

REVIEW ARTICLE

Updates on the sleep benefit phenomenon in Parkinson's disease: A narrative review

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Abstract

Parkinson's disease (PD) is the second most common neurodegenerative disorder globally, characterized by progressive motor and non-motor dysfunction due to multisystemic pathology. Although present pharmacological treatments offer symptomatic relief, they are often associated with adverse effects and do not halt disease progression, highlighting the need for complementary therapeutic strategies. An intriguing phenomenon termed "sleep benefit" has been reported in some PD patients, characterized by temporary improvements in motor and non-motor symptoms following sleep, either nocturnal or daytime naps. Sleep benefit may offer a lower risk profile compared to traditional pharmacotherapy; however, its underlying mechanisms and clinical significance remain poorly understood. In this review, we synthesize the existing literature on sleep benefit in PD, summarizing its clinical characteristics, prevalence, and associated factors. We also discuss distinctions between subjective and objective assessments and explore potential pathophysiological mechanisms underlying this phenomenon, aiming to provide new insights into non-pharmacological approaches for PD management.

Keywords: Parkinson's disease; Sleep benefit; Dopaminergic drugs; Motor symptoms; Potential mechanisms; Sleep disorder

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1. Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disorder worldwide, affecting over 6 million individuals in 2016, and is projected to impact approximately 25.2 million people globally by 2050.^{1,2} PD is primarily characterized by the progressive loss of dopaminergic neurons in the substantia nigra, resulting in cardinal motor symptoms, such as bradykinesia, rigidity, and resting tremor.³ However, its pathology extends beyond the nigrostriatal system, involving the accumulation and propagation of misfolded alpha-synuclein in non-dopaminergic brain regions as well.⁴ This multisystemic involvement gives rise to a broad spectrum of non-motor symptoms, including hyposmia, anxiety, depression, constipation, cognitive impairment, and sleep disturbances.

Among these, sleep disturbances are particularly prevalent and significantly impair the quality of life (QoL) of both patients and their caregivers.^{5,6} Up to 80% of PD patients report sleep-related problems within 5 years of diagnosis.⁷ Common nocturnal complaints include insomnia, excessive daytime sleepiness, rapid eye movement (REM) sleep behavior disorder, and restless legs syndrome.⁸⁻¹⁰ Interestingly, an intriguing phenomenon known as “sleep benefit” has been observed in some PD patients, where temporary improvements in both motor and non-motor symptoms occur following sleep.¹¹ This effect is notably seen after nocturnal sleep or even after brief daytime naps, with benefits comparable to the effects of pharmacological treatments.^{12,13}

The potential mechanisms underlying sleep benefit remain poorly understood, but exploring this phenomenon may open new avenues for non-pharmacological treatment strategies in PD management. This review aims to provide a comprehensive synthesis of the present literature on sleep benefit, summarizing its definition, epidemiology, methods of subjective and objective assessment, associated clinical determinants, and potential underlying mechanisms.

2. Definition and epidemiology of sleep benefit

The concept of “sleep benefit” was first introduced by Marsden *et al.* in 1980.¹⁴ They observed that some patients with PD experienced an improvement in motor function, particularly in flexibility, upon waking in the morning before taking their anti-PD medication.¹⁴ This improvement often resembled the “ON” state, where medication typically alleviates motor symptoms. It occurs before the administration of any medication during the

day, and in some cases, patients may even delay or reduce their morning dose of Parkinson's medication.^{11,15} Notably, subsequent research has found that PD patients also experience a temporary reduction in motor symptoms following short periods of sleep, whether during the day or at night.^{13,16} In 2013, van Gilst *et al.*¹⁷ reviewed existing definitions and formally characterized sleep benefit as follows: Sleep benefit is the experience of a temporary decrease in PD symptoms upon awakening after a period of sleep (night or daytime), before drug intake; the patient is feeling as good as “ON” (or better).

The reported prevalence of sleep benefit among patients with PD varies widely, ranging from 33% to 55%, with an average estimate of approximately 44%.¹⁷ Such variability likely reflects differences in study populations, assessment methods, and diagnostic criteria (Table 1). While sleep benefit is most commonly observed following nocturnal sleep, a substantial subset also experiences improvement after daytime naps. Studies have identified distinct phenotypic subgroups: Approximately 21% of patients experience benefit exclusively after nocturnal sleep, 13–21% exclusively after naps, and a significant portion benefit from both sleep periods.^{13,16} The prevalence of nap-related sleep benefit varies considerably depending on study protocols. Controlled trials using standardized 30–90 min afternoon naps have reported prevalence rates of 35–44%,^{18,19} whereas studies relying on spontaneous naps assessed by actigraphy or patient self-reports have found lower rates of 15–34%.^{11,13,20} These protocol differences suggest that structured nap timing and duration may influence the detection and expression of sleep benefit.

The duration of sleep benefit has been assessed using heterogeneous approaches, ranging from subjective

Table 1. Demographic and clinical characteristics of studies on sleep benefit in Parkinson's disease

Study (year)	Sample size (PD)	SB prevalence (%)	Age*, mean±SD (years)	Disease duration*, mean±SD (years)	Male* (%)	LEDD* (mg/day)	SB after naps (%)	Mean SB duration (min)	SB assessment method
Merello <i>et al.</i> (1997) ¹¹	312	55.1	69.6±9.0	8.8±7.1	66	NR	24.6	85.4±76.5	3-item questionnaire
Tandberg <i>et al.</i> (1999) ¹²	239	42.4	73.5±8.4	9.7±6.2	54	489.6±260.9	NR	NR	Yes/no question
van Gilst <i>et al.</i> (2012) ¹³	243	46.9	64.8±10.0	8.1±5.7	57.9	512±407	33.7	NR	Yes/no question
Lee <i>et al.</i> (2017) ¹⁸	92	21.7	65.6±7.9	9.3±3.7	55	1008±480	35	≥60	SBI+smartphone tests
van Gilst <i>et al.</i> (2015) ¹⁹	38	47.4	61.0±5.9	7.7±4.3	35	777±384	44.4	NR	Pegboard+finger-tapping
Hoggl <i>et al.</i> (1998) ²¹	20	50	58.2±7.9	8.3±4.1	60	628±264	NR	≥60	Questionnaire+UPDRS improvement
Currie <i>et al.</i> (1997) ²⁸	162	32.7	65.5±10.4	10.2±5.3	66	779±417	NR	NR	Subjective “ON” feeling at awakening
Sherif <i>et al.</i> (2014) ²⁰	173	30	69±9	13±6	59	698±474	15	NR	Yes/No question

Notes: *Values for age, disease duration, and sex distribution refer to the entire PD cohort, unless otherwise specified.

Abbreviations: ADL: Activities of daily living; LEDD: Levodopa equivalent daily dose; NR: Not reported; PD: Parkinson's disease; SB: Sleep benefit; SBI: Sleep benefit index; UPDRS: Unified Parkinson's Disease Rating Scale.

patient diaries to structured questionnaires combined with motor testing.^{11,18,21} Reported durations vary substantially across studies, with Merello *et al.*¹¹ reporting a mean of approximately 85 min, while Lee *et al.*¹⁸ and Hogg *et al.*²¹ reported mean durations of at least 60 min. This variability further supports the notion that sleep benefit may encompass distinct subtypes, highlighting the need for standardized measurement and classification.

3. Subjective and objective assessment of sleep benefit

Although the present concept of sleep benefit is predominantly derived from patients' subjective reports, accumulating evidence suggests that subjective perceptions may not always align with objectively measured motor function.^{18,19,21} Therefore, distinguishing between subjective and objective aspects of sleep benefit is crucial for a comprehensive understanding and future standardization.

3.1. Subjective sleep benefit

Subjective sleep benefit refers to the self-perceived improvement of symptoms following sleep, most commonly assessed through interviews, structured questionnaires, or patient diaries. Early studies identified sleep benefit based on patients' affirmative responses to structured questions regarding their morning motor state, including whether they felt "as if under medication" upon waking, experienced reduced tremor or gait difficulty, and perceived a general improvement in Parkinson's symptoms following sleep.¹¹

To more comprehensively assess the phenomenon of sleep benefit, van Gilst *et al.*²² developed a self-rating questionnaire in 2015 consisting of 12 items. The first 11 are structured questions addressing the timing, degree, duration, frequency, and pharmacological impact of sleep benefit, while the final item is an open-ended prompt allowing patients to describe their experience in their own words. Although this tool has not undergone formal validation, it represents the first comprehensive instrument specifically designed for research on sleep benefit in PD. In 2017, Lee *et al.*¹⁸ introduced a more quantitative assessment tool—the Sleep Benefit Index (SBI)—which assigns a total score out of 110 points. The SBI comprises two sections: Part A evaluates sleep-related changes in ten motor and non-motor symptoms of PD, while Part B uses a five-level Likert scale to quantify the frequency and extent of perceived benefit, including its impact on medication timing. A higher score reflects a greater degree of sleep benefit.¹⁸ Despite its structured design, the SBI relies entirely on patient self-report, without incorporating polysomnographic data or direct clinical

testing, which limits its ability to objectively confirm motor improvements.

3.2. Objective sleep benefit

Objective sleep benefit refers to measurable improvements in motor function following sleep, assessed using standardized clinical tools. While subjective reports remain central to most studies, objective evaluation is essential to reduce potential bias and false positives inherent in self-reported outcomes. Early investigations employed the Unified Parkinson's Disease Rating Scale (UPDRS) Part III to assess motor function before and after sleep, both during nocturnal and daytime episodes.²¹ However, this method has limitations, particularly for patients with transient improvements that may dissipate before assessment, and the use of clinician-rated scales introduces inter-rater variability.

In 2015, van Gilst *et al.*¹⁹ employed pegboard and finger tapping tasks to objectively evaluate motor performance in patients with PD, both before and after nighttime sleep and daytime naps. These tasks were designed to quantify fine motor dexterity and capture subtle changes potentially associated with sleep benefit. In 2017, Lee *et al.*²³ developed a validated smartphone-based application incorporating four brief motor assessments, including the timed tapping test, rapid alternating movements, tremor tracking, and cognitive interference test. Among these tools, the keyboard tapping test has emerged as one of the most widely used and validated methods for detecting short-term motor changes. More recently, an online version of the tapping test has been developed,^{24,25} offering a convenient, location-independent, and scalable approach to objectively assess motor fluctuations and potential sleep-related improvements.

Despite these advances, objective assessments of sleep benefit remain scarce, and standardized criteria for the magnitude and duration of symptom improvement have yet to be defined. Present subjective definitions—such as "feeling as good as on (or better)"—may lack sensitivity to detect milder but clinically relevant effects. Future research should aim to establish quantitative thresholds and minimal clinically important differences, as well as incorporate polysomnography (PSG) and direct motor testing to strengthen validity.

3.3. Relationship between subjective and objective sleep benefit

Although various subjective and objective methods have been developed to assess sleep benefit in PD, studies consistently demonstrate a mismatch between patients' perceived symptom improvement and objectively

measured motor changes. Hogg *et al.*²¹ observed that patients reporting subjective sleep benefit exhibited greater reductions in UPDRS-III scores after sleep compared to those without perceived benefit, suggesting a potential association. However, inconsistencies were also noted, with some patients describing significant morning improvement despite remaining objectively in an “OFF” state. Similarly, van Gilst *et al.*¹⁹ found no significant differences in fine motor performance, assessed through pegboard and tapping tasks, between patients who reported subjective sleep benefit and those who did not, indicating that the perceived benefit may not correspond to measurable motor improvement. Lee *et al.*²³ further confirmed this discrepancy using smartphone-based motor assessments, demonstrating that while 77% of patients reported subjective benefit, only 62% showed objective improvement, and just 22% achieved motor function comparable to or exceeding their “ON” state.

These findings collectively highlight that sleep benefit, although objectively verifiable in a subset of patients, predominantly remains a subjective experience in many cases. The underlying mechanisms contributing to this discrepancy are poorly understood and warrant further investigation.

4. Determinants of sleep benefit in PD

The phenomenon of sleep benefit, characterized by motor symptom alleviation without pharmacological intervention, offers an important window into the pathophysiology of PD. Understanding the determinants of sleep benefit may yield insights into disease mechanisms and therapeutic opportunities. Although many studies have investigated the clinical factors associated with sleep benefit, results have often been inconsistent.

A meta-analysis identified three factors consistently associated with sleep benefit: Longer disease duration, higher UPDRS-III motor scores, and lower sleep efficiency.²⁶ These findings collectively indicate that sleep benefit is more commonly observed in patients with more advanced disease and poorer sleep quality. However, substantial variability across individual studies highlights the complexity of this phenomenon. The following sections will examine the major factors that may contribute to the occurrence of sleep benefit in PD.

4.1. Sleep and sleep benefit

One central question is whether motor improvement after sleep is directly attributable to sleep itself. Most early studies assessed sleep subjectively, primarily through the Pittsburgh Sleep Quality Index.^{13,22,23} Overall, no consistent association was found between sleep benefit and subjective

reports of sleep time or sleep efficiency, although one study reported longer sleep duration among patients experiencing sleep benefit compared to non-benefit patients.²⁷

However, subjective sleep assessments are susceptible to recall bias and reporting errors. To date, only three studies have objectively evaluated sleep in patients with sleep benefit using PSG. The earliest study found no significant differences in sleep architecture, although patients with sleep benefit tended to have more nocturnal awakenings.²¹ In 2014, Sherif *et al.* observed that patients with sleep benefit had shorter total sleep time and longer sleep latency than those without, findings that were counterintuitive.²⁰ In 2015, van Gilst *et al.*¹⁹ assessed sleep in 38 patients with sleep benefit and reported no significant differences in any PSG variables between benefit and non-benefit groups. Taken together, present evidence does not support the existence of a distinct sleep architecture among patients experiencing sleep benefit.

4.2. Disease characteristics and sleep benefit

Several studies have examined the association between clinical characteristics of PD and the occurrence of sleep benefit. Earlier reports suggested that patients with sleep benefit tend to have a younger age at disease onset, a longer disease course, and greater motor severity.^{11,15,28} However, other studies have failed to replicate these findings.^{13,29} A study conducted in China further suggested that the tremor-dominant PD subtype may be more prevalent among patients reporting sleep benefit.²⁷

4.3. Dopaminergic medication and sleep benefit

Several studies have explored the association between dopaminergic therapy and the occurrence of sleep benefit. Early reports suggested that patients with sleep benefit tend to have a longer duration of dopaminergic medication use and higher levodopa equivalent daily doses (LEDD) compared to those without sleep benefit.^{12,28} However, subsequent studies failed to replicate these findings, reporting no significant differences in treatment duration or LEDD between benefit and non-benefit groups.^{13,29} Adding further complexity, a study by Du *et al.*²⁷ reported that patients with sleep benefit actually exhibited lower total LEDD, as well as reduced doses of levodopa and dopamine receptor agonists.

In an attempt to elucidate pharmacokinetic differences, one study conducted continuous monitoring of plasma levodopa concentrations in patients with and without sleep benefit.²¹ No significant differences in morning levodopa levels were observed between the two groups. However, patients with sleep benefit displayed a greater severity

of “OFF” symptoms following the drug-induced “ON” state, suggesting potential differences in dopaminergic responsiveness. It should be noted that the small sample size (only 10 participants) limits the generalizability of these findings. Consistent with the notion of altered dopaminergic sensitivity, Merello *et al.*¹¹ reported that the motor response to morning administration of dopamine agonists was weaker in patients with sleep benefit compared to those without. These observations further support the hypothesis that altered dopaminergic dynamics, rather than absolute drug levels, may contribute to the sleep benefit phenomenon.

4.4. Genetic factors and sleep benefit

Genetic factors may also contribute to the phenomenon of sleep benefit. Before the formal definition of sleep benefit, a clinical phenomenon characterized by milder motor symptoms in the morning and worsening symptoms at night was observed in patients with early-onset Parkinsonism. This was described as early-onset Parkinsonism with diurnal fluctuations (EPDF) in 1973.³⁰ Following the discovery of the parkin gene, EPDF was reclassified as PARK2-related young-onset PD, and sleep benefit emerged as one of its characteristic clinical features.³¹ Subsequent studies have reported that sleep benefit is highly prevalent in familial juvenile Parkinsonism associated with *PARK2* mutations, with estimated rates ranging from 50% to 95%.^{32–35}

In addition, sleep benefit has been reported in patients with *PARK6* mutations and in other movement disorders, such as episodic ataxias and hereditary progressive dystonia.^{35–37} For instance, a case of episodic ataxias type 2 associated with a *CACNA1A* mutation exhibited a sleep benefit phenomenon, and similar findings have been reported in hereditary progressive dystonia due to *GCH1* mutations.^{38–40} These observations suggest that sleep benefit may be linked to specific genetic mutations,

although the underlying molecular mechanisms remain unclear. Further research is needed to identify gene loci associated with sleep benefit and elucidate their potential pathophysiological roles.

The phenomenon of sleep benefit in PD appears to be influenced by a complex interplay of factors, including sleep-related characteristics, disease-specific clinical features, dopaminergic medication effects, and genetic predispositions (Figure 1). Despite substantial research, findings across studies remain heterogeneous and at times contradictory, highlighting the need for further systematic investigation. A deeper understanding of the mechanisms underlying sleep benefit could not only provide critical insights into PD pathophysiology but also inform the development of novel therapeutic strategies.

5. The potential mechanisms of sleep benefit

Although research on the mechanisms underlying sleep benefit remains limited, several main hypotheses have been proposed to explain this phenomenon.

5.1. Sleep mechanisms and circadian rhythm

The majority of present studies suggest that the sleep benefit may be partially attributed to the direct effects of sleep itself. It has been proposed that dopamine storage in nigral neuronal terminals increases during sleep, leading to improved motor symptoms upon awakening.^{11,12,15} This hypothesis is supported by the observation that sleep benefit can occur after both nighttime sleep and daytime naps. The PHASE study (2020)⁴¹ employed actigraphy to monitor 157 PD patients continuously over six days and quantitatively analyzed the relationship between objective sleep parameters and morning motor performance. Results demonstrated that higher sleep quality was significantly correlated with better morning flexibility.

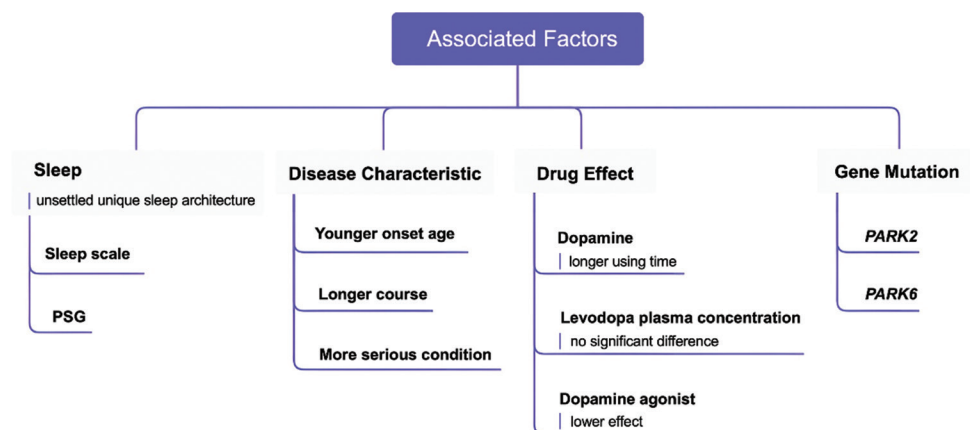


Figure 1. Factors associated with the sleep benefit in Parkinson's disease.

Abbreviation: PSG: Polysomnography.

However, paradoxical findings were also reported, as a subset of patients exhibited symptom improvement only after daytime naps rather than after prolonged nighttime sleep. The mechanisms underlying these discrepancies remain unclear and warrant further investigation.

Despite several studies evaluating sleep measures and architecture in patients with sleep benefit, no consistent differences have been identified between those with and without sleep benefit.^{19,21} These inconsistencies may be attributable, at least in part, to the limited sample sizes included in these studies. Hogl *et al.*²¹ further reported that patients with sleep benefit exhibited a tendency toward more nocturnal awakenings and reduced REM sleep duration. They speculated that sleep deprivation might paradoxically contribute to the emergence of sleep benefit; however, due to small sample sizes and the lack of corroborating evidence, this hypothesis requires further validation in larger cohorts.

In addition to sleep quality, circadian rhythm disturbances may also contribute to the phenomenon of sleep benefit. A subset of PD patients exhibit disorders, such as excessive daytime sleepiness and sleep apnea syndrome, both indicative of circadian rhythm dysregulation.⁴² Animal studies have demonstrated that circadian disruption can promote protein misfolding, impair proteostasis, and inhibit the clearance of metabolic waste products during sleep.^{43–46} Mounting evidence suggests a bidirectional relationship between circadian rhythm disturbances and neurodegenerative diseases, wherein disease progression exacerbates circadian dysfunction, and circadian disruption, in turn, accelerates neurodegeneration.^{47–49}

Given that patients with sleep benefit often exhibit longer disease duration and greater severity,^{11,15,28} it is hypothesized that sleep benefit may reflect a clinical manifestation of underlying circadian dysregulation. Nevertheless, evidence remains limited. To date, only one small-scale study has used the Morningness-Eveningness Questionnaire to examine the relationship between circadian preference and sleep benefit.²¹ No significant association was found between chronotype and the occurrence of sleep benefit, highlighting the need for further research utilizing more objective circadian biomarkers.

5.2. Symptom fluctuation hypothesis

Some studies have suggested that sleep benefit may represent a form of motor symptom fluctuation rather than a direct effect of sleep. Because sleep benefit typically occurs upon morning awakening, it has been proposed that it merely reflects diurnal motor fluctuations—improvement in the morning and worsening by evening.²⁹ Consequently, some have referred to sleep benefit as

“morning benefit.” However, other findings challenge this interpretation. Sleep benefit has also been reported in newly diagnosed PD patients without established motor fluctuations, whereas some patients with significant motor fluctuations do not experience sleep benefit.^{11,12} These contradictory observations suggest that sleep benefit cannot be fully explained as a manifestation of motor symptom fluctuation. Therefore, the symptom fluctuation hypothesis remains controversial and requires further investigation to clarify the relationship between sleep benefit and PD motor fluctuations.

5.3. Dopaminergic drug action hypothesis

Prior studies have explored the relationship between dopaminergic therapies and sleep benefit in PD. Some findings suggest that patients treated with levodopa or dopamine agonists are more likely to experience sleep benefit,²⁷ leading to the speculation that this phenomenon may be directly related to the pharmacological effects of dopaminergic drugs or higher morning plasma levodopa concentrations in affected individuals. However, studies involving continuous monitoring of plasma dopamine levels have not identified significant differences in morning dopamine concentrations between patients with and without sleep benefit.²¹ This suggests that mechanisms beyond simple plasma pharmacokinetics may be involved.

Levodopa's therapeutic effects are broadly categorized into a short-duration response (SDR), lasting approximately 1–5 h, and a long-duration response (LDR), which persists for days to weeks independent of plasma drug levels.^{50–52} The LDR is estimated to account for one-third to one-half of the total motor benefit conferred by levodopa.⁵¹ Given that nighttime sleep typically spans 6–8 h, it has been proposed that the persistence of LDR, despite overnight SDR washout, might contribute to the manifestation of sleep benefit. Variability in LDR expression among patients could partly explain why the phenomenon is not associated with morning dopamine plasma levels.

A clinical study found that patients exhibiting sleep benefit had lower LEDD, suggesting a potential association with preserved endogenous dopamine reserves.²⁷ Similarly, an animal study demonstrated impaired synaptic receptor regulation in models of dopaminergic dysfunction,⁵³ further supporting the notion that the sleep benefit phenomenon may reflect relatively intact dopaminergic system function. Notably, emerging evidence suggests that different antiparkinsonian medications may variably influence sleep architecture in patients with PD.⁵⁴ Nevertheless, the observation that sleep benefit occurs in patients with lower dopaminergic medication doses and is not consistently associated with higher morning

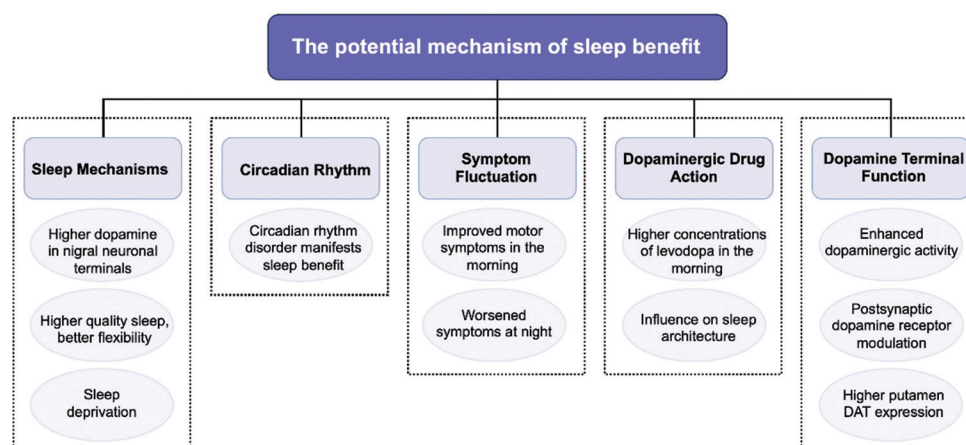


Figure 2. Proposed mechanisms underlying the sleep benefit in Parkinson's disease.
Abbreviation: DAT: Dopamine transporter.

plasma drug levels suggests that it is unlikely to be merely a pharmacological confounder. Further studies are warranted to elucidate the mechanisms through which sleep, medication, and dopaminergic system integrity interact to produce this phenomenon.

5.4. Dopamine terminal function hypothesis

Several researchers have hypothesized that the sleep benefit in PD may be related to alterations in presynaptic and postsynaptic dopaminergic function. One theory proposes that increased presynaptic dopamine release during sleep enhances dopaminergic transmission upon awakening, thereby contributing to sleep benefit.^{11,21} Alternatively, based on animal models, Galati *et al.*⁵³ suggested that sleep benefit might be mediated by modulation of post-synaptic dopamine receptor function, particularly influenced by slow-wave sleep activity.

Dopamine transporter (DAT) function is critical for regulating extracellular dopamine levels and maintaining intracellular dopamine storage capacity.⁵⁵ Recent work by Wang *et al.*⁵⁶ explored this potential link by comparing clinical characteristics, sleep assessments, and striatal 11C-CFT uptake (an indicator of DAT binding) between PD patients with and without sleep benefit. Their analysis demonstrated that striatal 11C-CFT uptake was significantly higher in patients exhibiting sleep benefit, even after adjusting for key clinical confounders. These findings support the hypothesis that elevated putamen DAT expression may be associated with, and potentially predictive of, the occurrence of sleep benefit in PD.

Several mainstream hypotheses regarding the mechanisms underlying the sleep benefit have been discussed (Figure 2). Notably, a subset of patients report early-morning worsening, known as morning akinesia,

likely reflecting pharmacokinetic limitations rather than sleep-related improvement.⁵⁷ This coexistence of contrasting motor states upon awakening highlights the heterogeneity of PD and suggests that sleep benefit involves distinct, possibly multifactorial, mechanisms.

6. Future directions

Sleep benefit remains an incompletely understood and inconsistently defined phenomenon in PD. To advance this field, future studies should prioritize: (i) Standardized objective assessments integrating smartphone-based motor tests and PSG; (ii) large-scale longitudinal designs to track its trajectory over disease progression; and (iii) mechanistic studies linking DAT expression, dopamine terminal function, and sleep architecture through neuroimaging and molecular techniques.

From a clinical perspective, routine prescription of scheduled naps is not currently supported. However, for patients with demonstrated objective sleep benefit, a structured, tiered approach may be considered: Initial screening with motor diaries and tapping tasks, followed by guided short naps embedded within broader sleep hygiene interventions. Importantly, sleep benefit may serve as a marker of residual dopaminergic function, potentially informing future personalized treatment strategies.

7. Conclusion

Sleep benefit, characterized by temporary improvements in motor and non-motor symptoms following sleep, represents a unique and intriguing phenomenon in PD. Recent literature suggests that sleep benefit is objectively existent in a subset of patients; however, its underlying mechanisms remain unclear. Present evidence points to a complex interplay of factors, including sleep architecture,

disease characteristics, dopaminergic dynamics, and genetic predispositions. Further research is warranted to clarify these mechanisms, define standardized assessment criteria, and explore the therapeutic potential of sleep benefit as a non-pharmacological adjunct in PD management. A deeper understanding of this phenomenon may ultimately contribute to the development of novel strategies aimed at improving patient outcomes and QoL.

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Conflict of interest

Chun-Feng Liu serves as an Associate Editor for this journal, but was not in any way involved in the editorial and peer-review process conducted for this paper, directly or indirectly. Separately, other authors declared that they have no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

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Consent for publication

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Availability of data

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