatically reduced verbal fluency (Foley et al., 2018, Foley et al., 2021; Peterson et al., 2021; Rittman et al., 2013). In CBS, there can also be distinctive early posterior cortical involvement, including impairments in visual perception and spatial processing, and constructional and/or limb apraxia, alongside alien limb phenomena (Burrell et al., 2014). In both PSP and CBS, the cognitive changes may

be accompanied by marked neuropsychiatric changes, including impulsivity, apathy and loss of empathy (Burrell et al., 2014; Southi et al., 2019).

Thus, diagnosis of Parkinson's can be supported by identifying the presence of the characteristic cognitive profile, and the absence of red flags of early involvement of cortically mediated cognitive functions, particularly language, praxis and visuospatial abilities, and/or prominent neuropsychiatric changes.

GRADING THE COGNITIVE IMPACT OF PARKINSON'S

People with Parkinson's can experience a wide spectrum of cognitive impairment, ranging from early subjective cognitive decline and mild cognitive impairment (MCI) to Parkinson's dementia (PDD).

Subjective cognitive decline is defined as self-perceived deterioration in cognitive ability in the absence of measurable change upon standardised neuropsychological assessment. Subjective cognitive decline is a new concept of growing interest in pwP, particularly for clinical trials of disease-modifying therapies (e.g. Oedekoven et al., 2022). Although it remains poorly understood, it is thought to confer a greater risk for future conversion to MCI/PDD (Erro et al., 2014).

Over the past decade, MCI has gained recognition as a construct and as a potential transitional state between normal cognition and dementia in both Alzheimer's and Parkinson's diseases (Goldman et al. 2018). Cross-sectional and longitudinal studies have revealed high rates of MCI and conversion to dementia in pwP, with up to 42.6% demonstrating MCI at baseline (Domellöf et al., 2015) and 60% converting to dementia within five years (Pedersen et al., 2017). There appear to be particular risks of cognitive impairment and dementia in pwP from Black and Hispanic communities (Willis et al., 2012). This may be related to genetic variations in PD, comorbid vascular changes or other comorbidities; however, it is important not to overlook the large-scale health inequalities that impact access to prompt diagnosis and appropriate medication options (Ben-Joseph et al., 2020).

MCI in pwP has been defined as a gradual decline in cognitive ability that is not severe enough to affect daily functioning (Litvan et al., 2012). Criteria include the presence of cognitive change, detected using either brief cognitive screening tools validated for use in pwP or comprehensive neuropsychological assessment, as indicated by performance approximately one to two standard deviations below appropriate norms, significant decline upon serial testing, and/or significant decline from estimated premorbid levels (Litvan et al., 2012). Cognitive change from premorbid level of function should be present on at least two neuropsychological tests across cognitive domains of attention and working memory, executive function, language, memory and visuospatial function.

Notably, these criteria permit considerable variation in diagnostic methods, which has yielded significant differences in prevalence rates of MCI subtypes (see Saredakis et al., 2019, and Wallace et al., 2022, for meta-analyses). When the MDS reviewed eight MCI studies, they reported that single-domain MCI was more common than multiple-domain MCI, and non-amnestic MCI was more likely than amnestic MCI (Litvan et al., 2011). However, even within these selected studies, there was significant variation in the design, sample, criteria and methods used for defining MCI and dementia.

Currently, these guidelines for MCI assessment indicate separate assessment of 'attention and working memory' from that of 'executive functions'. This deviates from many theoretical constructs, which consider working memory to be one of the executive functions (e.g. Baddeley & Hitch, 1974). Furthermore, they recommend the use of the Stroop Colour Word Test to assess

'attention and working memory', whereas many would argue that the Stroop test is a measure of the executive function of inhibition, associated with left prefrontal cortical circuits (Cipolotti et al., 2016). The Clock Drawing Test is also recommended to assess executive function. However, this test is also a measure of visuospatial construction, associated with posterior parietal cortical networks (Ino et al., 2003). The guidelines also make no recommendations for the assessment of speed of processing, despite this being a characteristic impairment in pwP.

Dementia in Parkinson's is a longer-established concept. In 2007, the MDS sought to operationalise criteria for its diagnosis, and it was subsequently defined as the presence of a gradual decline in cognitive ability, sufficiently severe to affect daily functioning (Dubois et al., 2007; Emre et al., 2007): changes should be present in at least two cognitive domains and represent a decline from estimated premorbid levels. Similar to Parkinson's MCI criteria, the diagnosis can be made either using brief cognitive screening tools or comprehensive neuropsychological assessment, with functional impact estimated from caregiver interview or formal questionnaires.

Parkinson's dementia (PDD) is thought to exist on a continuum with dementia with Lewy bodies (DLB), separated only by time of onset. DLB is diagnosed when the cognitive impairment precedes or occurs within one year of the onset of motor symptoms, whereas Parkinson's dementia is diagnosed when the cognitive impairment occurs at least one year after motor symptoms.

In pwP, there is considerable variation in the rate of cognitive decline, with some converting quickly to dementia and others demonstrating little cognitive change over a 20-year period (Aarsland et al., 2021; Hely et al., 2008; Zhang et al., 2019). Thus, there have been many attempts to identify predictors of faster cognitive decline.

Data-driven analyses and group comparisons have shown rate of cognitive decline to be associated with specific factors, for example, non-tremor dominant motor symptoms of slowness and gait dysfunction increase the risk of cognitive involvement (Domellöf et al., 2011; Lewis et al. 2005; Poletti et al., 2012), as well as hallucinations and mental health problems (Reijnders et al., 2009). These experiences may reflect greater degeneration of grey matter (Paulus & Jellinger, 1991; Rosenberg-Katz et al., 2013). Genetic analyses have also identified risk factors of cognitive impairment. Glucocerebrosidase (GBA) and α-synuclein (SNCA) mutations both pose greater risk to cognition (Lythe et al., 2017; Ramezani et al., 2021). Genome-wide association studies have revealed the *APOE* ε4 allele to be associated with dementia in pwP, likely because of increased burden of amyloid-β and/or α-synuclein pathology (Tan et al., 2021).

There have also been attempts at identifying cognitive signatures of faster cognitive decline. One influential theory, termed the 'dual syndrome hypothesis' (Kehagia et al., 2010; Kehagia et al., 2012), suggested the presence of two distinct cognitive subtypes, as evidenced by the longitudinal CamPaIGN study (Foltynie et al., 2004; Williams-Gray et al., 2007, 2009). The first of these is characterised by frontal executive impairments and thought to be mediated by frontostriatal dopaminergic dysfunction. Neuronal loss in the substantia nigra depletes dopamine levels in the striatum (Kish et al., 1988), resulting in deficient functioning of dorsolateral frontostriatal loops, and causing attentional and executive dysfunction (Alexander et al., 1986). The second subtype is one of posterior cortical visuospatial impairment, reflecting cholinergic denervation, and the early involvement of parietal and occipital cortices (Pappatá et al., 2011; Shoji et al., 2014). This latter subtype is suggested to be a dementia prodrome, with difficulties in copying interlocking pentagons increasing the odds of converting to dementia within five years by 5.2 (Williams-Gray et al., 2007).

The presence of hallucinatory phenomena may also be informative. Frank visual hallucinations are often accompanied by global cognitive dysfunction (Fénelon et al., 2000), with lower scores on tests of attentional and executive function (Ramírez-Ruiz et al., 2006), memory (Ibarretxe-Bilbao et al., 2010; Shin et al., 2012) and visual processing (Gallagher et al., 2011; Katzen et al., 2010; Koerts et al., 2010). Recent evidence also suggests that the feeling of a presence in the absence of objectively identifiable stimuli may also be an indicator of greater cognitive involvement (Puntambekar & Foley, 2023; Reckner et al., 2020). It is likely that these hallucinatory phenomena and advanced cognitive impairments both reflect increasing disruption of the posterior cortex.

Thus, the risk of a faster progression to dementia in pwP is considerably heightened by the presence of non-tremor dominant motor symptoms, candidate risk genes, a constructional apraxia and/or hallucinatory phenomena.

DETERMINING SUITABILITY FOR ADVANCED THERAPIES

As Parkinson's progresses, increasing amounts of levodopa therapy are required to manage motor symptoms. However, over time, many pwP develop motor complications of motor fluctuations ('on' and 'off' time) and dyskinesia as a consequence of levodopa therapy. These motor complications can become severe and necessitate the involvement of advanced therapies for ongoing management.

Advanced therapies for complex Parkinson's include

- subcutaneous apomorphine continuous infusion;
- Levodopa/carbidopa intestinal gel; and
- deep brain stimulation (DBS).

They all require hospital admission for their initiation, considerable self-management and frequent follow-up. Cognitive impairment does not necessarily preclude the first two of these three therapies, so long as a suitable support network is identified. However, the presence of cognitive impairment is currently an exclusionary criterion for DBS.

In DBS, electrodes are stereotactically implanted into the deep structures of the brain to deliver chronic, high-frequency electrical stimulation to specific neuroanatomical targets (Lozano et al., 2019). These electrodes are connected to a pulse generator placed under the clavicle. Post-operative programming of this pulse generator fine-tunes stimulation parameters to achieve optimal motor effects. Targeting the subthalamic nucleus or globus pallidus can lead to marked reductions in motor symptoms of tremor, rigidity and slowness, as well as fewer motor fluctuations and less dyskinesia (Pollak et al., 2002), and result in improvements in activities of daily living and quality of life for up to five years after surgery (Krack et al., 2003). However, there are also significant risks, including negative cognitive consequences. These include

- post-operative confusion (Kleiner-Fisman et al., 2006), which is usually transient, occurring in around 15.6%;
- subtle difficulties affecting frontal and subcortical cognitive functions, most commonly verbal fluency (Combs et al., 2015; Foley et al., 2018; Wang et al., 2016); and
- permanent global cognitive decline in around 9.4% (Aybek et al., 2007).

Risk factors for greater cognitive decline after DBS include older age, large ventricles, longer disease duration, more medical comorbidities (Strapasson et al., 2019) and greater cognitive impairment at baseline (Lang & Widner, 2002; Smeding et al., 2011). Pre-operative cognitive

assessment is important to determine the presence and extent of any cognitive impairment, not only to inform risk assessment, but also to gauge the capacity to consent to such an invasive procedure and the ability to comply with post-surgical device management.

DBS may also have an impact upon mood. There are several reports of worsened depression and a greatly increased risk of suicide (Giannini et al., 2019). Deterioration in mood may be explained by a rapid lowering of dopaminergic medication after surgery, but this tends to be short lasting (Cartmill et al., 2021). Risk factors for acts of harm towards self and longer-lasting depression include pre-operative depression, psychiatric medication use, previous acts with suicidal intent and family psychiatric history, as well as greater executive dysfunction (Giannini et al., 2019). Thus, DBS may be considered to be a stressful life event, by which those that have lower levels of psychological resilience and lower inhibitory control and problem-solving abilities are more likely to feel overwhelmed.

Apart from lowering mood, DBS may also increase levels of apathy (Zoon et al., 2021). Increased apathy is associated with greater reductions on tests of verbal fluency (Foley et al., 2018) and may negate any improvements in quality of life. Although some have found apathy to be related to decreases in levodopa medication (Wang et al., 2016), others have found apathy to manifest regardless of changes in medication or disease progression (Zoon et al., 2021). Further research is required to improve our understanding of baseline predictors of post-DBS apathy in order to quantify risk and counsel surgery candidates accordingly.

EVALUATING IMPACT OF MEDICAL INTERVENTIONS

Neuropsychological assessment may be routinely requested for surveillance of any cognitive side effects following a medical intervention. For example, neuropsychological assessment is often routinely repeated 6 to 12 months after DBS (Okun et al., 2007). In these re-assessments, focus should be on detecting any significant change in frontal and subcortical cognitive functions, particularly verbal fluency, as well as mood and apathy.

Neuropsychological assessment may also be requested when cognitive decline has been reported in the context of a medical treatment known to have possible cognitive side effects. For example, anticholinergic medications, frequently used to treat symptoms of Parkinson's, are known to increase the risk of cognitive impairment, confusion and hallucinations (Cooper et al., 1992; Ehrt et al., 2010). Similarly, dopamine replacement therapies, particularly dopamine agonists, are associated with psychosis in pwP, particularly in early disease (Weintraub et al., 2006). Therefore, when cognitive impact is reported, neuropsychological assessment may be requested to quantify any objective cognitive decline and provide a baseline before withdrawal of any medication.

ASSESSMENT

The specific referral question will guide hypothesis-driven neuropsychological assessment and formulation and inform any psychological interventions. Depending on the referral question, assessment may necessitate comparison of current intellectual function to optimal premorbid estimates, assessment of fronto-subcortical functions, and assessment of cortically mediated cognitive functions.

TABLE 1: GUIDE FOR NEUROPSYCHOLOGICAL ASSESSMENT IN PWP

COGNITIVE DOMAIN	SUBDOMAIN	EXAMPLE TESTS
Intellectual function	Current level	• Wechsler Adult Intelligence Scales (WAIS-IV-UK) (Wechsler, 2010) • Ravens Progressive Matrices (Raven & Raven 2003)
	Premorbid level	• Test of Premorbid Function (Wechsler, 2011)
Fronto-subcortical functions	Processing speed	• WAIS-IV-UK Coding (Wechsler, 2010) • WAIS-IV-UK Symbol Search (Wechsler, 2010) • Oral Symbol Digit Modalities Test (Smith, 1973)
	Attention and executive functions	• DKEFS Colour-Word Interference (Delis et al., 2001) • Stroop Colour Word Test (Stroop, 1935) • DKEFS Verbal Fluency (Delis et al., 2001) • Hayling Sentence Completion Test (Burgess & Shallice, 1997)
Cortically mediated functions	Language	• DKEFS Verbal Fluency (Delis et al., 2001) • Confrontation Naming tasks such as Graded Naming Test (McKenna & Warrington, 1980)
	Visuospatial function	• Visual Object Space Perception (Silhouettes; Cube Analysis) (Warrington & James, 1991) • Interlocking Pentagons (Folstein et al., 1975).
	Memory	• List Recall & Recognition Test such as California Verbal Learning Test-3 (CVLT-3) (Delis et al., 2017)

Throughout assessment, careful interpretation will be needed for those with English as an additional language, as neuropsychological assessment is embedded within Western culture, both in terms of the generation of content of subtests as well as the normative data from which scores are derived.

In pwP, decline in intellectual function is noted first on non-verbal measures of fluid intelligence, such as the WAIS-IV-UK Performance subscale (Brown & Marsden, 1987). On focal tests, there is decline in processing speed, accompanied by increasingly slow, inattentive and errorful performance on tests of attention and executive function. Decline in language and visuospatial functions are indicators of dementia. On tests of memory, there is often evidence of poor encoding, with worse performance on visual tests of recognition, but no convincing evidence of accelerated forgetting.

For differential diagnosis, additional tests of language (single word and sentence comprehension, single word and sentence repetition) and posterior cortical function (buccofacial and limb praxis, reading, spelling, arithmetic) may be required.

For grading cognitive impairment, the MDS recommend the use of at least two measures of each of their five cognitive domains (attention and working memory, executive function, language, memory and visuospatial function), and identify impairment as performance one to two standard deviations below appropriate norms, significant decline on serial testing, or significant decline from estimated premorbid levels.

For determining suitability for advanced therapies, full recommendations have been made elsewhere (Foley et al., 2018). These include undertaking careful assessment of cognitive function and mental health, with a focus on current and previous experience of depression/anxiety and suicidality, and family history of mental ill-health.

Neuropsychological assessments may need to be tailored according to the motor symptoms. For those presenting with dysarthria or marked hypophonia, assessments that require speeded verbal responses may be omitted. For those presenting with marked tremor or severe dyskinesia, assessments that require speeded motor responses may be omitted. Testing may also be paused if there are significant motor fluctuations affecting ability to engage. Any relevant cultural or linguistic factors should also be considered, and it may be appropriate to replace some tests with suitable substitutions for specific populations.

NEUROPSYCHOLOGICAL AND PSYCHOLOGICAL INTERVENTION

Neuropsychological interventions for people with cognitive impairment in Parkinson's include

- psychoeducation;
- cognitive rehabilitation;
- psychotherapy; and
- supporting family members and carers.

The document 'Nobody really knows us' (Parkinson's UK, 2021) stated that over half of pwP do not know symptoms of Parkinson's dementia. Furthermore, many never receive a formal diagnosis, which means that many have difficulty accessing the right support for their needs. Psychoeducational interventions should share neuropsychological formulations with the person with Parkinson's and their family to help them understand the symptoms and causes, as well as management options. This will also allow pwP and families to identify sources of appropriate support and make informed decisions about their care. To help with such discussions, a multidisciplinary group of clinicians and researchers have developed a Parkinson's dementia toolkit for clinicians on how to detect and manage the condition [https://www.parkinsons.org.uk/professionals/resources/toolkit-detecting-and-managing-parkinsons-dementia], and a complementary version for pwP, co-developed with people living with Parkinson's [https://www.parkinsons.org.uk/information-and-support/thinking-and-memory-changes#thinking-booklet].

Cognitive rehabilitation for pwP may be separated into restorative and compensatory approaches. Restorative cognitive training approaches have had moderate success in pwP (Leung et al., 2015), using pencil-and-paper or computerised cognitive training, but good quality randomised controlled trials are lacking. It is likely that the benefits of these approaches depend largely on the personalisation of the training, its cognitive focus, the participant's level of cognitive reserve and motivation to engage (see Biundo et al., 2017). Compensatory approaches that focus on adopting external aids to overcome specific cognitive changes may also be useful (Spencer et al., 2020).

Again, personalisation of the training to the participant's own goals and their own sociocultural context is important for its success and any functional improvements (Hindle et al., 2018)

Few published studies have examined the use of psychotherapy in pwP and cognitive impairment, with most studies excluding participants with dementia. However, pwP are often referred for help adjusting to a diagnosis of PDD. Sharing and discussing the neuropsychological formulation with the person with Parkinson's and their family is an important prelude to helping them consider how best to make meaning out of their experiences and identity alongside the Parkinson's diagnosis. Supportive psychotherapy may be useful as a conceptual framework in which to provide empathic listening, contain distress and help instil hope (Junaid & Hegde, 2007).

It is also critical to consider the psychological needs of family members and support networks. Studies consistently demonstrate that distress for family members is higher and more varied when supporting people with Parkinson's dementias than other dementias due to the combination of cognitive factors, the neuropsychiatric features of hallucinations and delusions, and issues of apathy, agitation, anxiety and depression (Svendsboe et al., 2016). In addition, there are higher levels of frailty and physical dependence than in other dementias, necessitating greater levels of physical and personal care, pharmacotherapy and community services, and support for almost all activities of daily living. As a result, intimate and sexual relationships can be eroded (Vatter et al., 2018), and families often have to endure high levels of chronic stress, with associated substantial costs in terms of distress and physical health. Some studies have suggested that Parkinson's caregiver burden may be even greater in families from minoritised communities (Bayram et al., 2024).

To date, there are few evidence-based psychological therapies designed specifically for family members of those with Parkinson's cognitive impairment. STrAtegies for RelaTives (START) is an eight-week, manualised, individual psychological intervention designed for family members of people with dementia. This was adapted for families supporting people with PDD and trialled by 10 relatives, with preliminary evidence that participation was associated with a reduction in emotional distress and improvement in quality of life (Foley et al., 2020). A recent pilot study exploring the use of Cognitive Stimulation Therapy in people with PDD and their families found no statistically significant improvement in participants' cognition, but significant improvements in family members' quality of life, relationship satisfaction, care burden and stress (Leroi et al., 2019), highlighting the benefits of therapist contact with family members. Families may also benefit from being directed to local or national support groups, such as Rare Dementia Support's Lewy Body Dementia support group [https://www.raredementiasupport.org/lewy-body-dementia/], and from the specialist support offered by local or national dementia nurses.

PSYCHOSIS

In 1883, Victor Parant was the first to describe psychosis in pwP and attributed these to changes in the brain (see Fénelon et al., 2021). It is now known that pwP can experience a spectrum of hallucinatory and delusional phenomena, which can occur at any stage of the disease process (Ffytche et al., 2017) and are a core clinical feature of DLB (McKeith et al., 2017). These experiences can be distressing and significantly reduce quality of life (McKinlay et al., 2008a). They can also be particularly stressful for family members, increasing family distress (McKinlay et al., 2008b), and are risk factors for nursing home placement (Zahodne & Fernandez, 2008) and mortality (Forsaa et al., 2010).

In the last decade, there have been considerable advances in our understanding of the presentation and pathogenesis of psychosis in pwP, although notable gaps remain.

HALLUCINATIONS

Visual hallucinations are common in pwP, affecting around a quarter of people in cross-sectional studies across ethnic groups (Fénelon et al., 2000; Rana et al., 2012) and up to 75% over a 20-year period (Diederich et al., 2009). Visual phenomena range from occasional illusory experiences, to felt presence and/or passage, to frank and intrusive visual hallucinations. Although initially occurring in an otherwise clear sensorium, visual hallucinations can gradually garner conviction over time (Ffytche & Aarsland, 2017).

Of the illusory experiences, the most common are kinetopsia, in which a stationary object is perceived as moving, and object misidentification, in which an object is misperceived for another (Nishio et al., 2018). Felt presence and passage are both extracampine phenomena, occurring just outside one's visual field. Felt presence is the sensation that somebody is nearby in the absence of any objectively identifiable stimuli. Most describe the presence as that of an unfamiliar human with neutral valence (Fénelon et al., 2011; Reckner et al., 2020). Those with reduced insight may be more likely to ascribe an identity to the presence, such as that of a dead relative (Reckner et al., 2020). Felt passage is the fleeting image or sensation of something moving. These images are usually reported as shadows or small animals (Pagonabarraga et al., 2016). The phenomenology of felt passage is less well studied than other visual phenomena in pwP.

Frank visual hallucinations are usually brief glimpses of humans and/or animals, neutral in valence. Usually occurring in evening or low light, or periods of low engagement, the images may become stereotyped and repetitive in nature. Over time, they may come to acquire additional sensory attributes, such as sounds, or become distressing (Diederich et al., 2009).

Hallucinations in other sensory modalities have also been described, although less commonly, and usually alongside visual phenomena (Weil & Reeves, 2020). For example, auditory hallucinations in pwP are usually muffled or distant neutral sounds, such as a bell, and only rarely voices (Fénelon et al., 2000; Weil & Reeves, 2020). Olfactory hallucinations are usually pungent odours, with occasional gustatory components (Kulick et al., 2018). Tactile hallucinations are usually sensations of contact with a small insect or animal (Diederich et al., 2009; Kulick et al., 2018).

Minor phenomena of visual illusions, presence or passage can occur in the early stages, even before diagnosis (Pagonabarraga et al., 2016), whereas frank visual hallucinations typically occur later, in more advanced disease (Weil & Reeves, 2020). Cross-sectional and longitudinal studies suggest that minor phenomena are forerunners for frank visual hallucinations (Lenka et al., 2019; Pagonabarraga et al., 2016; Wood et al., 2015), but additional pathology may be required for the manifestation of visual hallucinations (Doé de Maindreville et al., 2005; Ffytche et al., 2017; Marques et al., 2020). Should hallucinatory phenomena become distressing, specialist assessment and intervention is required (Weil & Reeves, 2020).

Hallucinations were rarely reported before the advent of levodopa treatment (Zhang et al., 2023) and initially these were dismissed as side effects of hyperdopaminergia. However, hallucinations can be divorced from dopaminergic treatments: they are not necessarily associated with a higher dosage of dopaminergic treatment (Fénelon et al., 2000), they can occur in the drug-naïve (Biousse et al., 2004; Pagonabarraga et al., 2016), they can occur after introduction of other drug treatments (Perry & Perry, 1995), and they rarely occur when dopaminergic treatments are used for other indications. Therefore, other mechanisms must also be involved.

A range of competing theories have now been offered to explain the pathogenesis of visual hallucinations in pwP. Common to many of these is the assumption that basic visual input is degraded and normal visual processing compromised (Barnes et al., 2003; Collerton et

al., 2005; Shine et al., 2011). Ocular pathology without neurological disorder can induce visual hallucinations, known as Charles Bonnet syndrome. These hallucinations have a similar phenomenology to that of Parkinson's disease (Barnes & David, 2001). Ocular pathology is often found in pwP, with evidence of greater dopamine-related retinal thinning (Archibald et al., 2009), more frequent reports of oculo-visual abnormalities such as dry eyes or double vision (Archibald et al., 2011), and greater difficulty on tests of basic visual functioning, including visual acuity, contrast sensitivity, colour perception and motion perception (Armstrong, 2011). Indeed, poor colour vision four-fold increases the risk of visual hallucinations for pwP (Diederich et al., 1998). Therefore, it has been suggested that in the absence of normal visual input, hallucinations may manifest as a sort of visual perseveration (Kinsbourne & Warrington, 1963) or cortical release phenomena (Cogan, 1973). However, ocular pathology does not always cause visual hallucinations, and visual hallucinations can occur in the absence of ocular pathology.

In addition to degraded basic visual input, visual hallucinations for pwP are thought to be contingent upon certain cognitive factors. Certainly, frank visual hallucinations are usually accompanied by cognitive impairment and are supportive for a diagnosis of PDD. Although minor hallucinatory phenomena were traditionally thought to be benign and insignificant, recent work has shown that these too are associated with cognitive impairments (Lenka et al., 2019; Puntambekar & Foley, 2023; Reckner et al., 2020).

Extant theories differ in their characterisation of the specific cognitive profiles involved in the emergence of visual hallucinations. Barnes and colleagues (Barnes, 2015; Barnes & Boubert, 2011; Barnes et al., 2003) argued for the role of impaired memory function. They suggest that, as source memory declines, there is increasing difficulty distinguishing between real and imaginary events, as well as a bias towards the erroneous attribution of internally generated events to external sources (Barnes et al., 2003). Several studies have found that visual hallucinations for pwP are associated with reduced performance on tests of verbal and visual memory (Ibarretxe-Bilbao et al., 2010; Ramírez-Ruiz, Junqué et al., 2007; Shin et al., 2012), a higher density of Lewy bodies in the parahippocampus (Harding et al., 2002) and greater hippocampal pathology upon fMRI (Yao et al., 2016).

Others have emphasised the role of impaired higher-order processing of perceptual data, particularly perceptual and spatial visual cognition (Collerton et al., 2005; Diederich, Goetz et al., 2005). On tests of perception, visual hallucinations in pwP are associated with relative impairments in object (Barnes et al., 2003), shape (Mosimann et al., 2004) and face discrimination (Ibarretxe-Bilbao et al., 2010; Ramírez-Ruiz, Junqué et al., 2007). On tests of spatial processing, there is greater difficulty estimating spatial relations (Cagnin et al., 2013; Davidsdottir et al., 2005), copying block designs (Hamilton et al., 2012), understanding overlapping figures (Mori et al., 2000), and perceiving motion (Mosimann, 2004). Accordingly, there is evidence of hypometabolism and atrophy in both ventral (Ramírez-Ruiz, Martí, et al., 2007; Williams & Lees, 2005) and dorsal visual processing streams (Boecker et al., 2007; Goldman et al., 2014; Marques et al., 2020; Pagonabarraga et al., 2014), alongside reductions in total brain volume. This is associated with disruptions to multiple neurotransmitter networks, including the serotoninergic network, which is thought to be involved in the early sensory processing and responses to visual inputs (Jacob & Nienborg, 2018).

Others still have emphasised that it is the combination of perceptual and attentional dysfunction that increases the risk of visual hallucinations (Collerton et al., 2005; Shine et al., 2011). For pwP, hallucinations are associated with increased daytime somnolence and REM sleep behavioural disorder (Fénelon et al., 2000; Marinus et al., 2018), suggesting that sleep regulation and basic arousal are involved. Furthermore, neuropsychological assessment reveals reduced

performance on tests of speed, attention and executive function, particularly on the Stroop test (Boubert & Barnes, 2015; Sanchez-Castaneda et al., 2010; Shine et al., 2011). This profile may be explained by dysfunction across attentional control networks. Visual hallucinations are related to difficulties recruiting dorsal and ventral attentional networks, resulting in increased engagement of the default mode network and overreliance upon endogenous attentional networks for perception (Shine, Keogh, et al., 2015; Yao et al., 2015). Thus, in this theory, impoverished bottom-up perceptual data arising from changes in basic and cortical visual processing combine with faulty top-down attentional control, to result in greater emphasis upon prior expectations and encouragement of false perceptual inferences (O'Callaghan et al., 2017; Zarkali et al., 2019). This process may be mediated in particular by the loss of cholinergic neurons and choline acetyltransferase in the cerebral cortex. Cholinergic insufficiency is thought to reduce signal-to-noise ratio in sensory information processing, resulting in poorer discriminatory abilities (Minces et al. 2017; Thiele, 2013).

In sum, our understanding of how and why hallucinatory phenomena occur for pwP has significantly evolved over recent years. No longer considered to be a mere side effect of dopaminergic treatment, current conceptualisations view hallucinations to reflect not only disruption in basic visual input, but also cognitive function. Specific theories diverge in their characterisation of the specific cognitive function critical for the manifestation of hallucinatory phenomena. There is greater understanding of how hallucinatory phenomena may be related to a raft of neurotransmitters, including dopaminergic, serotonergic and cholinergic networks. However, important questions remain. Firstly, it remains unknown if diverse hallucinatory phenomena share a single aetiology or rather a range of different mechanisms. Secondly, it remains unknown what informs the phenomenology of visual hallucinations: why are these sometimes experienced as benign, and other times threatening?

DELUSIONS

Delusions are considerably rarer than hallucinations, affecting only 1 in 100 pwP (Holroyd et al., 2001). However, when present, they are associated with high levels of distress and anxiety, and can result in violent and aggressive behaviour, usually towards the partner (Cipriani et al., 2012; Kingham & Gordon, 2004; Leong et al., 1994).

Delusions in pwP may present on their own or in tandem with hallucinations. When presenting alone, they tend to occur in those who are younger, with earlier-onset Parkinson's, and in those who demonstrate impulsive control behaviours (Warren et al., 2018). The delusions tend to be paranoid in nature, with themes of persecution, and delusional jealousy or 'Othello syndrome' (Warren et al., 2018). Misidentification syndromes, such as Capgras or Cotard, are less common (Kyrtsos et al., 2015).

In comparison to hallucinations, even less is known about the mechanisms associated with the development and maintenance of delusional beliefs. Usually, delusions in pwP are considered as a side effect of dopaminergic therapy (El Otmani et al., 2021). Indeed, delusions often commence after introduction of dopamine therapy (McNamara & Durso, 1991) and remit after its reduction. Moreover, dopamine therapy is known to have side effects, thought to be because of the uneven pattern of dopaminergic loss in the striatum and the relative 'overdosing' of intact ventral structures (Cools et al., 2001). As the ventral striatum is involved in reward and reinforcement learning, excessive dopaminergic stimulation may result in environmental stimuli, and particularly reward-related environmental stimuli, accruing anomalous salience and demanding greater attention.

It has been suggested that additional disruptions in cognitive functioning are required for delusions to develop (Poletti & Bonuccelli, 2013). Delusions in pwP are often thought to be by-products of increasing confusion (Fénelon & Alves, 2010). However, several studies have shown that they can also occur in the absence of dementia (Foley et al., 2017) and some have reported no correlation with cognitive performance (Factor et al., 2014). One case-controlled study found that pwP with delusional jealousy demonstrated difficulties on tests of response suppression, with high error rates on the Stroop and/or the Hayling tests (Foley et al., 2017). Further analysis revealed that the response suppression difficulties chiefly reflected limited strategy implementation.

The content of the delusion may also be revealing. Qualitative research has illustrated that, despite the considerable distress and confusion with which delusions are experienced, the content of the beliefs is often felt to be personally meaningful (Todd et al., 2010). For example, those who develop delusional jealousy may do so on a background of previous experience of betrayal and current relationship strain (Foley et al., 2017, 2023). Previous life events and current circumstances appear to shape the content of the delusion.

Thus, although dopamine therapy may provide the basic substrate for delusions, it is when this combines with previous life events and current circumstances that delusional beliefs are allowed to develop. Limitations in strategy implementation may prevent disengagement from habitual responses, enabling the delusion to persist.

ASSESSMENT

Assessment of psychosis in pwP is informative for understanding the lived experience and the impact upon the family/carer, as well as contributing to a neuropsychological formulation of cognitive function. People with Parkinson's may be referred for assessment of unusual beliefs or experiences, or these may be identified during neuropsychological assessment for other purposes. The National Institute of Health and Care Excellence guidelines for Parkinson's (NICE, 2017) recommend that such symptoms be discussed at every review appointment to determine if they are present and if they cause distress.

Assessment of experiences of psychosis within a psychological framework should involve detailing of the frequency of each experience reported. It is also important to gauge the level of distress caused and any associated beliefs or concerns. Table 2 lists the measures most commonly used for these purposes.

TABLE 2: ASSESSMENT OF PSYCHOSIS IN PWP

TEST	DESIGNED FOR PWP	SEPARATION OF TYPES OF EXPERIENCE	QUANTIFICATION OF FREQUENCY AND DISTRESS	SELF/ FAMILY RATING	LENGTH
Neuropsychiatric Inventory Questionnaire (Cummings et al., 1994)	✗	✗	✓	Family	15 minutes
Parkinson's Psychosis Questionnaire (Brandstaedter et al., 2005)	✓	✓	✓	Self- and family ratings	10 minutes
Scales for Outcomes in Parkinson's disease – Psychiatric Complications (Visser et al., 2007)	✓	✗	✓	Self- and family ratings	5–10 minutes
Psychosis and Hallucinations Questionnaire (Shine, Mills et al., 2015)	✓	✓	✓	Self	10 minutes

Most of the measures listed in Table 2 were designed for research studies. Most commonly used is the Neuropsychiatric Inventory Questionnaire, but this was designed to assess neuropsychiatric symptoms in dementia in general, and not specifically for Parkinson's-related psychosis. It can provide quantification of the severity and distress associated with psychosis, but this does not permit separation of different types of hallucinatory phenomena, nor assess delusional jealousy. Of the remaining measures, the Parkinson's Psychosis Questionnaire and the Psychosis and Hallucinations Questionnaire both assess the broad range of experiences encountered by pwP and their associated distress.

Identification of hallucinations and delusions will typically require full medical review. This will involve assessment of physical, ophthalmological and cognitive health, and review of all medications.

For designing clinical interventions, it will also be important to understand any specific antecedents and/or triggers, such as low light or periods of low engagement. Assessing such triggers with pwP and their families can build a better understanding of the cause of the experiences and opportunities for their amelioration. The sociocultural context of hallucinations and delusions for pwP should also be considered as an embedded part of the assessment, discussing with families the meaning made of the experiences and the sociocultural interpretations.

A more detailed neuropsychological assessment may also be beneficial to determine the presence of any global cognitive impairment, by comparing current intellectual function to optimal premorbid estimates. This involves assessment of fronto-subcortical functions and cortically

mediated cognitive functions, including memory, object perception, space processing, speed, attention and executive function.

INTERVENTION

Psychological interventions may be requested to help reduce any distress associated with these hallucinations and delusions and might include psychoeducation with the person with Parkinson's and their family members, as well as psychotherapy.

People with Parkinson's and their family members may feel frightened by these experiences. Providing neuropsychological formulations of why these experiences have occurred can support understanding and self-management. For hallucinations, any triggers that increase the risk should be identified. These may include situations in which there is low lighting, or times of the day in which there is low engagement in activities. These may be avoided by using more lighting and by scheduling engaging activities to bolster levels of arousal. Families can often feel shamed by delusional content, placing greater strain on already fractious relationships (Deutsch et al., 2021). Sharing neuropsychological formulations can help families make sense of why the delusion occurred and allow discussion about how to improve relationship functioning.

CBT may be useful to help challenge cognitive appraisal of experiences and help achieve less-distressing beliefs about them (Collerton & Dudley, 2004). This highlights several possible targets for intervention, including environmental changes, modification of attention templates, improving attention and perception, and modifying interpretation of experience or arousal or substituting actions.

EMOTIONAL IMPACT

The risk of clinically significant distress is elevated for pwP. Exact prevalence rates remain contentious and confounded by symptom overlap with underlying movement disorder, but several studies have suggested that at least 40% of pwP experience depression, anxiety and/or apathy during the course of the disease (Gallagher & Schrag, 2012), with some studies suggesting a significantly higher rate (e.g. Mindham, 1970). Emotional and psychological impact may be more disabling than motor symptoms and wield a greater influence upon quality of life (Müller et al., 2013). Higher levels of distress can affect motivation and engagement in rehabilitation or self-management programmes (e.g. Storch et al., 2013) and are associated with faster physical and cognitive decline, increased mortality and increased family distress (Hughes et al., 2004; Ravina et al., 2007; Starkstein et al., 1992; Uekermann et al., 2003).

Presentation of distress for pwP may vary in severity and is likely affected by a range of factors. There is some evidence that disruption to nigrostriatal pathways, and associated changes in dopaminergic function, contribute to psychological distress in pwP (Weintraub et al., 2005). Similarly, white matter changes within the cortical-limbic network have been associated with depression in pwP (Kostić et al., 2010). Indeed, anxiety and depression may be evident during the prodromal phase, preceding motor symptoms by up to five years (Ishihara & Brayne, 2006; Jacob et al., 2010; Leentjens et al., 2003). Specific psychological presentations are associated with different motor subtypes (Brown et al., 2011), with anxiety associated with younger age-of-onset and motor fluctuations, whereas depression is more related to axial motor symptoms (Burn et al., 2012). Treatment of motor symptoms may also reduce psychological distress (Leentjens, 2011). These findings suggest an important role of motor symptoms in the genesis and maintenance of psychological presentation, perhaps reflecting the underlying neurobiological mechanisms, as well as the way in which physical functioning has a significant role in levels of distress and participation.

Non-motor factors have been shown to be significantly more influential than disease variables in the development of distress associated with Parkinson's disease. Indeed, psychological and social factors have been shown to be three times as important in the prediction of psychological distress (Leentjens et al., 2013; MacCarthy & Brown, 1989). Here, we discuss the psychological basis to adjustment, anxiety, depression and apathy in pwP. This should be considered alongside the recent systematic review, which outlines the current evidence base and recommendations for psychological interventions for pwP (Simpson et al., 2021).

ADJUSTMENT & IDENTITY

A significant change in health status can represent a major life transition that challenges existing coping strategies and requires considerable adjustment (Stanton et al., 2007). Psychological adjustment to a chronic disabling illness, for which there is no cure, involves grieving for real and anticipated losses, and adapting to changes in function and roles across multiple life domains. Adjustment is an ongoing and dynamic process; fluctuating over time according to specific challenges faced and psychological resources available. Therefore, psychological response to Parkinson's is likely informed by early life experiences (Zimmermann et al., 2020) and sociocultural context, previous personal and family psychiatric history (Leentjens et al., 2002), as well as concurrent non-Parkinson's stressors (Blundell et al., 2023) and access to social and practical support (Garlovsky et al., 2016; MacCarthy & Brown, 1989).

Symptoms are constantly evolving for pwP, resulting in increasing levels of disability and dependence over time. Physical symptoms and cognitive changes may threaten personal identity as well as changes in occupational, social or functional roles (Blundell et al., 2023; Brown et al., 1988). This may be related to enacted or felt stigma (Eccles et al., 2023; Ma et al., 2016; Nehra et al., 2023; Schrag et al., 2001; Simpson et al., 2014), particularly within some minoritised communities (Mshana et al., 2011). Such changes may be more prominent in those who are younger (Brown et al., 1988) or experiencing greater physical or cognitive decline (Garlovsky et al., 2016).

Psychological adjustment for pwP is more important than disease severity for determining health-related quality of life (Suzukamo et al., 2006). However, psychological adjustment and disease severity are not unrelated; psychological adjustment is significantly affected by physical disability, as well as rate of health decline (MacCarthy & Brown, 1989). Disease progression can demand multiple readjustments, requiring different coping strategies for different transitions. Slower rate of progression may allow greater time to adjust to changes in function (Brown & Jahanshahi, 1995).

Psychological adjustment in pwP is also affected by self-esteem, coping style and availability of practical support (MacCarthy & Brown, 1989). Greater self-esteem may cushion against the effects of stress and threats to identity (Simpson et al., 2013). Positive coping strategies of problem solving (e.g. asking for practical advice) and distancing (e.g. turning attention to other activities) may reduce distress by allowing people to continue engaging in meaningful and enjoyable activities (MacCarthy & Brown, 1989).

Stigma associated with the symptoms of Parkinson's is experienced by up to 60% of pwP (Lin et al., 2022). The sociocultural contact of pwP also has a significant impact on experience of Parkinson's and stigma associated with the condition; for example, the intersectional nature of ethnicity and heritage alongside disability (de la Rosa & Scorza, 2024). However, the evidence base on the experience of Parkinson's in diverse and minoritised cultures is limited (Karacan et al., 2023).

In their review, de Ridder and colleagues (2008) discuss four strategies they suggest maximise psychological adjustment to chronic disease. These are remaining as active as possible, acknowledging and expressing negative emotions, engaging in self-management, and focusing on the potential positive outcomes of the illness. In pwP, psychological adjustment has been similarly related to maintaining a coherent sense of self, feeling in control, and being able to hold a positive mindset (Wieringa et al., 2022). Thus, interventions that focus on motivating and enabling self-management, at the same time as addressing the emotional impact of the disease, appear to be most promising for promoting successful psychological adjustment.

ANXIETY

Anxiety is the most common psychological presentation in pwP, affecting up to 55% across the course of the disease (Yamanishi et al., 2013), and one of the major determinants of quality of life (Balestrino & Martinez-Martin, 2017; Broen et al., 2016). Most commonly, it manifests as generalised or free-floating anxiety, characterised by an inability to relax, feelings of restlessness and excessive worry. Worries tend to be focused on motor symptoms and future disability (Dissanayaka et al., 2016). This type of anxiety is also associated with greater sleep disturbance, fatigue and autonomic dysfunction (Dissanayaka et al., 2016). There are also episodic anxiety disturbances unique to pwP, such as that associated with motor fluctuations (Pontone et al., 2009; Pontone & Mills, 2021). This can manifest as panic when dopaminergic medication is wearing off. Anxiety may exacerbate motor symptoms, particularly tremor, gait dysfunction and freezing (Ehgoetz Martens et al., 2014; Giladi & Hausdorff, 2006; Lauterbach et al., 2003). Worry about these symptoms, and their visibility, may contribute to social avoidance (Dissanayaka et al., 2016), alongside embarrassment, shame and stigma (Angulo et al., 2019; Eccles et al., 2023). Anxiety in pwP often co-presents with depression (Brown et al., 2011; Landau et al. 2016).

The nature and impact of anxiety in pwP is perhaps unsurprising, considering the condition's unpredictable nature. There is usually very little information provided on expected disease trajectories or future outcomes, little control over symptoms, and constantly changing treatment regimes. The person with Parkinson's is often rendered helpless in the face of a constantly changing condition, with little or no support. Indeed, it has been described as a 'constant struggle with unpredictability' (Haahr et al., 2011). Several studies have shown that the level of control a person believes they have over the condition is a particularly important determinant of psychological distress (Garlovsky et al., 2016), with low levels of control associated with greater levels of anxiety (Evans & Norman, 2009) and depression (Zampieri & de Souza, 2011). Perhaps related to this, emotion- or avoidant- rather than problem-focused styles of coping are associated with greater levels of psychological distress (Garlovsky et al., 2016; MacCarthy & Brown, 1989).

As such, coping strategies that enhance perceived control, through active problem solving and self-management, are associated with lower levels of psychological distress. Similarly, psychological interventions that have focused on enhancing tolerance of uncertainty, through relaxation practice, mindfulness and/or cognitive restructuring, have been found to be useful (see Zarotti et al., 2021), as has healthcare communication that promotes hope and a positive mindset (Hellqvist et al., 2018; Simpson et al., 2022).

DEPRESSION

Depression affects up to 50% of pwP (Aarsland et al., 2009; Cummings, 1992; Lemke, 2008). Depression affects the way that Parkinson's develops (Paumier et al., 2012), exacerbates disability (Pontone et al., 2016) and reduces quality of life (Aarsland & Karlsen, 1999). Core features may include a persistent feeling of sadness and reduced ability to enjoy previously enjoyable activities.

These may be accompanied by feelings of hopelessness or worthlessness, as well as rumination. Depression can be hard to disentangle from motor symptoms associated with Parkinson's; with both causing fatigue, pain and reluctance to engage in activities (Pontone & Mills, 2021). Feelings of inappropriate guilt, worthlessness or hopelessness, however, are signatures of depression (Pontone & Mills, 2021). People with Parkinson's are at a much higher risk of suicidal ideation (Kummer et al., 2009), with some evidence of increased risk of attempting or dying by suicide (Shepard et al., 2019).

Prevalence of depression in pwP has been found to have a bimodal distribution, with a small peak occurring early in the disease, when adjusting to the diagnosis, and a larger peak occurring later, when faced with high levels of disability (Schrag et al., 2001). This is particularly the case when the disability involves cognitive impairment (Schrag et al., 2001). Thus, depression in pwP is likely to be multi-factorial and related to changes in self-image and social roles, as well as loss of meaningful activity.

Several studies have revealed depression in pwP to be mediated by personality, coping style and illness beliefs, as well as access to social support (Garlovsky et al., 2016). These findings are important as they suggest that nonpharmacological treatment approaches may also be useful for managing psychological distress in PD.

APATHY

Apathy may be defined as a reduction in goal-directed activity that can manifest in behavioural, psychological and/or cognitive domains, and cannot be attributed to diminished level of consciousness, psychological distress and/or cognitive impairment. Rates of apathy in pwP vary widely with a prevalence of 29.1% (Mele et al., 2019).

Several attempts have been made to operationalise diagnostic criteria for apathy and its subcomponents and disambiguate it from depression or psychological distress. For example, Pagonabarraga and colleagues (2015) proposed a four-factor model of apathetic syndromes, which included (a) reward deficiency syndrome, affecting the ability to assign pleasure or value to a particular task, leading to diminished emotional interests and/or emotional blunting; (b) emotional distress; (c) executive dysfunction, affecting the ability to redirect attention, manipulate complex cognitive information or plan, leading to 'cognitive inertia'; and (d) disruption in auto-activation, leading to reductions in self-initiated behaviour and 'motor readiness'.

The pathological processes underlying apathy in pwP remain unclear but may be related to structural and functional disruption of the limbic system and its links to the prefrontal cortex (Muhammed & Husain, 2016; Pagonabarraga et al., 2015). Apathy rates increase after deep brain stimulation (Thobois et al., 2010), which may reflect a dopamine agonist withdrawal syndrome that can be reversed with dopaminergic medication. Post-operative apathy that does not respond to dopaminergic medication or psychiatric treatments may reflect a more diffuse cortical synucleinopathy (Braak et al., 2006). Apathy in pwP is associated with a poorer prognosis, with greater motor impairment, more severe executive dysfunction and faster conversion to dementia (Dujardin et al., 2009; Pagonabarraga et al., 2015). It also significantly increases family distress (Leroi et al., 2012).

ASSESSMENT

There is no definitive method for establishing the nature and intensity of distress that someone with Parkinson's may be experiencing. A clinical interview is used as a starting point to help develop a psychological formulation. This interview may wish to enquire about current emotional state and feelings of low mood and anxiety; journey to diagnosis; current Parkinson's motor and

non-motor symptoms, including subjective cognitive decline, psychosis and impulse control behaviours, as well as changes to sleep, appetite and experience of pain; early life experiences, previous mental health history and previous experience of psychological interventions; current living arrangements and levels of family and social support; and current engagement in physical and occupational or meaningful activities.

Assessment may be supported by rating scales to gauge severity of psychological distress. However, many of these were not developed for pwP and adjusted cut-off scores are often recommended. Those that have the best psychometric properties (see reviews by Dissanayaka et al., 2015; Leentjens et al., 2008; Schrag et al., 2007) can be found in Table 3.

TABLE 3: RATING SCALES TO ASSESS PSYCHOLOGICAL WELLBEING IN PWP

DOMAIN	RATING SCALE
Depression	Hamilton Depression Rating Scale (Hamilton, 1960)
	Montgomery-Åsberg Depression Rating Scale (Montgomery & Åsberg, 1979)
	Geriatric Depression Scale (Short Form) (Greenberg, 2007)
	Beck Depression Inventory (Beck et al., 1996)
	Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983)
Anxiety	Parkinson Anxiety Scale (Leentjens et al., 2014)
	Hamilton Anxiety Rating Scale (Hamilton, 1959)
	Geriatric Anxiety Scale (Segal et al., 2010)
Apathy	Apathy Evaluation Scale (Marin et al., 1991)
	Lille Apathy Rating Scale (Sockeel et al., 2006)
	Apathy Motivation Index (Ang et al., 2017)
	Neuropsychiatric Inventory (Cummings et al., 1994)
	Dimensional Apathy Scale (Radakovic & Abrahams, 2014)

When there is subjective cognitive decline and/or reports of apathy, it may be useful to undertake a comprehensive cognitive assessment to understand how cognitive function may contribute to the psychological presentation.

INTERVENTION

First-line treatment for distress for pwP is often antidepressant medication. However, a pooled analysis revealed that antidepressants have a statistically nonsignificant effect upon depression and anxiety for pwP (Troeung et al., 2013), and the evidence for their use remains weak (Starkstein & Brockman, 2017). In addition, there are concerns about adverse side effects (Ceravolo et al., 2000) and polypharmacy (Müller, 2002).

NICE guidelines for depression in adults with a chronic physical health problem (NICE, 2009) advise against reliance upon antidepressants, recommending instead that all with depression and/or anxiety be offered group-based psychological interventions. These should be delivered for a minimum of six to eight weeks and up to 18 weeks.

Randomised controlled trials have shown beneficial effects of CBT for anxiety in pwP (Moonen et al., 2021) and depression (see Zarotti et al., 2021 for a review), either delivered one-to-one (Dobkin et al., 2011) or in groups (Troeung et al., 2014). Indeed, many pwP prefer psychological interventions to yet more medication (Dobkin et al., 2013; Oehlberg et al., 2008). Evidence suggests that these interventions should focus on motivating and enabling self-management to enhance psychological adjustment and resilience for coping with future disease progression.

Leentjens and colleagues have provided free access to their 10-session cognitive behavioural intervention that is associated with significant reductions in anxiety, depression and apathy (Moonen et al., 2021). The therapist manual and workbook are available from their website (https://www.maastrichtuniversity.nl/research/mhens/research/projects/parkinsons-disease-research#CBT).

Delivery of any psychological intervention may need to negotiate certain Parkinson's-specific issues. These include the motor symptoms and any physical disability, which may limit ability to engage in therapy sessions and behavioural activation exercises. Furthermore, pwP advanced disease may have motor fluctuations, causing marked variation in motor and psychological distress. People with Parkinson's may be specific about the times of the day that they are 'on' and able to attend therapy. Any Parkinson's-related cognitive decline may also affect engagement, by limiting ability to sustain attention and encode new information during therapeutic sessions. In these cases, sessions may benefit from a slower pace, increased repetition and written summaries. Often pwP arrive for therapy with a family member, and it is important to discuss with the individual if they wish to involve the family members in the therapeutic sessions. Finally, it is important to consider the elevated risk of suicide and assess and monitor this accordingly.

IMPULSE CONTROL BEHAVIOUR

Impulse control disorder (ICD) is an 'inability to resist an impulse, drive or temptation to perform an act that is harmful to the person or others' (Prediger et al., 2012). ICDs in pwP include compulsive gambling, spending, sexual behaviour and eating (Weintraub et al., 2015). In addition, there can be other impulsive-compulsive behaviours (ICBs) of hobbyism and punding, the compulsive repetition of meaningless actions (O'Sullivan et al., 2007), and dopamine dysregulation syndrome, the compulsive desire for and overuse of dopamine medication (Weintraub et al., 2015). ICBs can have a significant impact upon an individual's life and family, disrupting financial security, relationships and social roles, as well as causing considerable shame, anxiety and low mood. Research has highlighted that rates of ICDs are consistently higher than in age-matched community samples (Weintraub & Potenza, 2006) with a prevalence of at least one type of ICD in 13.6% of pwP (Weintraub et al., 2010). However, it is likely that this prevalence is a significant underestimate.

ICDs are often linked to dopamine replacement treatments, most commonly D2 receptor-selective dopamine agonists (Ondo & Lai et al., 2008). These treatments are thought to lead to an 'overdose' of dopamine in the ventral striatum and connected limbic areas (Cools, 2006), areas important in reward-seeking actions. Therefore, it is perhaps unsurprising that ICDs occur with such treatment. These actions may be further mediated by impairment in executive functions of set-shifting, response inhibition and decision-making (Kelly et al., 2020; Martini et al., 2018), and reduced risk-reward processing (Weintraub et al., 2015).

However, not everyone who is treated with dopamine agonists will develop ICDs and not all those with ICDs are on dopamine agonists. It appears likely that there are also various psychological, social and cultural factors to consider in the development and maintenance of ICDs. These include

current distress; personal and family history of risky behaviours, such as gambling, excessive alcohol intake and/or cigarette smoking; personality variables of thrill-seeking or impulsivity; and current levels of psychological distress (Weintraub et al., 2015). It is often noted that ICDs are more common in males, those of younger age and those with specific personality factors (Leclerq & Corvol, 2024), but it is important to recognise that all ICDs can occur across the age range, in both men and women. Rates of ICDs in pwP vary between geographic regions, and the UK has some of the highest prevalence rates of ICD in pwP worldwide, highlighting the sociocultural and political influence on development and interpretation of ICDs (Parra-Diaz et al., 2021).

ASSESSMENT

ICDs are frequently underreported by pwP. This may be because of embarrassment and shame because they are not seen as linked to Parkinson's or because of concern about changes in medication that may result from disclosing ICDs. Clinicians can seek to normalise ICDs, reassuring pwP and their support networks.

It may also be useful to use formal scales to assess the presence and severity of any impulsive behaviours. There are several semi-structured screens available for this, as detailed in Table 4.

TABLE 4: RATING SCALES TO ASSESS ICDs IN PWP

RATING SCALE	CHARACTERISTICS
Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease – Rating Scale (Weintraub et al., 2012)	Questionnaire, to be completed by pwP or clinician
Ardouin Scale of Behaviour in Parkinson's Disease (Rieu et al., 2015)	Semi-structured interview, to be used by clinician familiar with Parkinson's
Parkinson's Impulse-Control Scale for the Severity Rating of Impulse-Control Behaviors in Parkinson's Disease (Okai et al., 2016)	Semi-structured interview, to be used by clinician familiar with Parkinson's

It will also be important to understand how the impulsivity sits within a wider cognitive profile. Thus, cognitive assessment should involve comprehensive neuropsychological assessment to determine overall deterioration in intellectual function; a broad assessment of fronto-subcortical functions of set-shifting, response inhibition and abstract reasoning; and assessment of cortically mediated functions to assess for evidence of advanced cognitive decline. On performance-based assessment, pwP with ICDs often take insufficient time to make decisions, demonstrating hasty responding.

Assessment should also include measures of depression, anxiety and apathy (see previous sections for suggested measures), recognising that the behaviours may represent both a response to and cause of psychological distress.

INTERVENTION

First-line treatment for ICDs is the reduction or removal of dopamine agonist medication, and/or rationalisation of all dopamine replacement therapies. However, this may result in reduced physical function, as well as inducing a dopamine-agonist withdrawal syndrome. This may prove intolerable or insufficient for reducing the problematic behaviours. Furthermore, once the ICD has abated, the pwP and family may be left devastated by the consequences. Thus, psychological interventions are often indicated.

Although very few studies have examined the effectiveness of psychological interventions for ICDs, these have shown that CBT can be useful (Okai et al., 2013), particularly in those who have less severe ICD, less disabling motor symptoms, lower levels of anxiety and higher social functioning (Okai et al., 2015). Therefore, NICE guidelines for Parkinson's disease (NICE, 2017) recommend offering specialist CBT targeted at ICDs, if modifying dopaminergic therapy is not effective. In addition, individual psychological therapy may be useful for helping the pwP to develop more positive coping strategies, and couple therapy for helping the dyad understand and reduce the psychological impact of the behaviours.

SLEEP DISORDERS

Changes in sleep are very common for pwP (Chaudhuri & Schapira, 2009), with sleep disorders affecting up to 60–90% of pwP (Schütz et al., 2022). Most commonly, these are insomnia (Chahine et al., 2017); daytime sleepiness with sleeping attacks; sleep-breathing disorders (Diederich, Vaillant et al., 2005); and REM sleep behaviour disorder (RBD; Berg et al., 2015). Indeed, RBD is thought of as the prodromal phase of Parkinson's, with 80% of people with idiopathic RBD later being diagnosed with Parkinson's (Berg et al., 2015). Even early in disease progression, pwP show reduction in overall sleep time and sleep efficiency, including reduction of slow-wave sleep (Joy et al., 2014).

In pwP, sleep quality may have an important impact upon physical, cognitive and psychological aspects (Hermann et al., 2020). When sleep breathing disorders are present, oxygen saturation and hypoxia may need to be considered in the assessment of cognition. It is important to note that sleep disturbance can further impact upon the family network, impacting social relationships.

This can include the motor symptoms of Parkinson's, such as night-time akinesia, dystonic pain and trouble turning over, and non-motor symptoms such as nocturia (Schütz et al., 2022). Parkinson's medication can also have specific effects upon sleep, such as dopamine agonists causing sleep attacks. In addition, pwP may experience degeneration of the sleep-regulatory pathways and to the circadian rhythm, including brainstem structures such as the raphe nuclei and locus coeruleus (Braak et al., 2003). This can interfere with neurotransmitter pathways (Schütz et al., 2022). The research indicates a bidirectional relationship between sleep quality and neurodegeneration, with reduction in sleep quality associated with reduced ability to clear neurotoxins (Hablitz & Nedergaard, 2021).

It is also important to consider the sociocultural context in which sleep disorders occur in pwP. Sleep is inherently impacted by the surrounding environment as well as internal processes, and sleep disorders may be influenced by socioeconomic factors and/or health inequalities (Valencia et al., 2023).

ASSESSMENT

It may be useful to undertake a comprehensive sleep history to help understand what may be causing the sleep disturbance, and how this may affect day-to-day functioning. This should include (Schütz et al., 2022)

- bed and waking times;
- bed-time routine;
- daytime naps;
- challenges in getting to sleep;
- challenges in staying asleep; and
- night-time awakenings and (if known) causes.

These assessments may be usefully supplemented by sleep diaries completed by the person themselves, with the possibility of family/carer report, where possible.

There are also several sleep-specific assessment tools for general use that may be useful. These are shown in Table 5.

TABLE 5: GUIDE FOR SLEEP ASSESSMENT IN PWP

AREA	MEASURE
General	Pittsburgh Sleep Quality Index (Högl et al., 2010)
	Epworth Sleepiness Scale (Martinez-Martin et al., 2008)
PD-Specific	Parkinson's Disease Sleep Scale (Trenkwalder et al., 2011)
	Scales for Outcomes in PD Sleep (Marinus et al., 2003).
Restless Leg Syndrome	RLS-diagnostic index (RLS-DI)
	International Restless Legs Symptoms Severity Scale (IRLSS) (Allen et al., 2003)
Insomnia	Insomnia Severity Index (Bastien et al., 2001)
REM Sleep Behaviour Disorder	REM Sleep Behavior Disorder Screening Questionnaire (RBDSQ) (Stiasny-Kolster et al., 2007)

In those with chronic and difficult-to-manage sleep disorder, actigraphy or video polysomnography at a sleep clinic may be indicated. These can provide detailed information about sleep integrity and quality, and access to specialised interventions.

Assessment around sleep should be multi-dimensional and should also include measures of mental health, distress, other physical health factors and hormonal status embedded within the sociocultural context of the individual and family.

INTERVENTION

Treatments for sleep disorders for pwP will be specific to the formulation, based on the type of sleep difficulty which is being experienced and the factors which are impacting on it (Schütz et al., 2022).

Medical Interventions: Medical colleagues may be involved in considering what medical management can support pwP, and this may involve adjusting dopamine treatment or other medications or treating other symptoms contributing to poor sleep, such as an overactive bladder. There is some evidence that pwP who have had DBS have some improvement in sleep quality, although there is lack of clear mechanisms: whether this relates to improvement in motor symptoms or whether this is a distinguishable outcome and is acting directly on the sleep-wake systems (Choi et al., 2019).

Psychological Interventions: Psychological interventions for sleep disorder may include psychoeducation about sleep hygiene (Seppi et al., 2019). CBT has been specifically adapted for insomnia (CBT-I), with some studies evidencing benefits in sleep efficiency and decreased nighttime waking (Humbert et al., 2017). This approach includes considering the thoughts, actions and physiological sensations associated with suboptimal sleep, paying particular attention to the thoughts that maintain suboptimal sleep. Fatigue management approaches may also be

helpful for pwP to support energy management and maintenance. However, it is worth noting that the evidence base is still limited in both nature and scope of interventions that have large-scale and high-quality supporting evidence, and there is further work to be done.

Formulation

None of the areas outlined in the preceding section occurs in isolation, and it is likely that, at any one time, an individual with Parkinson's will be experiencing a complex interplay of factors. As such, the role of practitioner psychologists/clinical neuropsychologists is frequently to bring this information together in a comprehensive and holistic biopsychosocial formulation.

History, from the clinical interview both with the individual and those who know them well, and from medical notes, should be integrated with performance and observations upon cognitive testing to provide a comprehensive analysis of neuropsychological function across the different cognitive domains (Savage, 2016). This will include consideration about how test performance is affected by linguistic, cultural and educational factors; any physical limitations, such as dysarthria, tremor, motor fluctuations or pain; and mood and engagement. Thus, neuropsychological formulation will conceptualise test performance using empirical and theoretical models of cognitive function (Wilson & Betteridge, 2019), and place this within biopsychosocial and systemic models to understanding the current needs of the individual and family (Evans, 2019). Neuropsychological formulation may then seek to conceptualise the current presentation within the context of predisposing, precipitating, perpetuating and protective factors. Relevant predisposing factors may include previous experience of adversity, personal and family psychological history, concepts of identity and illness, and coping style. Precipitating factors may involve current motor or non-motor symptoms, as well as other non-Parkinson's stressors. Perpetuating factors often involve cognitive changes and reduced engagement in or avoidance of social, occupational and/or physical activities. Relevant protective factors may include access to social and/or practical support or engagement in self-management regimes. This neuropsychological formulation can then be used to share assessment findings, answer the specific referral question, and inform intervention planning, where appropriate.

Whilst there are not specific models of formulation for pwP, there are examples that can be drawn from other populations of people with progressive neurological conditions, such as Huntington's Disease (Dale et al., 2022). The model outlined by Dale and colleagues considers how aspects of life story, values and sociocultural factors, alongside the motor, psychological and cognitive implications of the condition, influence how distress may manifest for the individual. This framework also includes the role of future anticipation, which is of key relevance for those diagnosed with a progressive condition. There are also biopsychosocial formulation models which can be drawn from the dementia literature, such as the biopsychosocial-ecological framework, which outlines ways in which progressive conditions can be viewed through a 'family-framed lens' to reflect on both the individual and relational impact (Podgorski et al., 2021).

Neuropsychological formulation within this context, then, provides the underpinning for the matched care model outlined at the beginning of this document. This formulation serves as a foundation for MDT and inter-agency working to create a shared understanding of the needs of an individual and their social support system. Models of formulation can then be used to inform wider system developments, such as screening programmes. Furthermore, training and supervision can be built on this dynamic integration of cognitive, psychological, physical and sociocultural factors.

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Service evaluation standards

Based on the matched care model, these are the suggested standards:

TABLE 6: PD SERVICE EVALUATION STANDARDS

1.	Access to specialist neuropsychology services for people with PD in the local area
2.	Neuropsychology provision integrated within the PD team
3.	Access to specialist neuropsychology supervision across the pathway
4.	Provision of a local pathway specific for people with PD in the local NHS Talking Therapies service, Memory Service and secondary care mental health services
5.	Access to Parkinson's-specific training and supervision for the local NHS Talking Therapies service, Memory Service and secondary care mental health services
6.	Access to training for PD-specialist staff, including neuropsychological implications and approaches to supporting people with PD
7.	Standardised screening for mood and cognition pathways, overseen by specialist neuropsychology services
8.	Adaptation of provision to address issues of health inequality and cultural and language diversity

These standards can be used for audit purposes, to consider gaps and comparisons across regional provision and to monitor progress around service improvements.

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Summary and conclusion

Parkinson's is a complex and multifaceted condition which affects individuals and their families in wide-reaching ways. Support needs vary across the course of the condition and, as such, a wide range of services will be required to support pwP. This involves both specialist and generalist services and close working relationships between organisations, including access to specialist training and supervision. There has been significant progress within the evidence base since the initial guidance was released, and these guidelines hope to support clinicians in the translation of this evidence into routine clinical practice.

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References

Aamodt, W.W., Willis, A.W. & Dahodwala, N. (2023). Racial and ethnic disparities in Parkinson's disease: A call to action. *Neurology: Clinical Practice, 13*, Article e200138. doi: 10.1212/CPJ.0000000000200138

Aarsland, D., Batzu, L., Halliday, G.M. et al. (2021). Parkinson disease-associated cognitive impairment. *Nature Reviews Disease Primers*, *7*(1), 1–21. doi: 10.1038/s41572-021-00280-3

Aarsland, D. & Karlsen, K. (1999). Neuropsychiatric aspects of Parkinson's disease. *Current Psychiatry Reports*, *1*, 61–68. doi: 10.1007/s11920-999-0011-3

Aarsland, D., Marsh, L. & Schrag, A. (2009). Neuropsychiatric symptoms in Parkinson's disease. *Movement Disorders*, *24*(15), 2175–2186. doi: 10.1002/mds.22589

Alexander, G.E., DeLong, M.R. & Strick, P.L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, *9*, 357–381. doi: 10.1146/annurev.ne.09.030186.002041

All Party Parliamentary Group for Parkinson's Disease (2009). *Please mind the gap: Parkinson's disease services today*. cdn.ps.emap.com/wp-content/uploads/sites/3/2009/07/APPG_Report_Please_Mind_the_Gap.pdf

All Party Parliamentary Group for Parkinson's Disease (2018). *Mental health matters too: improving mental health services for people with Parkinson's who experience anxiety and depression* (Issue: May). https://www.parkinsons.org.uk/sites/default/files/2018-05/APPG%20on%20Parkinson%27s%20mental%20health%20report%20-%20May%202018.pdf

Allen, R.P., Kushida, C.A., Atkinson, M.J. & RLS QoL Consortium (2003). Factor analysis of the International Restless Legs Syndrome Study Group's scale for restless legs severity. *Sleep Medicine, 4*(2), 133–135. doi: 10.1016/s1389-9457(02)00193-4

Ang, Y-S., Lockwood, P., Apps, M.A.J. et al. (2017). Distinct Subtypes of Apathy Revealed by the Apathy Motivation Index. *PLoS ONE, 12*(1), Article e0169938. doi: 10.1371/journal.pone.0169938

Angulo, J., Fleury, V., Péron, J.A. et al. (2019). Shame in Parkinson's disease: a review. *Journal of Parkinson's Disease*, *9*(3) 489–499. doi: 10.3233/JPD-181462

Archibald, N.K., Clarke, M.P., Mosimann, U.P. & Burn, D.J. (2009). The retina in Parkinson's disease. *Brain*, *132*(5), 1128–1145. doi: 10.1093/brain/awp068

Archibald, N.K., Clarke, M.P., Mosimann, U.P. & Burn, D.J. (2011). Visual symptoms in Parkinson's disease and Parkinson's disease dementia. *Movement Disorders*, *26*(13), 2387–2395. doi: 10.1002/mds.23891

Armstrong, R.A. (2011). Visual symptoms in Parkinson's disease. *Parkinson's Disease*, *2011*(1), 908306. doi: 10.4061/2011/908306

Aybek, S., Gronchi-Perrin, A., Berney, A. et al. (2007). Long-term cognitive profile and incidence of dementia after STN-DBS in Parkinson's disease. *Movement Disorders*, *22*(7), 974–981. doi: 10.1002/mds.21478

Baddeley, A.D. & Hitch, G.J. (1974). Working memory. In G.H. Bower (Ed.) *Psychology of Learning and Motivation, 8*, 47–89. New York: Academic Press. doi: 10.1016/s0079-7421(08)60452-1

Balestrino, R. & Martinez-Martin, P. (2017). Reprint of 'Neuropsychiatric symptoms, behavioural disorders, and quality of life in Parkinson's disease'. *Journal of the Neurological Sciences*, *374,* 3–8. doi: 10.1016/j.jns.2017.01.026.

Barnes, J. (2015). Neuropsychological approaches to understanding visual hallucinations. In D. Collerton, U. P. Mosimann & E. Perry (Eds.) *The Neuroscience of Visual Hallucinations* 193–215. Wiley Blackwell. doi: 10.1002/9781118892794.ch9

Barnes, J. & Boubert, L. (2011). Visual memory errors in Parkinson's disease patient with visual hallucinations. *International Journal of Neuroscience*, *121*(3), 159–164. doi: 10.3109/00207454.2010.539308

Barnes, J., Boubert, L., Harris, J. et al. (2003). Reality monitoring and visual hallucinations in Parkinson's disease. *Neuropsychologia*, *41*(5), 565–574. doi: 10.1016/s0028-3932(02)00182-3

Barnes, J. & David, A.S. (2001). Visual hallucinations in Parkinson's disease: a review and phenomenological survey. *Journal of Neurology, Neurosurgery & Psychiatry*, *70*(6), 727–733. doi: 10.1136/jnnp.70.6.727

Bastien, C.H., Vallieres, A. & Morin, C.M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine, 2*(4), 297–307. doi: 10.1016/s1389-9457(00)00065-4

Bayram, E., Liu, H., Luo, S. et al. (2024). Ethnoracial differences for caregiving burden in Parkinson's disease. *Parkinsonism & Related Disorders, 118,* 105927. doi: 10.1016/j.parkreldis.2023.105927

https://doi.org/10.53841/bpsrep.2025.rep189.8

Beck, A.T., Steer, R.A. & Brown, G. (1996). *Beck Depression Inventory–II (BDI-II).* APA PsycTests. doi: 10.1037/t00742-000

Ben-Joseph, A., Marshall, C.R., Lees, A.J. & Noyce, A.J. (2020). Ethnic variation in the manifestation of Parkinson's Disease: A narrative review. *Journal of Parkinson's Disease, 10*(1), 31–45. doi: 10.3233/JPD-191763

Berg, D., Postuma, R.B., Adler, C.H. et al. (2015). MDS research criteria for prodromal Parkinson's disease. *Movement Disorders*, *30*(12), 1600–1611. doi: 10.1002/mds.26431

Bhidayasiri, R., Rattanachaisit, W., Phokaewvarangkul, O., et al. (2019). Exploring bedside clinical features of parkinsonism: a focus on differential diagnosis. *Parkinsonism & Related Disorders*, *59*, 74–81. doi: 10.1016/j.parkreldis.2018.11.005

Bignall, T., Jeraj, S., Helsby, E. & Butt, J. (2019). *Racial disparities in mental health.* London: Race Equality Foundation.

Biousse, V., Skibell, B.C., Watts, R.L. et al. (2004). Ophthalmologic features of Parkinson's disease. *Neurology*, *62*(2), 177–180. doi: 10.1212/01.wnl.0000103444.45882.d8

Biundo, R., Weis, L., Fiorenzato, E. & Antonini, A. (2017). Cognitive rehabilitation in Parkinson's disease: is it feasible? *Archives of Clinical Neuropsychology*, *32*(7), 840–860. doi: 10.1093/arclin/acx092

Blundell, E.K., Grover, L.E., Stott, J. & Schrag, A. (2023). The experience of anxiety for people with Parkinson's disease. *npj Parkinson's Disease*, *9*(1), 75. doi: 10.1038/s41531-023-00512-1

Boecker, H., Ceballos-Baumann, A.O., Volk, D. et al. (2007). Metabolic alterations in patients with Parkinson disease and visual hallucinations. *Archives of Neurology*, *64*(7), 984–988. doi: 10.1001/archneur.64.7.984

Boubert, L. & Barnes, J. (2015). Phenomenology of visual hallucinations and their relationship to cognitive profile in Parkinson's disease patients: preliminary observations. *Sage Open*, *5*(2). doi: 10.1177/2158244015585827

Bougea, A., Maraki, M. I., Yannakoulia, M. et al. (2019). Higher probability of prodromal Parkinson disease is related to lower cognitive performance. *Neurology*, *92*(19), Article e2261–e2272. doi: 10.1212/WNL.0000000000007453

Braak, H., Del Tredici, K., Rub, U. et al. (2003). Staging of brain pathology related to sporadic Parkinson's disease. *Neurobiology of Aging, 24*(2), 197–211. doi: 10.1016/s0197-4580(02)00065-9

Braak, H., Müller, C. M., Rüb, U. et al. (2006). Pathology associated with sporadic Parkinson's disease—where does it end? In P. Reiderer, H. Reichmann, M.B.H. Youdim & M. Gerlach (Eds.) *Parkinson's Disease and Related Disorders. Journal of Neural Transmission. Supplementa, 70,* 89–97. Vienna: Springer. doi: 10.1007/978-3-211-45295-0_15

Brandstaedter, D., Spieker, S., Ulm, G. et al. (2005). Development and evaluation of the Parkinson Psychosis Questionnaire: A screening-instrument for the early diagnosis of drug-induced psychosis in Parkinson's disease. *Journal of Neurology*, *252*(9), 1060–1066. doi: 10.1007/s00415-005-0816-x

Broen, M.P., Narayen, N.E., Kuijf, M.L. et al. (2016). Prevalence of anxiety in Parkinson's disease: a systematic review and meta-analysis. *Movement Disorders*, *31*(8), 1125–1133. doi: 10.1002/mds.26643

Brown, R. & Jahanshahi, M. (1995). Depression in Parkinson's disease: a psychosocial viewpoint. *Advances in Neurology*, *65*, 61–84.

Brown, R.G., Landau, S., Hindle, J.V. et al. (2011). Depression and anxiety related subtypes in Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *82*(7), 803–809. doi: 10.1136/jnnp.2010.213652

Brown, R.G., MacCarthy, B., Gotham, A.M. et al. (1988). Depression and disability in Parkinson's disease: a follow-up of 132 cases. *Psychological Medicine*, *18*(1), 49–55. doi: 10.1017/s0033291700001872

Brown, R.G. & Marsden, C.D (1987). Neuropsychology and cognitive function in Parkinson's disease: an overview. In: C.D. Marsden & S. Fahn (Eds.) *Movement Disorders* (pp.99–123). New York and London: Butterworths.

BPS Professional Practice Board (2009). *Psychological services for people with Parkinson's disease.* Leicester: British Psychological Society. https://www.parkinsons.org.uk/sites/default/files/2017-12/psychologicalservices_briefingpaper.pdf

Burgess, P. W. & Shallice, T. (1997). *Hayling and Brixton Tests.* San Antonio, TX; The Psychological Corporation.

Burn, D.J., Landau, S., Hindle, J. V., et al. (2012). Parkinson's disease motor subtypes and mood. *Movement Disorders*, *27*(3), 379–386. doi: 10.1002/mds.24041

Burrell, J.R., Hodges, J.R. & Rowe, J.B. (2014). Cognition in corticobasal syndrome and progressive supranuclear palsy: A review. *Movement Disorders*, *29*(5), 684–693. doi: 10.1002/mds.25872

Cagnin, A., Gnoato, F., Jelcic, N. et al. (2013). Clinical and cognitive correlates of visual hallucinations in dementia with Lewy bodies. *Journal of Neurology, Neurosurgery & Psychiatry*, *84*(5), 505–510. doi: 10.1136/jnnp-2012-304095

Cartmill, T., Skvarc, D., Bittar, R. et al. (2021). Deep brain stimulation of the subthalamic nucleus in Parkinson's disease: a meta-analysis of mood effects. *Neuropsychology Review*, *31*(3) 385–401. doi: 10.1007/s11065-020-09467-z

Ceravolo, R., Nuti, A., Piccinni, A. et al. (2000). Paroxetine in Parkinson's disease: effects on motor and depressive symptoms. *Neurology*, *55*(8), 1216–1218. doi: 10.1212/wnl.55.8.1216

Chahine, L.M., Amara, A.W. & Videnovic, A. (2017). A systematic review of the literature on disorders of sleep and wakefulness in Parkinson's disease from 2005 to 2015. *Sleep Medicine Reviews*, *35*, 33–50. doi: 10.1016/j.smrv.2016.08.001

Chaudhuri, K.R. & Schapira, A.H. (2009). Non-motor symptoms of Parkinson's disease: dopaminergic pathophysiology and treatment. *The Lancet Neurology*, *8*(5), 464–474. doi: 10.1016/S1474-4422(09)70068-7

Choi, J.H., Kim, H.J., Lee, J.Y. et al. (2019). Long term effects of bilateral subthalamic nucleus stimulation on sleep in patients with Parkinson's disease. *PLoS One, 14*(8), Article e0221219. doi: 10.1371/journal.pone.0221219

Cipolotti, L., Spanò, B., Healy, C. et al. (2016). Inhibition processes are dissociable and lateralized in human prefrontal cortex. *Neuropsychologia*, *93*(Pt. A), 1–12. doi: 10.1016/j.neuropsychologia.2016.09.018

Cipriani, G., Vedovello, M., Nuti, A. & Di Fiorino, A. (2012). Dangerous passion: Othello syndrome and dementia. *Psychiatry and Clinical Neurosciences*, *66*(6), 467–473. doi: 10.1111/j.1440-1819.2012.02386.x

Cogan, D.G. (1973). Visual hallucinations as release phenomena. *Albrecht von Graefes Archiv für klinische und experimentelle Ophthalmologie*, *188*(2), 139–150. doi: 10.1007/BF00407835

Collerton, D. & Dudley, R. (2004). A cognitive behavioural framework for the treatment of distressing visual hallucinations in older people. *Behavioural and Cognitive Psychotherapy*, *32*(4), 443–455. doi: 10.1017/S1352465804001626

Collerton, D., Perry, E. & McKeith, I. (2005). Why people see things that are not there: a novel perception and attention deficit model for recurrent complex visual hallucinations. *Behavioral and Brain Sciences*, *28*(6), 737–757. doi: 10.1017/S0140525X05000130

Combs, H.L., Folley, B.S., Berry, D.T. et al. (2015). Cognition and depression following deep brain stimulation of the subthalamic nucleus and globus pallidus pars internus in Parkinson's disease: a meta-analysis. *Neuropsychology Review*, *25*, 439–454. doi: 10.1007/s11065-015-9302-0

Cools, R. (2006). Dopaminergic modulation of cognitive function-implications for L-DOPA treatment in Parkinson's Disease. *Neuroscience & Biobehavioral Reviews, 30*(1), 1–23. doi: 10.1016/j.neubiorev.2005.03.024

Cools, R., Barker, R.A., Sahakian, B.J. & Robbins, T.W. (2001). Enhanced or impaired cognitive function in Parkinson's disease as a function of dopaminergic medication and task demands. *Cerebral Cortex, 11*(12), 1136–1143. doi: 10.1093/cercor/11.12.1136

Cooper, J.A., Sagar, H.J., Doherty, S.M. et al. (1992). Different effects of dopaminergic and anticholinergic therapies on cognitive and motor function in Parkinson's disease: a follow-up study of untreated patients. *Brain*, *115*(6), 1701–1725. doi: 10.1093/brain/115.6.1701

Cummings, J.L. (1992). Depression and Parkinson's disease: A review. *The American Journal of Psychiatry*, *149*(4), 443–454. doi: 10.1176/ajp.149.4.443

Cummings, J.L., Mega, M., Gray, K. et al. (1994). The Neuropsychiatric Inventory: comprehensive assessment of psychopathology in dementia. *Neurology*, *44*(12), 2308. doi: 10.1212/wnl.44.12.2308

Dale, M., Wood, A., Zarotti, N. et al. (2022). Using a clinical formulation to understand psychological distress in people affected by Huntington's Disease: A descriptive, evidence-based model. *Journal of Personalized Medicine, 12*(8), 1222. doi: 10.3390/jpm12081222

Davidsdottir, S., Cronin-Golomb, A. & Lee, A. (2005). Visual and spatial symptoms in Parkinson's disease. *Vision Research*, *45*(10), 1285–1296. doi: 10.1016/j.visres.2004.11.006

de la Rosa, T. & Scorza, F.A. (2024). Contextualizing stigma in Parkinson's disease research. *Clinics, 79,* 100425. doi: 10.1016/j.clinsp.2024.100425

de Ridder, D., Geenen, R., Kuijer, R. & Van Middendorp, H. (2008). Psychological adjustment to chronic disease. *The Lancet*, *372*(9634), 246–255. doi: 10.1016/S0140-6736(08)61078-8

Delis, D.C., Kaplan, E. & Kramer, J.H. (2001). *Delis-Kaplan Executive Function System (DKEFS).* San Antonio, TX: The Psychological Corporation.

Delis, D.C., Kaplan, E. & Kramer, J.H. (2017). *California Verbal Learning Test-Version 3 (CVLT-3).* San Antonio, TX: The Psychological Corporation.

Deutsch, C.J., Robertson, N. & Miyasaki, J.M. (2021). Psychological impact of Parkinson disease delusions on spouse caregivers: A qualitative study. *Brain Sciences*, *11*(7), 871. doi: 10.3390/brainsci11070871

Diederich, N.J., Fénelon, G., Stebbins, G. & Goetz, C.G. (2009). Hallucinations in Parkinson disease. *Nature Reviews Neurology*, *5*(6), 331–342. doi: 10.1038/nrneurol.2009.62

Diederich, N.J., Goetz, C.G., Raman, R. et al. (1998). Poor visual discrimination and visual hallucinations in Parkinson's disease. *Clinical Neuropharmacology*, *21*(5), 289–295.

Diederich, N.J., Goetz, C.G. & Stebbins, G.T. (2005). Repeated visual hallucinations in Parkinson's disease as disturbed external/internal perceptions: focused review and a new integrative model. *Movement Disorders*, *20*(2), 130–140. doi: 10.1002/mds.20308

Diederich, N.J., Vaillant, M., Leischen, M. et al. (2005). Sleep apnea syndrome in Parkinson's disease. A case-control study in 49 patients. *Movement Disorders*, *20*(11), 1413–1418. doi: 10.1002/mds.20624

Dissanayaka, N.N., O'Sullivan, J.D., Pachana, N.A. et al. (2016). Disease-specific anxiety symptomatology in Parkinson's disease. *International Psychogeriatrics*, *28*(7), 1153–1163. doi: 10.1017/S1041610215002410

Dissanayaka, N.N., Torbey, E. & Pachana, N.A. (2015). Anxiety rating scales in Parkinson's disease: a critical review updating recent literature. *International Psychogeriatrics*, *27*(11), 1777–1784. doi: 10.1017/S1041610215000885

Dobkin, R.D., Menza, M., Allen, L.A. et al. (2011). Cognitive-behavioral therapy for depression in Parkinson's disease: a randomized, controlled trial. *American Journal of Psychiatry*, *168*(10), 1066–1074. doi: 10.1176/appi.ajp.2011.10111669

Dobkin, R.D., Rubino, J.T., Friedman, J. et al. (2013). Barriers to mental health care utilization in Parkinson's disease. *Journal of Geriatric Psychiatry and Neurology*, *26*(2), 105–116. doi: 10.1177/0891988713481269

Doé de Maindreville, A., Fénelon, G. & Mahieux, F. (2005). Hallucinations in Parkinson's disease: a follow-up study. *Movement Disorders*, *20*(2), 212–217. doi: 10.1002/mds.20263

Domellöf, M.E., Ekman, U., Forsgren, L. & Elgh, E. (2015). Cognitive function in the early phase of Parkinson's disease, a five-year follow-up. *Acta Neurologica Scandinavica*, *132*(2), 79–88. doi: 10.1111/ane.12375

Dorsey, E.R., Elbaz, A., Nichols, E., et al. (2018). Global, regional, and national burden of Parkinson's disease, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *The Lancet Neurology*, *17*(11), 939–953. doi: 10.1016/S1474-4422(18)30295-3

Dubois, B., Burn, D., Goetz, C. et al. (2007). Diagnostic procedures for Parkinson's disease dementia: recommendations from the movement disorder society task force. *Movement Disorders*, *22*(16), 2314–2324. doi: 10.1002/mds.21844

Dujardin, K., Sockeel, P., Delliaux, M. et al. (2009). Apathy may herald cognitive decline and dementia in Parkinson's disease. *Movement Disorders*, *24*(16), 2391–2397. doi: 10.1002/mds.22843

Eccles, F.J., Sowter, N., Spokes, T. et al. (2023). Stigma, self-compassion, and psychological distress among people with Parkinson's. *Disability and Rehabilitation*, *45*(3), 425–433. doi: 10.1080/09638288.2022.2037743

Ehgoetz Martens, K.A., Ellard, C.G. & Almeida, Q.J. (2014). Does anxiety cause freezing of gait in Parkinson's disease? *Plos one*, *9*(9), Article e106561. doi: 10.1371/journal.pone.0106561

Ehrt, U., Broich, K., Larsen, J.P. et al. (2010). Use of drugs with anticholinergic effect and impact on cognition in Parkinson's disease: a cohort study. *Journal of Neurology, Neurosurgery & Psychiatry*, *81*(2), 160–165. doi: 10.1136/jnnp.2009.186239

El Otmani, H., Sabiry, S., Bellakhdar, S. et al. (2021). Othello syndrome in Parkinson's disease: A diagnostic emergency of an underestimated condition. *Revue Neurologique*, *177*(6), 690–693. doi: 10.1016/j.neurol.2020.08.006

Emre, M., Aarsland, D., Brown, R. et al. (2007). Clinical diagnostic criteria for dementia associated with Parkinson's disease. *Movement Disorders*, *22*(12), 1689–1707. doi: 10.1002/mds.21507

Erro, R., Santangelo, G., Barone, P. et al. (2014). Do subjective memory complaints herald the onset of mild cognitive impairment in Parkinson disease? *Journal of Geriatric Psychiatry and Neurology*, *27*(4), 276–281. doi: 10.1177/0891988714532015

Evans, D. & Norman, P. (2009). Illness representation, coping and psychological adjustment to Parkinson's disease. *Psychology & Health, 24*(10), 1181–1196.

Evans, J. (2019). Neuropsychological rehabilitation: The Origins of Contemporary Practice. *Higher Brain Function Research*, *39*(3), 273–282. doi: 10.2496/hbfr.39.273

Factor, S.A., Scullin, M.K., Sollinger, A.B. et al. (2014). Cognitive correlates of hallucinations and delusions in Parkinson's disease. *Journal of the Neurological Sciences*, *347*(1–2), 316–321. doi: 10.1016/j.jns.2014.10.033

Fénelon, G. & Alves, G. (2010). Epidemiology of psychosis in Parkinson's disease. *Journal of the Neurological Sciences*, *289*(1–2), 12–17. doi: 10.1016/j.jns.2009.08.014

Fénelon, G., Mahieux, F., Huon, R. & Ziégler, M. (2000). Hallucinations in Parkinson's disease: prevalence, phenomenology and risk factors. *Brain*, *123*(4), 733–745. doi: 10.1093/brain/123.4.733

Fénelon, G., Parant, J. & de Langavant, L.C. (2021). Victor Parant (1848–1924) and the first report of psychosis in the course of Parkinson's disease with dementia. *Revue Neurologique*, *177*(10), 1221–1227. doi: 10.1016/j.neurol.2021.03.004

Fénelon, G., Soulas, T., de Langavant, L.C. et al. (2011). Feeling of presence in Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *82*(11), 1219–1224. doi: 10.1136/jnnp.2010.234799

Ffytche, D.H. & Aarsland, D. (2017). Psychosis in Parkinson's Disease. *International Review of Neurobiology*, *133*, 585–622. doi: 10.1016/bs.irn.2017.04.005

Ffytche, D.H., Creese, B., Politis, M. et al. (2017). The psychosis spectrum in Parkinson disease. *Nature Reviews Neurology*, *13*(2), 81–95. doi: 10.1038/nrneurol.2016.200

Foley, J.A., Chen, C., Paget, A. & Cipolotti, L. (2023). A Bayesian predictive processing account of Othello syndrome in Parkinson's disease. *Cognitive Neuropsychiatry*, *28*(4), 269–284. doi: 10.1080/13546805.2023.2229080

Foley, J.A., Dore, C., Zarkali, A. et al. (2020). Evaluation of START (STrAtegies for RelaTives) adapted for carers of people with Lewy body dementia. *Future Healthcare Journal*, *7*(3), e27–e29. doi: 10.7861/fhj.2020-0003

Foley, J.A., Foltynie, T., Limousin, P. & Cipolotti, L. (2018). Standardised neuropsychological assessment for the selection of patients undergoing DBS for Parkinson's disease. *Parkinson's Disease*, *2018*(1), 4328371. doi: 10.1155/2018/4328371

Foley, J.A., Niven, E.H., Abrahams, S. & Cipolotti, L. (2021). Phonemic fluency quantity and quality: Comparing patients with PSP, Parkinson's disease and focal frontal and subcortical lesions. *Neuropsychologia*, *153*, 107772. doi: 10.1016/j.neuropsychologia.2021.107772

Foley, J.A., Niven, E.H., Paget, A. et al. (2018). Sensitivity and specificity of the ECAS in Parkinson's disease and progressive supranuclear palsy. *Parkinson's Disease*, *2018*(1), 2426012. doi: 10.1155/2018/2426012

Foley, J., Warner, T.T. & Cipolotti, L. (2017). The neuropsychological profile of Othello syndrome in Parkinson's disease. *Cortex*, *96*, 158–160. doi: 10.1016/j.cortex.2017.08.009

Foley, J. & Willis, C. (2023). Prevalence and impact of mental health issues in Parkinson's disease unplanned hospital admissions. *Parkinsonism and Related Disorders, 115,* 105805. doi: 10.1016/j.parkreldis.2023.105805

Folstein, M.F., Folstein, S.E. & McHugh, P.R. (1975). 'Mini-mental state': A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatry Research, 12*(3), 189–198. doi: 10.1016/0022-3956(75)90026-6

Foltynie, T., Brayne, C.E., Robbins, T.W. & Barker, R.A. (2004). The cognitive ability of an incident cohort of Parkinson's patients in the UK. The CamPaIGN study. *Brain*, *127*(3), 550–560. doi: 10.1093/brain/awh067

Forsaa, E.B., Larsen, J.P., Wentzel-Larsen, T. & Alves, G. (2010). What predicts mortality in Parkinson disease? A prospective population-based long-term study. *Neurology*, *75*(14), 1270–1276. doi: 10.1212/WNL.0b013e3181f61311

Gallagher, D.A., Parkkinen, L., O'Sullivan, S.S. et al. (2011). Testing an aetiological model of visual hallucinations in Parkinson's disease. *Brain*, *134*(11), 3299–3309. doi: 10.1093/brain/awr225

Gallagher, D.A. & Schrag, A. (2012). Psychosis, apathy, depression and anxiety in Parkinson's disease. *Neurobiology of Disease*, *46*(3), 581–589. doi: 10.1016/j.nbd.2011.12.041

Garlovsky, J.K., Overton, P.G. & Simpson, J. (2016). Psychological predictors of anxiety and depression in Parkinson's disease: a systematic review. *Journal of Clinical Psychology*, *72*(10), 979–998. doi: 10.1002/jclp.22308

Gerstenecker, A. (2017). The neuropsychology (broadly conceived) of multiple system atrophy, progressive supranuclear palsy, and corticobasal degeneration. *Archives of Clinical Neuropsychology*, *32*(7), 861–875. doi: 10.1093/arclin/acx093

Getz, S.J. & Levin, B. (2017). Cognitive and neuropsychiatric features of early Parkinson's disease. *Archives of Clinical Neuropsychology*, *32*(7), 769–785. doi: 10.1093/arclin/acx091

Giannini, G., Francois, M., Lhommée, E. et al. (2019). Suicide and suicide attempts after subthalamic nucleus stimulation in Parkinson disease. *Neurology*, *93*(1), e97–e105. doi: 10.1212/WNL.0000000000007665

Giladi, N. & Hausdorff, J.M. (2006). The role of mental function in the pathogenesis of freezing of gait in Parkinson's disease. *Journal of the Neurological Sciences*, *248*(1–2), 173–176. doi: 10.1016/j.jns.2006.05.015

Goldman, J.G., Holden, S.K., Litvan, I. et al. (2018). Evolution of diagnostic criteria and assessments for Parkinson's disease mild cognitive impairment. *Movement Disorders*, *33*(4), 503–510. doi: 10.1002/mds.27323

Goldman, J.G., Stebbins, G.T., Dinh, V. et al. (2014). Visuoperceptive region atrophy independent of cognitive status in patients with Parkinson's disease with hallucinations. *Brain*, *137*(3), 849–859. doi: 10.1093/brain/awt360

Greenberg, S.A. (2007). How to Try This: The Geriatric Depression Scale: Short Form. *American Journal of Nursing, 107*(10), 60–69. doi: 10.1097/01.NAJ.0000292204.52313.f3

Greenland, J.C. & Barker, R.A. (2018). The differential diagnosis of Parkinson's disease. In T.B. Stoker & J.C. Greenland (Eds.) *Parkinson's Disease: Pathogenesis and Clinical Aspects* (pp.109–128). Brisbane: Codon Publications.

Greffard, S., Verny, M., Bonnet, A.M., et al. (2006). Motor score of the unified Parkinson disease rating scale as a good predictor of Lewy body–associated neuronal loss in the substantia nigra. *Archives of Neurology*, *63*(4), 584–588. doi: 10.1001/archneur.63.4.584

Haahr, A., Kirkevold, M., Hall, E.O. & Østergaard, K. (2011). Living with advanced Parkinson's disease: a constant struggle with unpredictability. *Journal of Advanced Nursing*, *67*(2), 408–417. doi: 10.1111/j.1365-2648.2010.05459.x

Hablitz, L.M. & Nedergaard, M. (2021). The glymphatic system: A novel component of fundamental neurobiology. *Journal of Neuroscience, 41*(37), 7698–7711. doi: 10.1523/JNEUROSCI.0619-21.2021

Hamilton, J.M., Landy, K.M., Salmon, D.P. et al. (2012). Early visuospatial deficits predict the occurrence of visual hallucinations in autopsy-confirmed dementia with Lewy bodies. *The American Journal of Geriatric Psychiatry*, *20*(9), 773–781. doi: 10.1097/JGP.0b013e31823033bc

Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology, 32*(1), 50–55. doi: 10.1111/j.2044-8341.1959.tb00467.x

Hamilton, M. (1960). *Hamilton Rating Scale for Depression (HRSD, HAM-D). Journal of Neurology, Neurosurgery and Psychiatry, 23*(1), 56–62. doi: 10.1136/jnnp.23.1.56

Harding, A.J., Broe, G.A. & Halliday, G.M. (2002). Visual hallucinations in Lewy body disease relate to Lewy bodies in the temporal lobe. *Brain*, *125*(2), 391–403. doi: 10.1093/brain/awf033

Heimrich, K.G., Schönenberg, A. & Prell, T. (2023). Social deprivation and exclusion in Parkinson's disease: A cross-sectional and longitudinal study. *BMJ Open, 13,* e074618. doi: 10.1136/bmjopen-2023-074618

Heinzel, S., Berg, D., Gasser, T. et al. (2019). Update of the MDS research criteria for prodromal Parkinson's disease. *Movement Disorders*, *34*(10), 1464–1470. doi: 10.1002/mds.27802

Hellqvist, C., Dizdar, N., Hagell, P. et al. (2018). Improving self-management for persons with Parkinson's disease through education focusing on management of daily life: Patients' and relatives' experience of the Swedish National Parkinson School. *Journal of Clinical Nursing*, *27*(19–20), 3719–3728. doi: 10.1111/jocn.14522

Hely, M.A., Reid, W.G., Adena, M.A. et al. (2008). The Sydney multicenter study of Parkinson's disease: the inevitability of dementia at 20 years. *Movement Disorders*, *23*(6), 837–844. doi: 10.1002/mds.21956

Hermann, W., Schmitz-Peiffer, H., Kasper, E. et al. (2020). Sleep disturbances and sleep disordered breathing impair cognitive performance in Parkinson's disease. *Frontiers in Neuroscience, 14,* 689. doi: 10.3389/fnins.2020.00689

Hindle, J.V., Watermeyer, T.J., Roberts, J. et al. (2018). Goal-orientated cognitive rehabilitation for dementias associated with Parkinson's disease – A pilot randomised controlled trial. *International Journal of Geriatric Psychiatry*, *33*(5), 718–728. doi: 10.1002/gps.4845

Högl, B., Arnulf, I., Comella, C. et al. (2010). Scales to assess sleep impairment in Parkinson's disease: critique and recommendations. *Movement Disorders*, *25*(16), 2704–2716. doi: 10.1002/mds.23190

Höglinger, G.U., Kassubek, J., Csoti, I. et al. (2017). Differentiation of atypical Parkinson syndromes. *Journal of Neural Transmission*, *124*, 997–1004. doi: 10.1007/s00702-017-1700-4

Holroyd, S., Currie, L. & Wooten, G.F. (2001). Prospective study of hallucinations and delusions in Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *70*(6), 734–738. doi: 10.1136/jnnp.70.6.734

Hughes, T.A., Ross, H.F., Mindham, R.H.S. & Spokes, E.G.S. (2004). Mortality in Parkinson's disease and its association with dementia and depression. *Acta Neurologica Scandinavica*, *110*(2), 118–123. doi: 10.1111/j.1600-0404.2004.00292.x

Humbert, M., Findley, J., Hernandez-Con, M. & Chahine, L.M. (2017). Cognitive behavioral therapy for insomnia in Parkinson's disease: a case series. *NPJ Parkinson's Disease, 3,* 25. doi: 10.1038/s41531-017-0027-z

Hwang, K.S., Beyer, M.K., Green, A.E. et al. (2013). Mapping cortical atrophy in Parkinson's disease patients with dementia. *Journal of Parkinson's Disease*, *3*(1), 69–76. doi: 10.3233/JPD-120151

Ibarretxe-Bilbao, N., Ramírez-Ruiz, B., Junque, C. et al. (2010). Differential progression of brain atrophy in Parkinson's disease with and without visual hallucinations. *Journal of Neurology, Neurosurgery & Psychiatry*, *81*(6), 650–657. doi: 10.1136/jnnp.2009.179655

Ino, T., Asada, T., Ito, J. et al. (2003). Parieto-frontal networks for clock drawing revealed with fMRI. *Neuroscience Research*, *45*(1), 71–77. doi: 10.1016/s0168-0102(02)00194-3

Ishihara, L. & Brayne, C. (2006). A systematic review of depression and mental illness preceding Parkinson's disease. *Acta Neurologica Scandinavica*, *113*(4), 211–220. doi: 10.1111/j.1600-0404.2006.00579.x

Jacob, E.L., Gatto, N.M., Thompson, A. et al. (2010). Occurrence of depression and anxiety prior to Parkinson's disease. *Parkinsonism & Related Disorders*, *16*(9), 576–581. doi: 10.1016/j.parkreldis.2010.06.014

Jacob, S.N. & Nienborg, H. (2018). Monoaminergic neuromodulation of sensory processing. *Frontiers in Neural Circuits*, *12*, 51. doi: 10.3389/fncir.2018.00051

Joy, S.P., Sinha, S., Pal, P.K. et al. (2014). Alterations in Polysomnographic (PSG) profile in drug-naïve Parkinson's disease. *Annals of Indian Academy of Neurology, 17*(3), 287–291. doi: 10.4103/0972-2327.138501

Junaid, O. & Hegde, S. (2007). Supportive psychotherapy in dementia. *Advances in Psychiatric Treatment*, *13*(1), 17–23. doi: 10.1192/apt.bp.105.002030

Karacan, A.V., Kibrit, S.N., Yekedüz, M.K. et al. (2023). Cross-cultural difference in stigma associated with Parkinson's Disease: A systematic review. *Journal of Parkinson's Disease, 13*(5), 699–715. doi: 10.3233/JPD-230050

Katzen, H., Myerson, C., Papapetropoulos, S. et al. (2010). Multi-modal hallucinations and cognitive function in Parkinson's disease. *Dementia and Geriatric Cognitive Disorders*, *30*(1), 51–56. doi: 10.1159/000314875

Kehagia, A.A., Barker, R.A. & Robbins, T.W. (2010). Neuropsychological and clinical heterogeneity of cognitive impairment and dementia in patients with Parkinson's disease. *The Lancet Neurology*, *9*(12), 1200–1213. doi: 10.1016/S1474-4422(10)70212-X

Kehagia, A.A., Barker, R.A. & Robbins, T.W. (2012). Cognitive Impairment in Parkinson's Disease: The Dual Syndrome Hypothesis. *Neurodegenerative Diseases, 11*(2), 79–82. doi: 10.1159/000341998

Kelly, M.J., Baig, F., Hu, M.T.M. & Okai, D. (2020). Spectrum of impulse control behaviours in Parkinson's disease: pathophysiology and management. *Journal of Neurology, Neurosurgery & Psychiatry*, *91*(7), 703–711. doi: 10.1136/jnnp-2019-322453

Kertesz, A., McMonagle, P., Blair, M. et al. (2005). The evolution and pathology of frontotemporal dementia. *Brain*, *128*(9), 1996–2005. doi: 10.1093/brain/awh598

Kingham, M. & Gordon, H. (2004). Aspects of morbid jealousy. *Advances in Psychiatric Treatment*, *10*(3), 207–215. doi: 10.1192/apt.10.3.207

Kinsbourne, M. & Warrington, E.K. (1963). A study of visual perseveration. *Journal of Neurology, Neurosurgery, and Psychiatry*, *26*(5), 468–475. doi: 10.1136/jnnp.26.5.468

Kish, S.J., Shannak, K. & Hornykiewicz, O. (1988). Uneven pattern of dopamine loss in the striatum of patients with idiopathic Parkinson's disease. *New England Journal of Medicine*, *318*(14), 876–880. doi: 10.1056/NEJM198804073181402

Kleiner-Fisman, G., Herzog, J., Fisman, D.N., et al. (2006). Subthalamic nucleus deep brain stimulation: summary and meta-analysis of outcomes. *Movement Disorders*, *21*(S14), S290–S304. doi: 10.1002/mds.20962

Koerts, J., Borg, M.A., Meppelink, A.M. et al. (2010). Attentional and perceptual impairments in Parkinson's disease with visual hallucinations. *Parkinsonism & Related Disorders*, *16*(4), 270–274. doi: 10.1016/j.parkreldis.2010.01.003

https://doi.org/10.53841/bpsrep.2025.rep186.10

Kostić, V.S., Agosta, F., Petrović, I. et al. (2010). Regional patterns of brain tissue loss associated with depression in Parkinson disease. *Neurology*, *75*(10), 857–863. doi: 10.1212/WNL.0b013e3181f11c1d

Krack, P., Batir, A., Van Blercom, N. et al. (2003). Five-year follow-up of bilateral stimulation of the subthalamic nucleus in advanced Parkinson's disease. *New England Journal of Medicine*, *349*(20), 1925–1934. doi: 10.1056/NEJMoa035275

Kulick, C.V., Montgomery, K.M. & Nirenberg, M.J. (2018). Comprehensive identification of delusions and olfactory, tactile, gustatory, and minor hallucinations in Parkinson's disease psychosis. *Parkinsonism & Related Disorders*, *54*, 40–45. doi: 10.1016/j.parkreldis.2018.04.008

Kummer, A., Cardoso, F. & Teixeira, A.L. (2009). Suicidal ideation in Parkinson's disease. *CNS Spectrums*, *14*(8), 431–436. doi: 10.1017/s109285290002040x

Kyrtsos, C.R., Stahl, M.C., Eslinger, P. et al. (2015). Capgras syndrome in a patient with Parkinson's Disease after bilateral subthalamic nucleus deep brain stimulation: A case report. *Case Reports in Neurology, 7*(2), 127–133. doi: 10.1159/000431081

Landau, S., Harris, V., Burn, D.J. et al. (2016). Anxiety and anxious-depression in Parkinson's disease over a 4-year period: a latent transition analysis. *Psychological Medicine*, *46*(3), 657–667. doi: 10.1017/S0033291715002196

Lang, A.E. & Widner, H. (2002). Deep brain stimulation for Parkinson's disease: patient selection and evaluation. *Movement Disorders*, *17*(S3), S94–S101. doi: 10.1002/mds.10149

Lau, Y.H., Podlewska, A., Ocloo, J. et al. (2022). Does ethnicity influence recruitment into clinical trials of Parkinson's Disease? *Journal of Parkinson's Disease, 12*(3), 975–981. doi: 10.3233/JPD-213113

Lauterbach, E.C., Freeman, A. & Vogel, R.L. (2003). Correlates of generalized anxiety and panic attacks in dystonia and Parkinson disease. *Cognitive and Behavioral Neurology*, *16*(4), 225–233. doi: 10.1097/00146965-200312000-00004

Leclerq, V. & Corvol, J.-C. (2024). Impulse control disorder: Review on clinical, pharmacologic, and genetic risk factors. *Revue Neurologique, 180*(10), 1071–1077. doi: 10.1016/j.neurol.2024.07.001

Leentjens, A.F.G. (2011). The role of dopamine agonists in the treatment of depression in patients with Parkinson's disease: a systematic review. *Drugs*, *71*(3), 273–286. doi: 10.2165/11585380-000000000-00000

Leentjens, A.F.G., Dujardin, K., Marsh, L. et al. (2008). Anxiety rating scales in Parkinson's disease: critique and recommendations. *Movement Disorders*, *23*(14), 2015–2025. doi: 10.1002/mds.22233

Leentjens, A.F.G., Dujardin, K., Pontone, G.M. et al. (2014). The Parkinson Anxiety Scale (PAS): Development and validation of a new anxiety scale. *Movement Disorders, 29*(8), 1035–1043. doi: 10.1002/mds.25919

Leentjens, A.F.G., Lousberg, R. & Verhey, F.R.J. (2002). Markers for depression in Parkinson's disease. *Acta Psychiatrica Scandinavica*, *106*(3), 196–201. doi: 10.1034/j.16000447.2002.02045.x

Leentjens, A.F.G., Moonen, A.J., Dujardin, K. et al. (2013). Modeling depression in Parkinson disease: disease-specific and nonspecific risk factors. *Neurology*, *81*(12), 1036–1043. doi: 10.1212/WNL.0b013e3182a4a503

Leentjens, A.F.G., Van den Akker, M., Metsemakers, J.F. et al. (2003). Higher incidence of depression preceding the onset of Parkinson's disease: a register study. *Movement Disorders*, *18*(4), 414–418. doi: 10.1002/mds.10387

Lemke, M.R. (2008). Depressive symptoms in Parkinson's disease. *European Journal of Neurology*, *15*(S1), 21–25. doi: 10.1111/j.1468-1331.2008.02058.x

Lenka, A., Pagonabarraga, J., Pal, P.K. et al. (2019). Minor hallucinations in Parkinson disease: a subtle symptom with major clinical implications. *Neurology*, *93*(6), 259–266. doi: 10.1212/WNL.0000000000007913

Leong, G.B., Arturo Silva, J., Garza-Treviño, E.S. et al. (1994). The dangerousness of persons with the Othello syndrome. *Journal of Forensic Sciences*, *39*(6), 1445–1454.

Leroi, I., Harbishettar, V., Andrews, M. et al. (2012). Carer burden in apathy and impulse control disorders in Parkinson's disease. *International Journal of Geriatric Psychiatry*, *27*(2), 160–166. doi: 10.1002/gps.2704

Leroi, I., Vatter, S., Carter, L.A. et al. (2019). Parkinson's-adapted cognitive stimulation therapy: a pilot randomized controlled clinical trial. *Therapeutic Advances in Neurological Disorders*, *12*, 1756286419852217. doi: 10.1177/1756286419852217

Leung, I.H.K., Walton, C.C., Hallock, H. et al. (2015). Cognitive training in Parkinson disease: A systematic review and meta-analysis. *Neurology, 85*(21), 1843–1851. doi: 10.1212/WNL.0000000000002145

Lewis, S.J.G., Foltynie, T., Blackwell, A.D. et al. (2005). Heterogeneity of Parkinson's disease in the early clinical stages using a data driven approach. *Journal of Neurology, Neurosurgery & Psychiatry*, *76*(3) 343–348. doi: 10.1136/jnnp.2003.033530

Lin, J., Ou, R., Wei, Q. et al. (2022). Self-stigma in Parkinson's disease: a 3-year prospective cohort study. *Frontiers in Ageing & Neuroscience, 14,* 790897. doi: 10.3389/fnagi.2022.790897

Ling, H., O'Sullivan, S.S., Holton, J.L. et al. (2010). Does corticobasal degeneration exist? A clinicopathological re-evaluation. *Brain*, *133*(7), 2045–2057. doi: 10.1093/brain/awq123

Litvan, I., Aarsland, D., Adler, C.H. et al. (2011). MDS Task Force on mild cognitive impairment in Parkinson's disease: critical review of PD–MCI. *Movement Disorders*, *26*(10), 1814–1824. doi: 10.1002/mds.23823

Litvan, I., Goldman, J.G., Tröster, A.I. et al. (2012). Diagnostic criteria for mild cognitive impairment in Parkinson's disease: Movement Disorder Society Task Force guidelines. *Movement Disorders*, *27*(3), 349–356. doi: 10.1002/mds.24893

Lozano, A.M., Lipsman, N., Bergman, H. et al. (2019). Deep brain stimulation: current challenges and future directions. *Nature Reviews Neurology*, *15*(3), 148–160. doi: 10.1038/s41582-018-0128-2

Lythe, V., Athauda, D., Foley, J. et al. (2017). GBA-associated Parkinson's disease: progression in a deep brain stimulation cohort. *Journal of Parkinson's Disease*, *7*(4), 635–644. doi: 10.3233/JPD-171172

Ma, H.I., Saint-Hilaire, M., Thomas, C.A. & Tickle-Degnen, L. (2016). Stigma as a key determinant of health-related quality of life in Parkinson's disease. *Quality of Life Research*, *25*, 3037–3045. doi: 10.1007/s11136-016-1329-z

MacCarthy, B. & Brown, R. (1989). Psychosocial factors in Parkinson's disease. *British Journal of Clinical Psychology*, *28*(1), 41–52. doi: 10.1111/j.2044-8260.1989.tb00810.x

Marin, R.S., Biedrzycki, R.C. & Firinciogullari, S. (1991). Reliability and validity of the Apathy Evaluation Scale. *Psychiatry Research, 38*(2), 143–162. doi: 10.1016/0165-1781(91)90040-v

Marinus, J., Visser, M., van Hilten, J.J., et al. (2003). Assessment of sleep and sleepiness in Parkinson disease. *Sleep*, *26*(8), 1049–1054. doi: 10.1093/sleep/26.8.1049

Marinus, J., Zhu, K., Marras, C. et al. (2018). Risk factors for non-motor symptoms in Parkinson's disease. *The Lancet Neurology*, *17*(6), 559–568. doi: 10.1016/S1474-4422(18)30127-3

Marques, A., Beze, S., Pereira, B. et al. (2020). Visual hallucinations and illusions in Parkinson's disease: the role of ocular pathology. *Journal of Neurology*, *267*(10), 2829–2841. doi: 10.1007/s00415-020-09925-x

Marsili, L., Rizzo, G. & Colosimo, C. (2018). Diagnostic criteria for Parkinson's disease: from James Parkinson to the concept of prodromal disease. *Frontiers in Neurology*, *9*, 156. doi: 10.3389/fneur.2018.00156

Martinez-Martin, P., Visser, M., Rodriguez-Blazquez, C. et al. (2008). SCOPA-sleep and PDSS: two scales for assessment of sleep disorder in Parkinson's disease. *Movement Disorders*, *23*(12), 1681–1688. doi: 10.1002/mds.22110

Martini, A., Dal Lago, D., Edelstyn, N.M. et al. (2018). Impulse control disorder in Parkinson's disease: a meta-analysis of cognitive, affective, and motivational correlates. *Frontiers in Neurology*, *9*, 654. doi: 10.3389/fneur.2018.00654

McKeith, I.G., Boeve, B.F., Dickson, D.W. et al. (2017). Diagnosis and management of dementia with Lewy bodies: Fourth consensus report of the DLB Consortium. *Neurology*, *89*(1), 88–100. doi: 10.1212/WNL.0000000000004058

McKenna, P. & Warrington, E.K. (1980). Testing for nominal dysphasia. *Journal of Neurology, Neurosurgery & Psychiatry, 43*(9), 781–788. doi: 10.1136/jnnp.43.9.781

McKinlay, A., Grace, R.C., Dalrymple-Alford, J.C. et al. (2008a). A profile of neuropsychiatric problems and their relationship to quality of life for Parkinson's disease patients without dementia. *Parkinsonism & Related Disorders*, *14*(1), 37–42. doi: 10.1016/j.parkreldis.2007.05.009

McKinlay, A., Grace, R.C., Dalrymple-Alford, J.C. et al. (2008b). Neuropsychiatric problems in Parkinson's disease: comparisons between self and caregiver report. *Aging and Mental Health*, *12*(5), 647-653. doi: 10.1080/13607860802343225

McNamara, P. & Durso, R. (1991). Reversible pathologic jealousy (Othello syndrome) associated with amantadine. *Journal of Geriatric Psychiatry and Neurology*, *4*(3), 157–159. doi: 10.1177/089198879100400306

Mele, B., Merrikh, D., Ismail, Z. & Goodarzi, Z. (2019). Detecting apathy in individuals with Parkinson's disease: a systematic review. *Journal of Parkinson's Disease*, *9*(4), 653–664. doi: 10.3233/JPD-191619

Minces, V., Pinto, L., Dan, Y. & Chiba, A.A. (2017). Cholinergic shaping of neural correlations. *Proceedings of the National Academy of Sciences of the United States of America*, *114*(22), 5725–5730. doi: 10.1073/pnas.1621493114

Mindham, R.H. (1970). Psychiatric symptoms in Parkinsonism. *Journal of Neurology, Neurosurgery, and Psychiatry*, *33*(2), 188–191. doi: 10.1136/jnnp.33.2.188

Montgomery, S.A. & Åsberg, M. (1979) A new depression scale designed to be sensitive to change. *British Journal of Psychiatry, 134,* 382–389. doi: 10.1192/bjp.134.4.382

Moonen, A.J., Mulders, A.E., Defebvre, L. et al. (2021). Cognitive behavioral therapy for anxiety in Parkinson's disease: a randomized controlled trial. *Movement Disorders*, *36*(11), 2539–2548. doi: 10.1002/mds.28533

Mori, E., Shimomura, T., Fujimori, M. et al. (2000). Visuoperceptual impairment in dementia with Lewy bodies. *Archives of Neurology*, *57*(4), 489–493. doi: 10.1001/archneur.57.4.489

Mosimann, U.P., Mather, G., Wesnes, K.A. et al. (2004). Visual perception in Parkinson disease dementia and dementia with Lewy bodies. *Neurology*, *63*(11), 2091–2096. doi: 10.1212/01.wnl.0000145764.70698.4e

Moustafa, A.A. & Poletti, M. (2013). Neural and behavioral substrates of subtypes of Parkinson's disease. *Frontiers in Systems Neuroscience*, *7*, 117. doi: 10.3389/fnsys.2013.00117

Mshana, G., Dotchin, C.L. & Walker, R.W. (2011). 'We call it the shaking illness': perceptions and experiences of Parkinson's disease in rural northern Tanzania. *BMC Public Health*, *11*, 219. doi: 10.1186/1471-2458-11-219

Muhammed, K. & Husain, M. (2016). Clinical significance of apathy in Parkinson's disease. *European Medical Journal: Neurology*, *4*(1), 56–63. doi: 10.33590/emjneurol/10312945.

Müller, B., Assmus, J., Herlofson, K. et al. (2013). Importance of motor vs. non-motor symptoms for health-related quality of life in early Parkinson's disease. *Parkinsonism & Related Disorders*, *19*(11), 1027–1032. doi: 10.1016/j.parkreldis.2013.07.010

Müller, T. (2002). Drug treatment of non-motor symptoms in Parkinson's disease. *Expert Opinion on Pharmacotherapy*, *3*(4), 381-388. doi: 10.1517/14656566.3.4.381

National Institute for Health and Care Excellence. (2009). *Depression in adults with a chronic physical health problem: recognition and management (NICE guideline CG91).* https://www.nice.org.uk/guidance/cg91

National Institute for Health and Care Excellence (2017). *Parkinson's disease in adults: diagnosis and management (NICE guideline NG71).* www.nice.org.uk/guidance/ng71.

Nehra, A., Makkar, R., Singh, R.K. et al. (2023). Is Disease a Threat to Identity? A Systematic Review of Parkinson's Disease and Personal Identity. *Indian Journal of Public Health, 67*(1), 117–122. doi: 10.4103/ijph.ijph_1283_22

Nishio, Y., Yokoi, K., Hirayama, K. et al. (2018). Defining visual illusions in Parkinson's disease: Kinetopsia and object misidentification illusions. *Parkinsonism & Related Disorders*, *55*, 111–116. doi: 10.1016/j.parkreldis.2018.05.023

O'Callaghan, C., Hall, J.M., Tomassini, A. et al. (2017). Visual hallucinations are characterised by impaired sensory evidence accumulation: insights from hierarchical drift diffusion modeling in Parkinson's disease. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *2*(8), 680–688. doi: 10.1016/j.bpsc.2017.04.007

Oedekoven, C., Egeri, L., Jessen, F. et al. (2022). Subjective cognitive decline in idiopathic Parkinson's disease: A systematic review. *Ageing research reviews*, *74*, 101508. doi: 10.1016/j.arr.2021.101508

Oehlberg, K., Barg, F.K., Brown, G.K. et al. (2008). Attitudes regarding the etiology and treatment of depression in Parkinson's disease: a qualitative study. *Journal of Geriatric Psychiatry and Neurology*, *21*(2), 123–132. doi: 10.1177/0891988708316862

Okai, D., Askey-Jones, S., Mack, J. et al. (2016). Parkinson's impulse-control scale for the severity rating of impulse-control behaviors in Parkinson's disease: a semistructured clinical assessment tool. *Movement Disorders Clinical Practice*, *3*(5), 494–499. doi: 10.1002/mdc3.12316

Okai, D., Askey-Jones, S., Samuel, M. et al. (2013). Trial of CBT for impulse control behaviors affecting Parkinson patients and their caregivers. *Neurology*, *80*(9), 792–799. doi: 10.1212/WNL.0b013e3182840678

Okai, D., Askey-Jones, S., Samuel, M. et al. (2015). Predictors of response to a cognitive behavioral intervention for impulse control behaviors in Parkinson's disease. *Movement Disorders*, *30*(5), 736–739. doi: 10.1002/mds.26108

Okun, M.S., Rodriguez, R.L., Mikos, A. et al. (2007). Deep brain stimulation and the role of the neuropsychologist. *The Clinical Neuropsychologist*, *21*(1), 162–189. doi: 10.1080/13825580601025940

Ondo, W.G. & Lai, D. (2008). Predictors of impulsivity and reward seeking behavior with dopamine agonists. *Parkinsonism & Related Disorders*, *14*(1), 28–32. doi: 10.1016/j.parkreldis.2007.05.006

O'Sullivan, S.S., Evans, A.H. & Lees, A.J. (2007). Punding in Parkinson's disease. *Practical Neurology*, *7*(6), 397–399. doi: 10.1136/jnnp.2007.129015

O'Sullivan, S.S., Williams, D.R., Gallagher, D.A. et al. (2008). Nonmotor symptoms as presenting complaints in Parkinson's disease: a clinicopathological study. *Movement Disorders*, *23*(1), 101–106. doi: 10.1002/mds.21813

Pagonabarraga, J., Kulisevsky, J., Strafella, A.P. & Krack, P. (2015). Apathy in Parkinson's disease: clinical features, neural substrates, diagnosis, and treatment. *The Lancet Neurology*, *14*(5), 518–531. doi: 10.1016/S1474-4422(15)00019-8

Pagonabarraga, J., Martinez-Horta, S., Fernández de Bobadilla, R., et al. (2016). Minor hallucinations occur in drug-naive Parkinson's disease patients, even from the premotor phase. *Movement Disorders*, *31*(1), 45–52. doi: 10.1002/mds.26432

Pagonabarraga, J., Soriano-Mas, C., Llebaria, G. et al. (2014). Neural correlates of minor hallucinations in non-demented patients with Parkinson's disease. *Parkinsonism & Related Disorders*, *20*(3), 290–296. doi: 10.1016/j.parkreldis.2013.11.017

Pappatá, S., Santangelo, G., Aarsland, D. et al. (2011). Mild cognitive impairment in drug-naive patients with PD is associated with cerebral hypometabolism. *Neurology*, *77*(14), 1357–1362. doi: 10.1212/WNL.0b013e3182315259

Parkinson, J. (2002). An essay on the shaking palsy. 1817. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *14*(2), 223–236. doi: 10.1176/jnp.14.2.223

Parkinson's Foundation (2021) *Statistics*. Accessed 26 September 2024 from https://www.parkinson.org/understanding-parkinsons/statistics

Parkinson's UK (2021). *Nobody really knows us: The state of health and social care for people with Parkinson's-related dementia*. https://www.parkinsons.org.uk/sites/default/files/2021-11/PUK0973_Dementia%20policy%20report_CS3739_web_SIGN%20OFF.pdf

Parra-Diaz, P., Chico-Garcia, J.L., Beltran-Corbellini, A. et al. (2021). Does the country make a different in impulse control disorders? A systematic review. *Movement Disorders Clinical Practice, 8*(1), 25–32. doi: 10.1002/mdc3.13128

Paulus, W. & Jellinger, K. (1991). The neuropathologic basis of different clinical subgroups of Parkinson's disease. *Journal of Neuropathology & Experimental Neurology*, *50*(6), 743–755. doi: 10.1097/00005072-199111000-00006

Paumier, K.L., Siderowf, A.D., Auinger, P. et al. (2012). Tricyclic antidepressants delay the need for dopaminergic therapy in early Parkinson's disease. *Movement Disorders*, *27*(7), 880–887. doi: 10.1002/mds.24978

Paviour, D.C., Price, S.L., Jahanshahi, M. et al. (2006). Regional brain volumes distinguish PSP, MSA-P, and PD: MRI-based clinico-radiological correlations. *Movement Disorders*, *21*(7), 989–996. doi: 10.1002/mds.20877

Pedersen, K.F., Larsen, J.P., Tysnes, O.B. & Alves, G. (2017). Natural course of mild cognitive impairment in Parkinson disease: a 5-year population-based study. *Neurology*, *88*(8), 767–774. doi: 10.1212/WNL.0000000000003634

Perry, E.K. & Perry, R.H. (1995). Acetylcholine and hallucinations: disease-related compared to drug-induced alterations in human consciousness. *Brain and Cognition*, *28*(3), 240–258. doi: 10.1006/brcg.1995.1255

Peterson, K.A., Patterson, K. & Rowe, J.B. (2021). Language impairment in progressive supranuclear palsy and corticobasal syndrome. *Journal of Neurology*, *268*(3), 796–809. doi: 10.1007/s00415-019-09463-1

Podgorski, C.A., Anderson, S.D. & Parmar, J. (2021). A biopsychosocial-ecological framework for family-framed dementia care. *Frontiers in Psychiatry, 12,* 744806. doi: 10.3389/fpsyt.2021.744806

Poletti, M., Frosini, D., Pagni, C. et al. (2012). Mild cognitive impairment and cognitive-motor relationships in newly diagnosed drug-naive patients with Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *83*(6), 601–606. doi: 10.1136/jnnp-2011-301874

Poletti, M. & Bonuccelli, U. (2013). From aberrant salience to jumping to conclusions: dopaminergic pathways to delusions in Parkinson disease. *Journal of Clinical Psychopharmacology*, *33*(2), 149–151. doi: 10.1097/JCP.0b013e3182854989

Pollak, P., Fraix, V., Krack, P. et al. (2002). Treatment results: Parkinson's disease. *Movement Disorders*, *17*(S3), S75–S83. doi: 10.1002/mds.10146

Pontone, G.M., Bakker, C.C., Chen, S. et al. (2016). The longitudinal impact of depression on disability in Parkinson disease. *International Journal of Geriatric Psychiatry*, *31*(5), 458–465. doi: 10.1002/gps.4350

Pontone, G.M. & Mills, K.A. (2021). Optimal treatment of depression and anxiety in Parkinson's disease. *The American Journal of Geriatric Psychiatry*, *29*(6), 530–540. doi: 10.1016/j.jagp.2021.02.037

Pontone, G.M., Williams, J.R., Anderson, K.E. et al. (2009). Prevalence of anxiety disorders and anxiety subtypes in patients with Parkinson's disease. *Movement Disorders*, *24*(9), 1333–1338. doi: 10.1002/mds.22611

Prediger, R.D., Matheus, F.C., Schwarzbold, M.L. et al. (2012). Anxiety in Parkinson's disease: a critical review of experimental and clinical studies. *Neuropharmacology*, *62*(1), 115–124. doi: 10.1016/j.neuropharm.2011.08.039

Puntambekar, I. & Foley, J.A. (2023). Neuropsychological correlates of visual hallucinatory phenomena in Lewy body disease. *International Journal of Geriatric Psychiatry*, *38*(6), e5950. doi: 10.1002/gps.5950

Radakovic, R. & Abrahams, S. (2014). Developing a new apathy measurement scale: Dimensional Apathy Scale. *Psychiatry Research, 219*(3), 658–663. doi: 10.1016/j.psychres.2014.06.010

Ramezani, M., Mouches, P., Yoon, E. et al. (2021). Investigating the relationship between the SNCA gene and cognitive abilities in idiopathic Parkinson's disease using machine learning. *Scientific Reports*, *11*(1), 4917. doi: 10.1038/s41598-021-84316-4

Ramírez-Ruiz, B., Junqué, C., Martí, M.J. et al. (2006). Neuropsychological deficits in Parkinson's disease patients with visual hallucinations. *Movement Disorders*, *21*(9), 1483–1487. doi: 10.1002/mds.20965

Ramírez-Ruiz, B., Junqué, C., Martí, M.J. et al. (2007). Cognitive changes in Parkinson's disease patients with visual hallucinations. *Dementia and Geriatric Cognitive Disorders*, *23*(5), 281–288. doi: 10.1159/000100850

Ramírez-Ruiz, B., Martí, M.J., Tolosa, E. et al. (2007). Cerebral atrophy in Parkinson's disease patients with visual hallucinations. *European Journal of Neurology*, *14*(7), 750–756. doi: 10.1111/j.1468-1331.2007.01768.x

Rana, A.Q., Athar, A., Owlia, A. et al. (2012). Impact of ethnicity on non-motor symptoms of Parkinson's Disease. *Journal of Parkinson's Disease, 2*(4), 281–285. doi: 10.3233/JPD-129002

Raven, J. & Raven, J. (2003). Raven Progressive Matrices. In R.S. McCallum (Ed.), *Handbook of nonverbal assessment* (pp.223–237). New York: Kluwer Academic/Plenum Publishers. doi: 10.1007/978-1-4615-0153-4_11

Ravina, B., Camicioli, R., Como, P. G., et al. (2007). The impact of depressive symptoms in early Parkinson disease. *Neurology, 69*(4), 342–347. doi: 10.1212/01.wnl.0000268695.63392.10

Reckner, E., Cipolotti, L. & Foley, J.A. (2020). Presence phenomena in parkinsonian disorders: Phenomenology and neuropsychological correlates. *International Journal of Geriatric Psychiatry*, *35*(7), 785–793. doi: 10.1002/gps.5303

Rees, R.N., Acharya, A.P., Schrag, A. & Noyce, A.J. (2018). An early diagnosis is not the same as a timely diagnosis of Parkinson's disease. *F1000Research*, *7*(Faculty Rev) 1106. doi: 10.12688/f1000research.14528.1

Reijnders, J.S.A.M., Ehrt, U., Lousberg, R. et al. (2009). The association between motor subtypes and psychopathology in Parkinson's disease. *Parkinsonism & Related Disorders*, *15*(5), 379–382. doi: 10.1016/j.parkreldis.2008.09.003

Rieu, I., Martinez-Martin, P., Pereira, B. et al. (2015). International validation of a behavioral scale in Parkinson's disease without dementia. *Movement Disorders*, *30*(5), 705–713. doi: 10.1002/mds.26223

Rittman, T., Coyle-Gilchrist, I.T. & Rowe, J.B. (2016). Managing cognition in progressive supranuclear palsy. *Neurodegenerative Disease Management*, *6*(6), 499–508. doi: 10.2217/nmt-2016-0027

Rittman, T., Ghosh, B.C., McColgan, P. et al. (2013). The Addenbrooke's Cognitive Examination for the differential diagnosis and longitudinal assessment of patients with parkinsonian disorders. *Journal of Neurology, Neurosurgery & Psychiatry*, *84*(5), 544–551. doi: 10.1136/jnnp-2012-303618

Rocca, W.A. (2018). The burden of Parkinson's disease: a worldwide perspective. *The Lancet Neurology*, *17*(11), 928–929. doi: 10.1016/S1474-4422(18)30355-7

Rosenberg-Katz, K., Herman, T., Jacob, Y. et al. (2013). Gray matter atrophy distinguishes between Parkinson disease motor subtypes. *Neurology*, *80*(16), 1476–1484. doi: 10.1212/WNL.0b013e31828cfaa4

Sanchez-Castaneda, C., Rene, R., Ramírez-Ruiz, B. et al. (2010). Frontal and associative visual areas related to visual hallucinations in dementia with Lewy bodies and Parkinson's disease with dementia. *Movement Disorders*, *25*(5), 615–622. doi: 10.1002/mds.22873

Saredakis, D., Collins-Praino, L.E., Gutteridge, D.S. et al. (2019). Conversion to MCI and dementia in Parkinson's disease: a systematic review and meta-analysis. *Parkinsonism & Related Disorders*, *65*, 20–31. doi: 10.1016/j.parkreldis.2019.04.020

Savage, G. (2016). Cognitive neuropsychological formulation. In J.A.B. Macniven (Ed.) *Neuropsychological Formulation: A Clinical Casebook* (pp.221–239). New York: Springer.

Schrag, A., Barone, P., Brown, R.G. et al. (2007). Depression rating scales in Parkinson's disease: critique and recommendations. *Movement Disorders*, *22*(8), 1077–1092. doi: 10.1002/mds.21333

Schrag, A., Horsfall, L., Walters, K. et al. (2015). Prediagnostic presentations of Parkinson's disease in primary care: a case-control study. *The Lancet Neurology*, *14*(1), 57–64.

Schrag, A., Jahanshahi, M. & Quinn, N.P. (2001). What contributes to depression in Parkinson's disease? *Psychological Medicine*, *31*(1), 65–73. doi: 10.1017/s0033291799003141

Schütz, L., Sixel-Döring, F. & Hermann, W. (2022). Management of sleep disturbances in Parkinson's Disease. *Journal of Parkinson's Disease, 12*(7), 2029–2058. doi: 10.3233/JPD-212749

Segal, D.L., June, A., Payne, M. et al. (2010). Development and initial validation of a self-report assessment tool for anxiety among older adults: The Geriatric Anxiety Scale. *Journal of Anxiety Disorders, 24*(7), 709–714. doi: 10.1016/j.janxdis.2010.05.002

Seppi, K., Ray Chaudhuri, K., Coelho, M. et al. (2019). Update on treatments for nonmotor symptoms of Parkinson's disease – an evidence-based medicine review. *Movement Disorders*, *34*(2), 180–198. doi: 10.1002/mds.27602

Shepard, M.D., Perepezko, K., Broen, M.P.G. et al. (2019). Suicide in Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *90*(7), 822–829. 10.1136/jnnp-2018-319815

Shin, S., Lee, J.E., Hong, J.Y. et al. (2012). Neuroanatomical substrates of visual hallucinations in patients with non-demented Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *83*(12), 1155–1161. doi: 10.1136/jnnp-2012-303391

Shine, J.M., Halliday, G.M., Naismith, S.L. & Lewis, S.J. (2011). Visual misperceptions and hallucinations in Parkinson's disease: dysfunction of attentional control networks? *Movement Disorders*, *26*(12), 2154–2159. doi: 10.1002/mds.23896

Shine, J.M., Keogh, R., O'Callaghan, C. et al. (2015). Imagine that: elevated sensory strength of mental imagery in individuals with Parkinson's disease and visual hallucinations. *Proceedings of the Royal Society B: Biological Sciences*, *282*(1798), 20142047. doi: 10.1098/rspb.2014.2047

Shine, J.M., Mills, J.M., Qiu, J. et al. (2015). Validation of the Psychosis and Hallucinations Questionnaire in non-demented patients with Parkinson's disease. *Movement Disorders Clinical Practice*, *2*(2), 175–181. doi: 10.1002/mdc3.12139

Shoji, Y., Nishio, Y., Baba, T. et al. (2014). Neural Substrates of Cognitive Subtypes in Parkinson's Disease: A 3-Year Longitudinal. *PLoS One, 9* (10), e110547. doi: 10.1371/journal.pone.0110547

Simonet, C., Bestwick, J., Jitlal, M. et al. (2022). Assessment of risk factors and early presentations of Parkinson Disease in primary care in a diverse UK population. *JAMA Neurology, 79*(4), 359–369. doi: 10.1001/jamaneurol.2022.0003

Simpson, J., Eccles, F. & Zarotti, N. (2021). *Psychological interventions for people with Huntington's disease, Parkinson's disease, motor neurone disease, and multiple sclerosis.* Leicester: British Psychological Society.

Simpson, J., Lekwuwa, G. & Crawford, T. (2013). Illness beliefs and psychological outcome in people with Parkinson's disease. *Chronic Illness*, *9*(2), 165–176. doi: 10.1177/1742395313478219

Simpson, J., Lekwuwa, G. & Crawford, T. (2014). Predictors of quality of life in people with Parkinson's disease: evidence for both domain specific and general relationships. *Disability and Rehabilitation*, *36*(23), 1964–1970. doi: 10.3109/09638288.2014.883442

Simpson, J., Zarotti, N., Varey, S. et al. (2022). 'It's a double whammy': A qualitative study of illness uncertainty in individuals with Parkinson's disease in the context of COVID-19. *Chronic Illness*, *18*(4), 860–873. doi: 10.1177/17423953211043101

Smeding, H.M., Speelman, J.D., Huizenga, H.M. et al. (2011). Predictors of cognitive and psychosocial outcome after STN DBS in Parkinson's Disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *82*(7), 754–760. doi: 10.1136/jnnp.2007.140012

Smith, A. (1973). *Symbol Digit Modalities Test.* Los Angeles, CA; Western Psychological Services.

Sockeel, P., Dujardin, K., Devos, D. et al. (2006). The Lille apathy rating scale (LARS), a new instrument for detecting and quantifying apathy: validation in Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry, 77*(5) 579–584. doi: 10.1136/jnnp.2005.075929

Southi, N., Honan, C.A., Hodges, J.R. et al. (2019). Reduced capacity for empathy in corticobasal syndrome and its impact on carer burden. *International Journal of Geriatric Psychiatry*, *34*(3), 497–503. doi: 10.1002/gps.5045

Spencer, K.A., Paul, J., Brown, K.A. et al. (2020). Cognitive rehabilitation for individuals with Parkinson's disease: Developing and piloting an external aids treatment program. *American Journal of Speech-Language Pathology*, *29*(1), 1–19. doi: 10.1044/2019_AJSLP-19-0078

Stanković, I., Krismer, F., Jesic, A. et al. (2014). Cognitive impairment in multiple system atrophy: a position statement by the Neuropsychology Task Force of the MDS Multiple System Atrophy (MODIMSA) study group. *Movement Disorders*, *29*(7), 857–867. doi: 10.1002/mds.25880

Stanković, I., Petrović, I., Pekmezović, T., et al. (2019). Longitudinal assessment of autonomic dysfunction in early Parkinson's disease. *Parkinsonism & Related Disorders*, *66*, 74–79. doi: 10.1016/j.parkreldis.2019.07.008

Stanton, A.L., Revenson, T.A. & Tennen, H. (2007). Health psychology: psychological adjustment to chronic disease. *Annual Review of Psychology*, *58*(1), 565–592. doi: 10.1146/annurev.psych.58.110405.085615

Starkstein, S.E. & Brockman, S. (2017). Management of depression in Parkinson's disease: a systematic review. *Movement Disorders Clinical Practice*, *4*(4), 470–477. doi: 10.1002/mdc3.12507

Starkstein, S.E., Mayberg, H.S., Leiguarda, R., et al. (1992). A prospective longitudinal study of depression, cognitive decline, and physical impairments in patients with Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *55*(5), 377–382. doi: 10.1136/jnnp.55.5.377

Stiasny-Kolster, K., Mayer, G., Schäfer, S. et al. (2007). The REM sleep behavior disorder screening questionnaire – a new diagnostic instrument. *Movement Disorders, 22*(16), 2386–2393. doi: 10.1002/mds.21740

Storch, A., Schneider, C.B., Wolz, M. et al. (2013). Nonmotor fluctuations in Parkinson disease: severity and correlation with motor complications. *Neurology*, *80*(9), 800–809. doi: 10.1212/WNL.0b013e318285c0ed

Strapasson, A.C.P., Antunes, Á.C.M., Oppitz, P.P. et al. (2019). Postoperative confusion in patients with Parkinson disease undergoing deep brain stimulation of the subthalamic nucleus. *World Neurosurgery*, *125*, e966–e971. doi: 10.1016/j.wneu.2019.01.216

Stroop, J.R. (1935). Studies of interference in serial verbal reaction. *Journal of Experimental Psychology, 18*(6), 643–662. doi: 10.1037/h0054651

Suzukamo, Y., Ohbu, S., Kondo, T. et al. (2006). Psychological adjustment has a greater effect on health-related quality of life than on severity of disease in Parkinson's disease. *Movement Disorders*, *21*(6), 761–766. doi: 10.1002/mds.20817

Svendsboe, E., Terum, T., Testad, I. et al. (2016). Caregiver burden in family carers of people with dementia with Lewy bodies and Alzheimer's disease. *International Journal of Geriatric Psychiatry, 31*(9), 1075–1083. doi: 10.1002/gps.4433

Tan, M.M., Lawton, M.A., Jabbari, E. et al. (2021). Genome-wide association studies of cognitive and motor progression in Parkinson's disease. *Movement Disorders*, *36*(2), 424–433. doi: 10.1002/mds.28342

Thiele, A. (2013). Muscarinic signaling in the brain. *Annual Review of Neuroscience*, *36*(1), 271–294. doi: 10.1146/annurev-neuro-062012-170433

Thobois, S., Ardouin, C., Lhommée, E. et al. (2010). Non-motor dopamine withdrawal syndrome after surgery for Parkinson's disease: predictors and underlying mesolimbic denervation. *Brain*, *133*(4), 1111–1127. doi: 10.1093/brain/awq032

Todd, D., Simpson, J. & Murray, C. (2010). An interpretative phenomenological analysis of delusions in people with Parkinson's disease. *Disability and Rehabilitation*, *32*(15), 1291–1299. doi: 10.3109/09638280903514705

Trenkwalder, C., Kohnen, R., Högl, B. et al. (2011). Parkinson's disease sleep scale – validation of the revised version PDSS-2. *Movement Disorders*, *26*(4), 644–652. doi: 10.1002/mds.23476

Troeung, L., Egan, S.J. & Gasson, N. (2013). A meta-analysis of randomised placebo-controlled treatment trials for depression and anxiety in Parkinson's disease. *PloS one*, *8*(11), e79510. doi: 10.1371/journal.pone.0079510

Troeung, L., Egan, S.J. & Gasson, N. (2014). A waitlist-controlled trial of group cognitive behavioural therapy for depression and anxiety in Parkinson's disease. *BMC Psychiatry*, *14*, 19. doi: 10.1186/1471-244X-14-19

Uekermann, J., Daum, I., Peters, S. et al. (2003). Depressed mood and executive dysfunction in early Parkinson's disease. *Acta Neurologica Scandinavica*, *107*(5), 341–348. doi: 10.1034/j.1600-0404.2003.02155.x

Valencia, D.Y., Gorovoy, S., Tubbs, A. et al. (2023). Sociocultural context of sleep health: Modeling change over time. *Sleep, 46*(1), zsac258. doi: 10.1093/sleep/zsac258

Vatter, S., McDonald, K.R., Stanmore, E. et al. (2018). A qualitative study of female caregiving spouses' experiences of intimate relationships as cognition declines in Parkinson's disease. *Age and Ageing*, *47*(4), 604–610. doi: 10.1093/ageing/afy049

Visser, M., Verbaan, D., van Rooden, S.M. et al. (2007). Assessment of psychiatric complications in Parkinson's disease: the SCOPA-PC. *Movement Disorders*, *22*(15), 2221–2228. doi: 10.1002/mds.21696

Vossius, C., Nilsen, O.B. & Larsen, J.P. (2010). Parkinson's disease and hospital admissions: frequencies, diagnoses and costs. *Acta Neurologica Scandinavica*, *121*(1), 38–43. doi: 10.1111/j.1600-0404.2009.01239.x

Wallace, E.R., Segerstrom, S.C., van Horne, C.G. et al. (2022). Meta-analysis of cognition in Parkinson's disease mild cognitive impairment and dementia progression. *Neuropsychology Review*, *32*(1), 149–160. doi: 10.1007/s11065-021-09502-7

Wang, J.W., Zhang, Y.Q., Zhang, X.H. et al. (2016). Cognitive and psychiatric effects of STN versus GPi deep brain stimulation in Parkinson's disease: a meta-analysis of randomized controlled trials. *PLoS One*, *11*(6), e0156721. doi: 10.1371/journal.pone.0156721

Warren, N., O'Gorman, C., Hume, Z. et al. (2018). Delusions in Parkinson's disease: a systematic review of published cases. *Neuropsychology Review*, *28*(3), 310–316. doi: 10.1007/s11065-018-9379-3

Warrington, E.K. & James, M. (1991). *Visual Object and Space Perception Battery.* Bury St Edmunds, UK: Thames Valley Test Company.

Wechsler, D. (2010). *Wechsler Adult Intelligence Scale UK – Fourth Edition.* San Antonio, TX; The Psychological Corporation.

Wechsler, D. (2011). *Test of Premorbid Function (TOPF).* San Antonio, TX; The Psychological Corporation.

Weil, R.S. & Reeves, S. (2020). Hallucinations in Parkinson's disease: new insights into mechanisms and treatments. *Advances in Clinical Neuroscience & Rehabilitation: ACNR*, *19*(4), ONNS5189. doi: 10.47795/ONNS5189

Weintraub, D., David, A.S., Evans, A.H. et al. (2015). Clinical spectrum of impulse control disorders in Parkinson's disease. *Movement Disorders*, *30*(2), 121–127. doi: 10.1002/mds.26016

Weintraub, D., Koester, J., Potenza, M.N. et al. (2010). Impulse control disorders in Parkinson disease: A cross-sectional study of 3090 patients. *Archives of Neurology, 67*(5), 589–595. doi: 10.1001/archneurol.2010.65

Weintraub, D., Mamikonyan, E., Papay, K., et al. (2012). Questionnaire for impulsive-compulsive disorders in Parkinson's Disease–Rating Scale. *Movement Disorders*, *27*(2), 242–247. doi: 10.1002/mds.24023

Weintraub, D., Morales, K.H., Duda, J.E. et al. (2006). Frequency and correlates of co-morbid psychosis and depression in Parkinson's disease. *Parkinsonism & Related Disorders*, *12*(7), 427–431. doi: 10.1016/j.parkreldis.2006.03.006

Weintraub, D., Morales, K.H., Moberg, P.J. et al. (2005). Antidepressant studies in Parkinson's disease: a review and meta-analysis. *Movement Disorders*, *20*(9), 1161–1169. doi: 10.1002/mds.20555

Weintraub, D. & Potenza, M.N. (2006). Impulse control disorders in Parkinson's disease. *Current Neurology and Neuroscience Reports*, *6*(4), 302–306. doi: 10.1007/s11910-006-0022-y

Wenning, G.K., Litvan, I., Jankovic, J. et al. (1998). Natural history and survival of 14 patients with corticobasal degeneration confirmed at postmortem examination. *Journal of Neurology, Neurosurgery & Psychiatry*, *64*(2), 184–189. doi: 10.1136/jnnp.64.2.184

Wieringa, G., Dale, M. & Eccles, F.J. (2022). Adjusting to living with Parkinson's disease; a meta-ethnography of qualitative research. *Disability and Rehabilitation*, *44*(23), 6949–6968. doi: 10.1080/09638288.2021.1981467

Williams, D.R. & Lees, A.J. (2005). Visual hallucinations in the diagnosis of idiopathic Parkinson's disease: a retrospective autopsy study. *The Lancet Neurology*, *4*(10), 605–610. doi: 10.1016/S1474-4422(05)70146-0

Williams-Gray, C.H., Evans, J.R., Goris, A. et al. (2009). The distinct cognitive syndromes of Parkinson's disease: 5 year follow-up of the CamPaIGN cohort. *Brain*, *132*(11), 2958–2969. doi: 10.1093/brain/awp245

Williams-Gray, C.H., Foltynie, T., Brayne, C.E.G. et al. (2007). Evolution of cognitive dysfunction in an incident Parkinson's disease cohort. *Brain*, *130*(7), 1787–1798. doi: 10.1093/brain/awm111

Willis, A.W., Schootman, M., Kung, N. et al. (2012). Predictors of survival in patients with Parkinson disease. *Archives of Neurology, 69*(5), 601–607. doi: 10.1001/archneurol.2011.2370

Wilson, B.A. & Betteridge, S. (2019). *Essentials of neuropsychological rehabilitation*. New York: The Guilford Press.

Wood, R.A., Hopkins, S.A., Moodley, K.K. & Chan, D. (2015). Fifty percent prevalence of extracampine hallucinations in Parkinson's disease patients. *Frontiers in Neurology*, *6*, 263. doi: 10.3389/fneur.2015.00263

Yamanishi, T., Tachibana, H., Oguru, M., et al. (2013). Anxiety and depression in patients with Parkinson's disease. *Internal Medicine*, *52*(5), 539–545. doi: 10.2169/internalmedicine.52.8617

Yao, N., Cheung, C., Pang, S., et al. (2016). Multimodal MRI of the hippocampus in Parkinson's disease with visual hallucinations. *Brain Structure and Function*, *221*, 287–300. doi: 10.1007/s00429-014-0907-5

Yao, N., Pang, S., Cheung, C., et al. (2015). Resting activity in visual and corticostriatal pathways in Parkinson's disease with hallucinations. *Parkinsonism & Related Disorders*, *21*(2), 131–137. doi: 10.1016/j.parkreldis.2014.11.020

Zahodne, L.B. & Fernandez, H.H. (2008). Course, prognosis, and management of psychosis in Parkinson's disease: are current treatments really effective? *CNS spectrums*, *13*(S4), 26–33. doi: 10.1017/S1092852900017296

Zampieri, M. & de Souza, E.A.P. (2011). Locus of control, depression, and quality of life in Parkinson's Disease. *Journal of Health Psychology, 16*(6), 980–987. doi: 10.1177/1359105310397220

Zarkali, A., Adams, R.A., Psarras, S. et al. (2019). Increased weighting on prior knowledge in Lewy body-associated visual hallucinations. *Brain Communications, 1*(1), fcz007. doi: 10.1093/braincomms/fcz007

Zarotti, N., Eccles, F.J., Foley, J.A. et al. (2021). Psychological interventions for people with Parkinson's disease in the early 2020s: Where do we stand? *Psychology and Psychotherapy: Theory, Research and Practice*, *94*(3), 760–797. doi: 10.1111/papt.12321

Zhang, C., Reeves, S., David, A.S. et al. (2023). Neuropsychiatric features of Parkinson's disease in the era prior to the use of dopaminergic therapies. *Cognitive Neuropsychiatry*, *28*(4), 243–252. doi: 10.1080/13546805.2023.2212151

Zhang, X., Chou, J., Liang, J. et al. (2019). Data-driven subtyping of Parkinson's disease using longitudinal clinical records: a cohort study. *Scientific reports*, *9*(1), 797. doi: 10.1038/s41598-018-37545-z

Zigmond, A.S. & Snaith, R.P. (1983). *Hospital Anxiety and Depression Scale (HADS). Acta Psychiatrica Scandinavica, 67*(6), 361–370. doi: 10.1111/j.0447.1983-1600.tb09716.x

Zimmermann, M., Chong, A.K., Vechiu, C. & Papa, A. (2020). Modifiable risk and protective factors for anxiety disorders among adults: A systematic review. *Psychiatry research*, *285*, 112705. doi: 10.1016/j.psychres.2019.112705

Zoon, T.J., van Rooijen, G., Balm, G.M. et al. (2021). Apathy induced by subthalamic nucleus deep brain stimulation in Parkinson's disease: a meta-analysis. *Movement Disorders*, *36*(2), 317–326. doi: 10.1002/mds.28390

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