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Cognitive processes and neurophysiological mechanisms of time processing in Parkinson's disease: A narrative review

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ABSTRACT

Parkinson's disease (PD) is defined by the presence of motor symptoms, such as bradykinesia. However, it also involves less well-understood cognitive deficits, including impairments in time processing: the ability to perceive, estimate, and produce time intervals accurately. In this review, we summarize the existing literature on time processing in PD, with an emphasis on the different tasks used to study it, as well as on the cognitive processes and neurophysiological mechanisms contributing to time processing deficits in PD.

Findings: show that temporal processing deficits in PD span both motor and cognitive/perceptual domains. While dopamine replacement therapy can improve motor timing, its effects on cognitively controlled tasks—particularly those requiring attention and longer intervals—are limited. These deficits reflect dysfunction across basal ganglia, prefrontal, cerebellar, and other brain circuits, and may involve additional neurotransmitter systems such as acetylcholine, serotonin, and noradrenalin. The variety of experimental tasks used to study timing reveals the need for more precise assessments that clearly separate motor and cognitive components. Furthermore, different cognitive processes, such as explicit, implicit, sub-second, and supra-second timing as well as attention and working memory, are involved in time processing in PD. Temporal dysfunction in PD is multidimensional, resulting from a complex system of interacting neural processes. A more complete understanding of time processing in PD is needed, focusing on exploration of the non-dopaminergic aspects of time processing, and improving the design of timing tasks to better identify specific deficits and treatment targets.

1. Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder that predominantly affects motor function. Among its primary motor symptoms, bradykinesia – characterized by marked slowness in initiating and executing movements – is often the most disabling (Poewe et al., 2017; Berardelli et al., 2001). This phenomenon primarily reflects impairment in motor control, but also cognitive control, including deficits in time processing: the ability to perceive, estimate, and produce time intervals (Jones et al., 2008; Pastor et al., 1992a):a less understood aspect of PD that is nonetheless noteworthy. Cognitive functions such as attention, working memory, and executive control are fundamental to accurate time processing, and impairments in these domains in PD can negatively impact daily life (Kudlicka et al., 2011).

Understanding the cognitive processes and neurophysiological mechanisms that underlie time processing impairment in PD is important for several reasons. First, it can clarify the broader cognitive deficits affecting individuals with PD (Kudlicka et al., 2011). Second, it may help identify the neurophysiological systems involved in time processing in general and in PD specifically (Bernardinis et al., 2019). Third, as time processing is closely related to cognitive and motor functions, providing information on its mechanisms can improve our understanding of bradykinesia and bradyphrenia (slowness of thought and information processing), which are hallmark symptoms of PD. Ultimately, it is possible to adequately evaluate the various experimental time processing tasks employed in this research area, assessing their quality, usability, and practicality to enhance research consistency and reliability. Evaluating the effectiveness and frequency of these tasks is a critical step in

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determining which are the most reliable and informative for future studies.

Previous research has made progress in awareness and knowledge of time processing in PD. The mechanisms behind time processing are complex and involve interactions between cognitive functions, neurophysiological systems, and neurotransmitters, possibly extending beyond dopamine (Bohnen and Albin, 2011). In PD, the degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to a dopamine deficit in the striatum, which disrupts the normal functioning of these timing-related circuits, is its major neurophysiological characteristic (Jones et al., 2008; Dauer and Przedborski, 2003). This neurochemical imbalance impairs the facilitatory effect of dopamine on the direct pathway neurons and triggers the inhibitory output of basal ganglia nuclei to the thalamus, ultimately reducing the brain’s ability to initiate and execute voluntary movements and affecting processing (Bohnen and Albin, 2011; Dauer and Przedborski, 2003; Coull and Nobre, 2008; Maniadakis and Trahanias, 2014a; Rocha et al., 2023; DeLong, 1990). In addition to dopamine, several other neurotransmitter systems contribute to the pathophysiology of PD. Cholinergic dysfunction has been strongly associated with attentional and cognitive deficits. Serotonergic abnormalities, including reduced serotonin concentrations in the basal ganglia and cortex, are linked not only to depression and sleep disturbances but also to modulation of basal ganglia circuits, thereby influencing dopamine release and perhaps timing-related functions. Similarly, noradrenergic deficits contribute to impaired arousal, attention, and other executive functions, and have been shown to exacerbate motor symptoms and non-motor features such as depression and autonomic dysfunction (Barone, 2010).

This review aims to assemble and summarize the existing literature on time processing in PD, laying the groundwork for understanding the cognitive processes and neurophysiological mechanisms contributing to time processing deficits in PD. It also focuses on the role of neurotransmitters, with a particular focus on dopamine, and evaluates experimental tasks used to objectively assess time processing deficits in PD.

2. Methodology

This review was completed following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Peer-reviewed, English-language studies were included if they examined cognitive and neurophysiological aspects of temporal/time processing in PD. The search strategy combined terms related to time processing with terms such as attention, memory, executive function, neurophysiological mechanisms, neurotransmitter systems, bradyphrenia, and bradykinesia. Eligible study designs included original research articles, case-control studies, clinical trials, systematic reviews, and meta-analyses. Excluded were dissertations, editorials, book reviews, conference abstracts, and non-peer-reviewed papers. A detailed description of the search strategy and terms used to find and select the relevant papers necessary for this review are given bellow and in the Supplementary material.

2.1. Information sources and search strategy

A PubMed search (1989 - July 2024) was performed using combinations of keywords including: ‘temporal processing’, time processing’, ‘time perception’, ‘Parkinson’s disease’, ‘cognitive processes’, ‘attention’, ‘memory’, neurophysiological mechanisms’, ‘basal ganglia’, ‘neural circuits’, ‘brain connectivity’, ‘neurotransmitters’, ‘dopamine’, ‘acetylcholine’, ‘bradykinesia’, and ‘bradyphrenia’. Boolean operators (AND/OR) refined the search, and no restrictions were imposed on publication dates. The search was structured in phases, initially focusing on neuroanatomical and neurophysiological correlates, and then expanding to cognitive domains and experimental time-processing tasks.

Title and abstract screening, full-text assessment, and data extraction were performed by one co-author (LB) and checked for accuracy by another (DG). A total of 3203 articles were retrieved. After removing 1663 duplicates, 1540 unique articles remained for screening based on title and abstract. Full texts of 106 articles were assessed in detail, resulting in the inclusion of 42 studies. An additional 20 articles were included through citation searching and suggestions from co-authors, bringing the total to 62 included studies (Fig. 1).

2.2. Study selection and data extraction

Studies were included if they involved human participants with PD at any disease stage; investigated time processing regarding attention, working memory, executive function, bradyphrenia, or bradykinesia; examined neurophysiological mechanisms (e.g., fMRI, PET, EEG) or neurotransmitter involvement (e.g., dopamine, acetylcholine); used experimental tasks to assess timing; provided sufficient methodological detail or novel data/perspective; and were published in peer-reviewed journals. Studies were excluded if they did not involve participants with PD, did not address time processing or related cognitive mechanisms, focused on unrelated neurological disorders, or lacked sufficient relevance to the aims of this review. As this study was designed as a narrative review rather than a systematic review or meta-analysis, no formal inter-rater reliability analysis or standardized risk-of-bias assessment tool, such as the Cochrane Risk of Bias Tool, was applied. Data extraction focused on the study’s objective, design, methods, and key findings related to time processing mechanisms in PD. Because the aim of this review was qualitative synthesis, no meta-analytic statistical methods or effect size calculations were performed.

3. Results

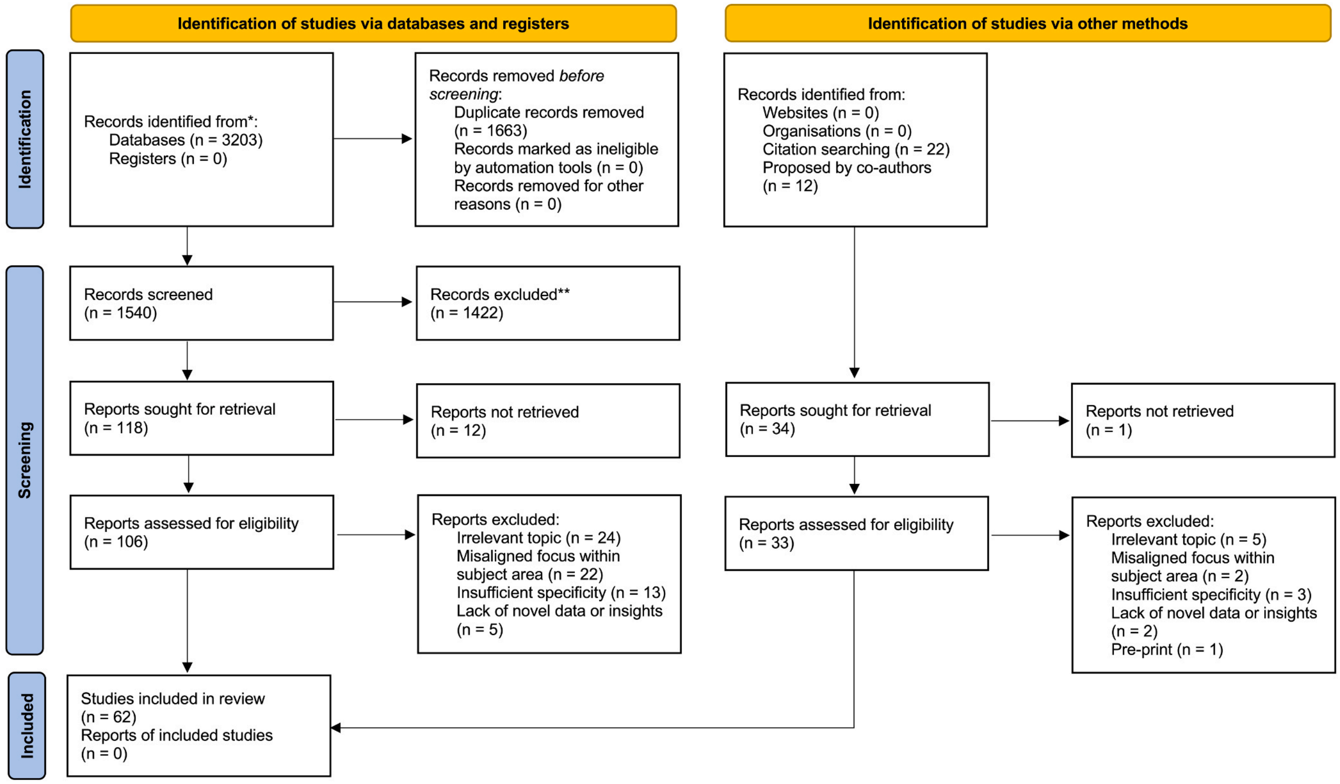
Impairments in time processing in PD reflect the involvement of cognitive, motor, and neurophysiological domains, engaging brain areas such as the prefrontal cortex, basal ganglia, and cerebellum. To provide context for the findings, this section begins with an overview of the experimental tasks commonly employed to investigate time processing in PD (Table 1). The articles included in this review, with the main findings, are listed in Table 2.

3.1. Experimental tasks for assessing temporal cognition in PD

Experimental tasks commonly used to study time processing in PD originate from broader cognitive neuroscience but have become central tools for exploring how PD alters temporal cognition. These tasks probe perceptual, motor, and executive components of timing and together illustrate how different cognitive and neural systems contribute to performance. Research in this area draws on a diverse set of tasks, each designed to isolate specific timing processes. Collectively, they examine how PD affects both the perception of time and the ability to synchronize, produce, or estimate durations. Fig. 2 summarizes the seven most frequently used tasks identified in the reviewed studies.

Among these, the *time reproduction task* is the most widely used. Participants are exposed to a stimulus for a specific duration (e.g., a tone or visual cue) and then asked to reproduce the same interval by pressing or tapping a button. Intervals can range from milliseconds to several seconds. Some protocols introduce aids such as silent counting or feedback to improve precision. Time reproduction tasks are particularly suited to studying how dopamine influences interval timing, since performance can be compared across medicated and unmedicated states. However, individual strategies like covert counting can add variability between participants (Jones et al., 2008; Pastor et al., 1992a; Terao et al., 2021; Fang et al., 2020; Torta et al., 2010; Koch et al., 2009, 2008; Honma et al., 2016; Malapani et al., 1998; Dušek et al., 2012).

A related method is the *duration production task*, in which participants actively generate specified intervals, often by marking the start



*Consider, if feasible to do so, reporting the number of records identified from each database or register searched (rather than the total number across all databases/registers).
**If automation tools were used, indicate how many records were excluded by a human and how many were excluded by automation tools.

Fig. 1. PRISMA Flow Diagram. The diagram shows the literature screening and selection process of the articles used in this review.

and end of a target duration through a keypress (e.g., 2, 5, or 10 s). Variations may include continuous tapping or silent counting for longer durations. This task helps to assess internal timing by evaluating the accuracy of interval generation. Findings from PD cohorts consistently show that the disease disrupts interval production, although dopaminergic medication can sometimes normalize performance (Jones et al., 2008; Terao et al., 2021; Honma et al., 2016; Perbal et al., 2005).

Reaction time tasks, including *simple* (SRT) and *choice* (CRT) versions, remain a cornerstone for studying temporal aspects of motor and cognitive slowing. SRT tasks are used to assess processing speed in PD. In these tasks, participants respond as quickly as possible to a stimulus (e.g., a light or tone), often following a warning cue. By varying the interval between the warning and the target stimulus, researchers can explore the effects of temporal expectation on response speed (Jahanshahi et al., 1992). CRT tasks add a further layer of complexity: rather than making the same response every time, participants must first identify the stimulus, then select and execute the corresponding response. Because CRT tasks involve additional stages such as stimulus identification, response selection, and programming, comparing performance on SRT and CRT can help to separate motor initiation from higher-order cognitive processes (Jahanshahi et al., 1992).

While reaction time tasks are often used to infer motor (bradykinesia) and cognitive (bradyphrenia) slowing, it is essential to note that reaction time reflects a combination of perceptual processing, cognitive decision-making, and motor execution. The specific contribution of each component can vary depending on the response modality (e.g., manual, verbal) and task design, making it challenging to dissociate motor and cognitive effects in PD. To further examine these processes, some tasks incorporate temporal cues (to anticipate stimulus timing) or movement-parameter cues (to specify the required response in advance), with the preparatory interval between cue and imperative stimulus strongly influencing performance. More sophisticated designs include electromyography or neuroimaging to separate premotor from motor activity, though such methods are less feasible in clinical settings. Nonetheless, RT tasks remain valuable tools for probing temporal cognition in PD and for capturing general slowness as well as variability across tasks, disease stages, and patient subtypes (Koch et al., 2008; Reif et al., 2023; Arroyo et al., 2021; Breska and Ivry, 2018).

The *duration bisection task* takes a different approach by testing interval categorization rather than reproduction. After learning short and long reference durations, participants judge whether subsequent intervals more closely match one or the other. This design is particularly informative for studying temporal discrimination and dopaminergic modulation of perceptual timing, though it does not capture all aspects of timing control (Terao et al., 2021; Torta et al., 2010; Coull et al., 2012; DiMarco et al., 2023).

The *temporal discrimination task* involves comparing two intervals and deciding which one was shorter or longer. Some variants include comparing visual changes (e.g., color shifts) to judge duration. This task provides insight into how PD affects the ability to discriminate between intervals (Bernardinis et al., 2019; Riesen and Schnider, 2001; da Silva et al., 2015).

In the *verbal estimation task*, participants are asked to estimate the duration of intervals by stating how long they believe a period lasted. Variants with or without internal counting assess both automatic and controlled time estimation, revealing dysfunction in various timing systems (Pastor et al., 1992a; Riesen and Schnider, 2001).

Another commonly used task is the *paced finger tapping (PFT) task*, which assesses motor timing and synchronization. Participants tap their index finger in synchrony with tones at 600-ms intervals for 30 s (synchronization phase), then continue tapping cues for an additional 30 s (continuation phase), with a rest period in between. Performance measures include inter-response intervals and variability, which assess rhythmic accuracy and consistency. Although the task captures motor timing well, it may be influenced by motor or cognitive deficits (Arroyo et al., 2021; Harrington et al., 2011). Similar approaches have also been

Table 1
Time processing tasks used to determine and objectify temporal timing. The list of the tasks is based on the articles included in this review. PD=Parkinson's Disease.

Task	Times used (n)	Protocol	Evaluation
Temporal orienting (Blandini et al., 2000**)**	1	Participants responded to events cued by predictive or non-predictive signals.	Good for assessing anticipation and prediction but limited to a narrow domain of temporal cognition.
Duration bisection (Terao et al., 2021; DiMarco et al., 2023; Fontes et al., 2016; Seo et al., 2025**)**	4	Judge whether a stimulus duration is closer to a short or long anchor. Trials use visual and auditory stimuli.	Strong tool for evaluating duration judgment. Reliable and well-suited for PD but focused on one temporal domain.
Duration production (Jones et al., 2008; Terao et al., 2021; Honma et al., 2016; Perbal et al., 2005**)**	4	Produce or tap out target time intervals (e.g., 60 s by tapping every subjective second).	Good assessment of internal timing. Sensitive to strategies like counting; still robust in PD research.
Time reproduction (Jones et al., 2008; Pastor et al., 1992a; Terao et al., 2021; Honma et al., 2016; Malapani et al., 1998; Koch et al., 2008; Dušek et al., 2012; Perbal et al., 2005; Mioni et al., 2018; Cohn-Sheehy and Ranganath, 2017; Pan and Luo, 2012**)**	11	Reproduce the duration of visual or auditory intervals. Varied formats and durations used.	High-quality, widely used method. Captures broad aspects of temporal processing in PD.
Reaction time trials (Jahanshahi et al., 1992; Arroyo et al., 2021; Breska and Ivry, 2018; DiMarco et al., 2023; Han and Proctor, 2022; Singh et al., 2023; Rolando et al., 2024**)**	7	Respond to stimuli as quickly as possible; often a distinction is made between simple and choice reaction time trials with and without warning; trials include rhythmic and non-rhythmic conditions	Effective for evaluating timing-related motor and cognitive response delays in PD.
Manual PRP blocks (Reif et al., 2023**)**	1	Press key in response to stimulus as quickly as possible.	Captures cognitive and motor delays in PD.
Vocal PRP blocks (Reif et al., 2023**)**	1	Vocalize correct response rapidly after given stimulus.	Assesses cognitive and vocal motor delay.
Temporal comparison / discrimination task - temporal order detection (Bernardinis et al., 2019; Terao et al., 2021; Riesen and Schnider, 2001; da Silva et al., 2015; Harrington et al., 2011; Parker et al., 2013**)**	6	Judge duration and compare which was longer or order of stimuli with varying intervals. Includes visual and auditory trials.	Robust tasks for assessing interval discrimination and temporal order; covers visual and auditory domains.
Subjective ranking of stimuli complexity (Zarkali et al., 2024**)**	1	Rank complexity of rhythmic stimuli (10 rhythms x 2 modes x 3 tempi).	Evaluates perception of rhythmic complexity; rich auditory temporal processing task.
Motor reproduction task (Torta et al., 2010**)**	1	Unscrew a bolt, then reproduce the time taken by tapping.	Combines motor execution with time estimation; ecologically valid.
(Verbal) Time estimation (Pastor et al., 1992a; Riesen and Schnider, 2001**)**	2	Estimate durations with or without concurrent tasks (tapping/reading)	Useful but susceptible to internal counting and task interference.
Temporal Generalization (Fontes et al., 2016**)**	1	Judge if intervals match a 400 ms standard with feedback.	Measures ability to generalize durations; structured but limited in variability.
Threshold Determination (Fontes et al., 2016**)**	1	Discriminate filled/unfilled intervals using adaptive procedure.	Comprehensive discrimination task; benefits from varied stimuli.
Time estimation (Howard and Eichenbaum, 2015**)**	1	Press a button after 1.2 s visual stimulus.	Simple and direct measure of internal timing; affected by attention.
Temporal Order Judgment (Perbal et al., 2005; da Silva et al., 2015**)**	2	Judge which of two tactile stimuli (left/right hand) came second; includes catch trials.	Assesses tactile temporal sequencing and interhemispheric processing; robust but complex.
Paced Finger Tapping (PFT) or wrist movement task (synchronization-continuation) (Arroyo et al., 2021; Pastor et al., 1992b; Singh et al., 2021; Rolando et al., 2024; Banquet et al., 2021**)**	5	Tap or move fingers or wrist respectively in sync with tones, then continue without tones; repeated with rest periods.	Measures motor timing and rhythmic continuation; reliable but affected by motor control.
Inspection time (Smith et al., 2007**)**	1	Identify longer line in briefly presented "pi" figure; stimulus duration adaptively adjusted.	Sensitive to perceptual speed; visually demanding, may be affected by acuity or attention.
Simple reaction time – sustained attention to response (Arroyo et al., 2021**)**	1	Press for digits 1–9 (Go); inhibit for "3" (No-Go).	Assesses sustained attention and inhibition; simple, but limited depth.
Variable foreperiod task (Teghil and Wittmann, 2025**)**	1	Respond to arrow after warning cue, interval varies randomly.	Captures temporal preparation and motor readiness; may be challenging for some due to variability.

applied to other effectors, such as repetitive wrist flexion-extension movements paced by auditory tones and continued without cues, sometimes combined with EMG to separate timing from movement execution. This task, like the PFT, probes internal timekeeping mechanisms and the extent to which external cues can stabilize rhythmic performance in PD (Pastor et al., 1992b).

Results from these tasks demonstrate the impact of PD on time processing across motor and cognitive domains, while also providing a means to assess affected neural circuits and therapeutic responses.

3.2. Cognitive processes involved in time processing in PD

3.2.1. Explicit, implicit, sub-second, and supra-second timing

Research by Mioni et al. (2018) has shown a dissociation between explicit and implicit timing in PD. *Explicit timing* refers to the conscious estimation or (re)production of temporal intervals and relies on basal

Table 2
Summary of studies (study type, aim, and main results) included in the review. 2AFC task=two-alternative forced-choice task, ACT-R= Adaptive Control of Thought—Rational, CD=Cerebellar Disease, CNV=Contingent Negative Variation, CRT=Choice Reaction Time, DaT=Dopamine Transporter, DBS=Deep Brain Stimulation, DLPFC=Dorsolateral Prefrontal Cortex, EMG=Electromyography, ERP=Event Related Potentials, fMRI=functional Magneting Resonance Imaging, HD=Huntington's Disease, IT=Inspection Time, MFC=Medical Frontal Cortex, MT=Motor Time, NMS=non-motor symptoms, ON=On dopaminergic medication, OFF=Off dopaminergic medication, PD=Parkinson's Disease, PFC=Prefrontal Cortex, RT=Reaction Time, SAS=Startling Acoustic Stimulus, SMA=Supplementary Motor Area, SRT=Simple Reaction Task, tDCS=transcranial Direct Current Stimulation, TMS=Transcranial Magnetic Stimulation, TOJ= Temporal Order Judgment, PSP=Progressive Supranuclear Palsy.

Title & author	Study type & aim	Main results
Evarts and Teräväinen (1981). *Reaction time in Parkinson's disease*	EMG and reaction time study comparing motor control and treatment effects in PD.	Movement time was more consistently impaired than reaction time. Reaction time can be independently affected. Movement time is a better treatment marker. Bradykinesia reflects impaired agonist activation.
Jahanshahi et al. (1992). *Simple and choice reaction time and the use of advance information for motor preparation in Parkinson's disease*	Experimental study to investigate simple and choice reaction time performance in PD and the effects of advance information.	PD patients were slower than controls in both SRT and CRT tasks, with greater impairment in CRT. Both groups benefited from warning signals and advance information, though patients required longer preparatory intervals to fully preprogramme responses.
Pastor et al. (1992). *Performance of repetitive wrist movements in Parkinson's disease*	Experimental study to assess the accuracy and variability of repetitive wrist flexion-extension movements in PD patients compared to controls, and to evaluate the effect of levodopa.	At higher movement rates, PD patients were significantly less accurate to controls. Patients showed greater variability in inter-response intervals, timekeeper, and motor delay components. Levodopa significantly improved timing accuracy and reduced variability.
Pastor et al. (1992). *Time estimation and reproduction is abnormal in Parkinson's disease*	Experimental study comparing PD patients and controls on time estimation/reproduction, with/without feedback and medication.	PD patients underestimated time verbally and overproduced it behaviorally. Feedback improved accuracy. Levodopa improved performance, suggesting dopamine's role in internal timekeeping.
Jahanshahi et al. (1993). *A comparative study of simple and choice reaction time in Parkinson's, Huntington's and cerebellar disease*	Experimental study to compare RT performance across PD, HD, and cerebellar disease patients using simple and choice RT tasks with varied warning signals and cues.	HD patients showed significantly longer SRT's than PD patients. All groups benefited from warning signals and advance response information, though PD patients were more dependent on warning cues to reduce RT. Similar RT deficits were observed though underlying mechanisms may differ.
Malapani et al. (1998). *Coupled temporal memories in Parkinson's disease: a dopamine-related dysfunction*	Experimental study examining interval timing in PD patients on and off medication, focusing on an internal time keeping mechanism, memory interference and dopamine effects.	PD patients off medication showed distorted timing and memory interference. Findings highlight dopamine's role in maintaining separate temporal memories and regulating internal timing mechanisms.
Riesen and Schnider (2001). *Time estimation in Parkinson's disease: normal long duration estimation despite impaired short duration discrimination*	Experimental study assessing short versus long duration timing in PD.	PD patients were impaired in short-duration discrimination but estimated long durations accurately. Findings suggest attentional, not intrinsic temporal, deficits in shorter interval tasks.
Elsinger et al. (2003). *Neural basis for impaired time reproduction in Parkinson's disease: an fMRI study*	fMRI during paced finger tapping task; compared brain activity on/off medication.	PD patients had reduced sensorimotor and SMA activity. Levodopa partially normalized brain activity without changing performance. It shows that fMRI detects compensation beyond behavioral measures.
Lewis and Miall (2003). *Distinct systems for automatic and cognitively controlled time measurement: evidence from neuroimaging*	Review to evaluate neuroimaging evidence on timing and determine whether different task characteristics engage distinct neural systems.	Two separate timing systems are suggested to be present: automatic timing, typically engaged in sub-second and motor-based tasks reliant on motor circuits including basal ganglia, cerebellum, and SMA, and cognitively controlled timing, required for supra-second and attention-demanding tasks, involving prefrontal and parietal regions – a dual-system framework.
Johnson et al. (2004). *Visual inspection time in Parkinson's disease: deficits in early stages of cognitive processing*	Two experimental studies examining IT in PD patients on and off medication to evaluate whether PD slows early perceptual processing and whether this is dopamine sensitive.	PD patients had longer IT than healthy controls, indication perceptual slowing. Levodopa had no effect on IT, suggesting bradyphrenia in PD is not responsive to dopaminergic medication and may involve alternative neurotransmitter systems.
Jones et al. (2004) *The right dorsolateral prefrontal cortex is essential in time reproduction: an investigation with transcranial magnetic stimulation*	Experimental study with rTMS to assess the role of the right dorsolateral prefrontal cortex and supplementary motor area in short and long interval timing during a time reproduction task.	rTMS over the right DLPFC led to underestimation of intervals compared with stimulation over a control motor area, especially for long intervals and during reproduction phase implicating the right DLPFC in working memory processes required for supra-second timing.
Praamstra and **Pope. (2007).** *Slow brain potential and oscillatory EEG manifestations of impaired temporal*	Experimental EEG study to assess whether PD patients show impaired temporal preparation and altered EEG responses during timing tasks.	PD patients lacked typical slow cortical potentials (CNV) and showed abnormal oscillatory activity compared to controls. Despite being able to

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Table 2 (*continued*)

Title & author	Study type & aim	Main results
preparation in Parkinson's disease		encode interval durations, PD patients did not engage in anticipatory neural activity, indicating a deficit in temporal preparation and striatal-based learning.
Smith et al. (2007). *The effect of Parkinson's disease on time estimation as a function of stimulus duration range and modality*	Experimental study that tested PD patients on duration bisection tasks (auditory/visual, short/long intervals).	PD patients had reduced sensitivity, especially at longer durations. Deficits were unaffected by meds. Suggests basal ganglia-related timing disruption.
Taatgen et al. (2007). *An integrated theory of prospective time interval estimation: the role of cognition, attention, and learning*	Computational modeling study to develop and test an integrated theory of prospective time perception that incorporates attention and learning within the ACT-R cognitive architecture.	Introduction of a time perception module fitted to existing datasets on interval estimation, bisection and dual-task effects, successfully predicting how attention and learning influence time estimation under varying conditions. Time perception interacts closely with cognitive control and learning processes.
Jones et al. (2008). *Basal ganglia, dopamine and temporal processing: performance on three timing tasks on and off medication in Parkinson's disease*	Comparative study of timing tasks in PD patients on/off medication versus controls.	PD patients showed abnormal timing variability and production errors. Dopaminergic medication worsened some timing tasks, supporting the dopamine overdose hypothesis.
Koch et al. (2008). *Impaired reproduction of second but not millisecond time intervals in Parkinson's disease*	Experimental study testing PD patients' reproduction of short (ms) and long (sec) intervals on/off L-dopa.	PD patients off L-dopa underestimated supra-second intervals but preserved millisecond timing. Timing impairments surfaced under increased cognitive load and were not restored by dopamine therapy.
Merchant et al. (2008). *Interval timing and Parkinson's disease: heterogeneity in temporal performance*	Experimental study using multiple timing tasks to assess variability and dopaminergic influence in PD.	PD patients showed heterogeneous timing abilities. Dopaminergic therapy reduced variability and restored inter-task correlations, supporting the basal ganglia-thalamocortical pathway's role in temporal processing.
Wearden et al. (2008). *Stimulus timing by people with Parkinson's disease*	Experimental, assessing timing tasks in PD patients on/off medication versus controls.	PD patients showed similar performance to controls in most, non-motor, tasks; minor deficits appeared in sequential processing, suggesting possible attentional rather than pure timing issues. Medication had minimal impact.
Wild-Wall et al. (2008). *Time estimation in healthy ageing and neurodegenerative basal ganglia disorders*	Comparative study across PD, HD, and control groups; tested interval timing.	PD, HD, and aging showed impaired timing compared to young adults. No difference between older adults and patient groups. It suggests frontostriatal dysfunction affects timing regardless of severity.
Koch et al. (2009). *Neural networks engaged in milliseconds and seconds time processing: evidence from transcranial magnetic stimulation and patients with cortical or subcortical dysfunction*	Review of imaging and brain stimulation studies on timing, including PD and cortical/subcortical lesion data.	Timing relies on a distributed network: cerebellum for milliseconds; basal ganglia and PFC for supra-second intervals. Fronto-parietal and subcortical loops mediate context-dependent time representation.
Barone et al. (2010). *Neurotransmission in Parkinson's disease: beyond dopamine*	Narrative review to examine the role of non-dopaminergic neurotransmission systems in PD, particularly in relation to non-motor symptoms and their interaction with dopamine.	Non-motor symptoms are major contributors to disability in PD and can appear before motor onset. Neuropathological evidence shows that cholinergic, serotonergic, noradrenergic, and other systems are involved alongside dopamine. Treatments targeting only dopamine fail to address these symptoms fully.
Torta et al. (2010). *Dissociation between time reproduction of actions and of intervals in patients with Parkinson's disease*	Experimental study comparing reproduction of action durations and externally presented intervals.	PD patients had impaired reproduction of self-paced action durations but not passive intervals, implicating motor and attentional demands in timing deficits and informing rehabilitation strategies.
Harrington et al. (2011). *Neurobehavioral mechanisms of temporal processing deficits in Parkinson's disease*	fMRI study examining brain activity during timing tasks on/off dopamine therapy.	Timing deficits were linked to disrupted activity in striatum, SMA, and fronto-parietal regions. DA therapy improved motor symptoms but not time perception or cortical-striatal connectivity.
Jones et al. (2011). *Modeling accuracy and variability of motor timing in treated and untreated Parkinson's disease and healthy controls*	Experimental study comparing motor timing accuracy and variability in de novo PD patients, treated PD patients (on/off medication), and healthy controls using a synchronization-continuation tapping task at different intervals.	Treated PD patients performed ahead of the beat, whereas de novo PD patients and controls lagged. This hastening may reflect motor festination. Variability increased at longer intervals, particularly during continuation, overall suggesting that motor timing accuracy best distinguishes PD patients from controls.
Wojtecki et al. (2011). *Modulation of human time processing by subthalamic deep brain stimulation*	Experimental within-subject study examining the effect of different STN-DBS frequencies on time processing in PD.	Low-frequency (10 Hz) STN-DBS worsened underproduction of longer (15 s) time intervals, whereas high-frequency stimulation did not. Timing in the millisecond range was unaffected, suggesting dissociation between timing systems. Findings indicate a frequency-specific modulation of interval timing, supporting basal ganglia involvement in supra-second timing.

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Table 2 (*continued*)

Title & author	Study type & aim	Main results
Allman and **Meck. (2012).** *Pathophysiological distortions in time perception and timed performance*	Review of how basal ganglia dysfunction and dopamine alterations affect timing in neurological and psychiatric disorders.	In PD, off-medication states cause timing distortions (e.g., 'migration effect'). Dopamine improves timing only partially. Timing deficits impact cognition and behavior; rhythm-based interventions may offer therapeutic benefit.
Dušek et al. (2012). *Abnormal activity in the precuneus during time perception in Parkinson's disease: an fMRI study*	fMRI study examined the neural basis of timing deficits and the 'migration effect' in PD ON/OFF state.	Greater precuneus deactivation in OFF state during reproduction phase was linked to timing errors. It suggests memory retrieval, not clock-speed, deficits; dopamine may compensate by restoring precuneus activity.
Carlsen et al. (2013). *Using a startling acoustic stimulus to investigate underlying mechanisms of bradykinesia in Parkinson's disease*	Experimental RT task using SAS to test whether PD motor slowing reflects deficits in initiation versus programming.	SAS reduced RT and normalized movement speed in PD, indicating intact motor programming but impaired movement initiation due to basal ganglia dysfunction. EMG analysis confirmed partial motor recovery with SAS, suggesting potential for initiation-targeted interventions.
Claassen et al. (2013) *Deciphering the impact of cerebellar and basal ganglia dysfunction in accuracy and variability of motor timing*	Experimental study to compare motor timing accuracy and variability in patients with cerebellar disorders, PD, and healthy controls using a synchronization-continuation tapping task at different intervals.	CD patients tended to lag the beat, while medicated PD patients tended to hasten ahead of the beat. Variability was greater in CD patients than controls, PD patients showed less variability. There are distinct contributions of cerebellar and basal ganglia dysfunction to motor timing, with cerebellar deficits linked to variability and basal ganglia dysfunction linked to systematic timing shifts.
Dirnberger and Jahanshahi (2013). *Executive dysfunction in Parkinson's disease: a review*	A review to determine the nature of executive dysfunction in PD, its natural substrates, the effects of dopaminergic treatment and its clinical implications.	Executive dysfunction is common even in early PD, affecting attention control, set shifting, planning, inhibition, dual tasking, decision-making, and social cognition. It correlates with motor symptoms and psychiatric features. Dopaminergic treatment has mixed effects.
Parker et al. (2013). *Executive dysfunction in Parkinson's disease and timing deficits*	Review focusing on how executive dysfunction contributes to perceptual timing deficits in PD.	Timing deficits in PD relate to frontostriatal dysfunction. May serve as cognitive biomarkers.
Bella et al. (2015). *Effects of musically cued gait training in Parkinson's disease: beyond a motor benefit*	Review article analyzing gait disorders and auditory cueing interventions in PD. Aim is to assess whether gait timing deficits in PD can be improved via auditory cueing and whether benefits extend beyond motor control.	Auditory cueing (e.g., musically cued gait training) improves spatiotemporal gait parameters in PD and enhances sensorimotor timing. Benefits may generalize to perceptual and cognitive timing and help mitigate nonmotor deficits such as language impairments.
da Silva et al. (2015). *Aging and Parkinson's disease as functional models of temporal order perception*	Studies experimentally visual TOJ tasks in young, elderly, and PD patients on medication.	PD and elderly had poorer temporal order resolution than young adults. Attention affected performance. Dopamine loss likely increases noise in perceptual decision-making, impairing timing.
Nishikawa et al. (2015). *Effects of aging and idiopathic Parkinson's disease on tactile temporal order judgment*	A comparative experimental study comparing tactile TOJ in PD, age-matched, and young controls.	PD did not impair TOJ, but aging did. Longer judgment reversals and more errors in elderly, especially arms crossed. TOJ seems independent of dopamine-related circuits.
Avanzino et al. (2016). *Time Processing and Motor Control in Movement Disorders*	Narrative review exploring timing mechanisms and motor control in various movement disorders.	Abnormal timing in PD is linked to basal ganglia dysfunction. Rhythmic auditory cues improve motor symptoms; implicit and explicit timing rely on distinct circuits.
Cameron et al. (2016). *The Effect of Dopaminergic Medication on Beat-Based Auditory Timing in Parkinson's Disease*	Experimental study. Participants with PD were tested on beat perception tasks, ON/OFF medication. Controls were tested twice to assess learning effects. Aim was to examine the effect of dopaminergic medication on beat-based auditory timing in PD.	PD patients performed worse than controls; performance declined with greater disease severity. Controls improved across sessions, PD patients did not, indicating learning deficits. Dopaminergic medication altered rhythm perception, especially for complex rhythms, suggesting nuanced effects on timing mechanisms.
Honma et al. (2016). *Dysfunctional counting of mental time in Parkinson's disease*	Experimental using time production/reproduction tasks and DaT imaging to study PD timing.	PD patients underestimated long intervals in production tasks due to faster internal clock cycles. DaT deficits correlated with underestimation. Reproduction tasks were intact, suggesting pacemaker-specific dysfunction.
Vlagsma et al. (2016). *Mental slowness in patients with Parkinson's disease: Associations with cognitive functions?*	Empirical study using RT and MT measures to differentiate between mental (RT) and motor (MT) slowness in PD and assess impact on neuropsychological test performance.	PD patients had prolonged RTs (mental slowness) but no MT difference from controls. RT and MT shared limited variance. Mental slowness did not affect neuropsychological test performance. Motor speed needs to be considered when interpreting cognitive assessments.
Zhang et al. (2016). *Time on timing: Dissociating premature responding from interval sensitivity in Parkinsons' disease*	Behavioral and computational modeling study assessing timing sensitivity and premature responding during temporal discrimination in PD.	PD patients showed reduced timing sensitivity and altered perception of time intervals in both bisection and trisection tasks. They also exhibited faster responses under

(continued on next page)

Table 2 (*continued*)

Title & author	Study type & aim	Main results
		ambiguous conditions, indicating premature or impulsive decision-making. Modeling revealed both impaired temporal precision and altered decision processes contributing to timing deficits.
Breska and Ivry (2018). *Double dissociation of single-interval and rhythmic temporal prediction in cerebellar degeneration and Parkinson's disease*	Experimental, giving a neuropsychological comparison of PD, cerebellar degeneration, and healthy controls to test whether rhythm- and interval-based timing rely on distinct neural circuits.	PD patients were impaired in rhythm-based predictions but intact in single-interval timing. The reverse pattern was found in cerebellar degeneration. Findings show dissociable timing mechanisms: basal ganglia for rhythm-based prediction and cerebellum for interval-based prediction.
Magalhães et al. (2018). *Neurochemical changes in basal ganglia affect time perception in parkinsonians*	Review examined how dopaminergic dysfunction in PD affects time perception.	Time perception involves multiple brain areas; dopamine regulates internal clock speed. Levodopa improves motor/temporal function but may impair cognition. PD affects timing via basal ganglia damage and executive function deficits.
Mioni et al. (2018). *Dissociating Explicit and Implicit Timing in Parkinson's Disease Patients: Bisection and Foreperiod Tasks*	Experimental study comparing PD patients' performance on explicit (bisection) and implicit (foreperiod) timing tasks.	PD patients showed deficits in explicit, but not implicit timing, supporting a dissociation between these processes. Explicit timing relies on basal ganglia and dopamine; implicit timing appears spared.
Rosqvist et al. (2018) *Dopaminergic Effect on Non-Motor Symptoms in Late-Stage Parkinson's Disease*	Clinical study to evaluate the effect of dopaminergic therapy on NMS in late-stage PD and explore its relation to motor response.	NMS were highly prevalent, levodopa significantly improved overall NMS scores generally, but greater in motor responders. Dopaminergic therapy provides meaningful relief of NMS even in late-stage PD.
Bareš et al. (2019). *Consensus paper: Decoding the Contribution of the Cerebellum as a Time Machine. From Neurons to Clinical Applications*	Consensus review synthesizing evidence of the cerebellum's role in timing across motor and cognitive domains.	The cerebellum contributes to timing at multiple levels. Its role in PD may be compensatory or integrated within broader timing networks involving basal ganglia and cortex.
Behroozmand and Johari (2019). *Sensorimotor Impairment of Speech and Hand Movement Timing Processing in Parkinson's Disease*	Experimental study examining reaction times for speech and hand movement under predictable versus unpredictable cues.	PD patients did not benefit from temporal predictability, suggesting impaired predictive sensorimotor timing mechanisms across motor domains.
Bernardinis et al. (2019). *Differential Temporal Perception Abilities in Parkinson's Disease Patients Based on Timing Magnitude*	Experimental 2AFC task assessing visual temporal discrimination in PD across different therapies.	PD patients had impaired visual temporal perception, unaffected by Levodopa or DBS. Sub-second timing was spared, implicating involvement of the basal ganglia in interval timing deficits.
Tomassini et al. (2019). *Learning from the past and expecting the future in Parkinsonism: Dopaminergic influence on predictions about the timing of future events*	Experimental study comparing medicated and unmedicated PD patients with controls using a variable foreperiod RT task to examine dopamine's role in forming temporal predictions and regulating temporal uncertainty in PD.	PD patients OFF medication showed impaired use of prior information to prepare for timed events, leading to slower responses. Dopaminergic medication restored performance to control levels. Dopamine depletion increased subjective temporal uncertainty and cautious responding, as modeled by changes in drift-diffusion parameters.
Vikene et al. (2019). *Abnormal phasic activity in saliency network, motor areas, and basal ganglia in Parkinson's disease during rhythm perception*	Experimental fMRI study: it analyzed brain activation during rhythm perception in PD versus controls.	PD patients showed hyperactivity in auditory, parietal and frontal areas, likely compensating for basal ganglia deficits. Rhythm was perceived as more complex by PD patients.
Yin et al. (2019). *The right dorsolateral prefrontal cortex is essential in seconds range timing, but not in milliseconds range timing: An investigation with transcranial direct current stimulation*	Experimental study with tDCS to examine the causal role of the right dorsolateral prefrontal cortex in milliseconds-versus seconds-range timing using a temporal bisection task.	The right dorsolateral prefrontal cortex is specifically involved in supra-second timing, likely through cognitive processes such as working memory, supporting dual-system models of time perception.
Zhang et al. (2019). *Scopolamine and Medial Frontal Stimulus-Processing during Interval Timing*	Experimental mouse study investigating effects of cholinergic inhibition on MFC (medial frontal cortex) activity during interval timing.	Scopolamine impaired timing performance but preserved MFC ramping activity. Increased stimulus processing suggests cholinergic dysfunction affects timing through non-MFC mechanisms.
Arroyo et al. (2021). *Components determining the slowness of information processing in Parkinson's disease*	Experimental, multi-component reaction time task study to determine whether PD-related cognitive slowness affects specific or general processing stages.	PD patients showed generalized slowing across tasks. Slowness was most linked to deficits in motor and perceptual components, as well as sustained alertness. Cognitive decision-making components were largely preserved.
Honma et al. (2021). *Stopwatch training improves cognitive functions in patients with Parkinson's disease*	Intervention study evaluating effects of feedback training on time perception and cognition.	Training improved duration estimation and cognitive control, but effects diminished after a month.
Singh et al. (2021). *Timing variability and midfrontal ~4 Hz rhythms correlate with cognition in Parkinson's disease*	Cross-sectional EEG study investigating the relationship between timing variability, midfrontal ~4 Hz activity, and cognitive dysfunction in PD.	PD patients showed increased variability in time estimation despite similar mean performance compared to controls. Reduced midfrontal ~4 Hz EEG activity was observed and correlated with worse cognitive function. Trial-by-trial analyses showed that lower ~4 Hz power predicted greater timing variability, linking cortical

(*continued on next page*)

Table 2 (*continued*)

Title & author	Study type & aim	Main results
		oscillations to cognitive deficits.
Terao et al. (2021). *Time Distortion in Parkinsonism*	Experimental study examining timing distortions in PD and PSP across motor and perceptual tasks.	Timing in PD is task-dependent: motor tasks show accelerated perception and processing; perceptual tasks show opposite effects. Feedback helps but is short-lived.
Zokaei et al. (2021). *Temporal orienting in Parkinson's disease*	Experimental study on attention to temporal orienting tasks using predictive cues in PD.	PD patients show reduced ability to orient attention in time, impairing prediction of event timing
Han and Proctor (2022). *Revisiting variable-foreperiod effects: evaluating the repetition priming account*	Experimental study to test whether sequential foreperiod effects in reaction time tasks are better explained by repetition priming or by the multiple-trace theory.	Short foreperiods showed larger sequential effects than longer ones, inconsistent with repetition-priming predictions. Results aligned with the multiple-trace theory, which attributes variability in reaction times to memory traces of prior intervals.
Low et al. (2002). *Response slowing in Parkinson's disease: a psychophysiological analysis of premotor and motor processes*	ERP-based experimental study, dissected reaction time into premotor and motor stages.	Reaction time delays in PD stem from both motor and premotor deficits. Premotor slowing appears early and without dementia. Response complexity has more impact than stimulus difficulty.
Miyawaki (2022). *Review: Subjective Time Perception, Dopamine Signaling, and Parkinsonian Slowness*	Narrative review to synthesize evidence on dopamine's role in time perception and its relation to motor symptoms like bradykinesia in PD.	Dopamine loss disrupts short-term temporal calibration, contributing to misperceptions like overestimation of short intervals and underestimation of long ones. Subjective timing dysfunction may underlie motor symptoms like festination and freezing in PD.
Su et al. (2022). *Parkinson's disease deficits in time perception to auditory as well as visual stimuli – A large online study*	Large-scale online experimental study to examine time perception deficits in PD across sensory modalities and assess feasibility of virtual cognitive testing.	PD patients showed reduced duration discrimination sensitivity in both auditory and visual modalities. Findings support a basal ganglia role in suprasecond interval processing. Online testing proved effective for assessing cognitive deficits in PD at scale.
DiMarco et al. (2023). *Time perception reflects individual differences in motor and non-motor symptoms of Parkinson's disease*	Experimental study investigating how individual differences in motor and non-motor symptoms in PD relate to interval timing behavior and dopaminergic dysfunction.	No overall timing difference was found between on- and off-medication states. Interval timing behavior predicted treatment type, impulse control disorders, depression, and disease duration, suggesting its potential as a behavioral biomarker in PD.
Reif et al. (2023). *Prolonged Processing Time for Manual and Vocal Responses in Parkinson Disease*	Experimental study using a dual-task paradigm to assess motor versus cognitive response delays in PD.	PD patients showed longer response times and greater delays in high-overlap tasks, indicating central processing bottlenecks and heightened cognitive demands.
Singh et al. (2023) *Evoked midfrontal activity predicts cognitive dysfunction in Parkinson's disease*	Observational EEG study examining whether midfrontal delta/theta activity predicts cognitive dysfunction across tasks in PD.	PD patients with greater cognitive impairment showed slower responses and reduced midfrontal delta/theta activity across tasks. Low-frequency EEG activity strongly correlated with cognitive scores. Results suggest that impaired midfrontal oscillations are a neural marker of cognitive dysfunction in PD.

ganglia function and dopaminergic pathways. This form of timing is typically assessed using tasks such as finger tapping, time reproduction, time production, and time estimation, and is consistently impaired in PD. In contrast, *implicit timing* operates without conscious estimation of duration and reflects the brain's ability to use temporal regularities to predict when events will occur. Implicit timing remains relatively preserved in PD. One used task to assess implicit timing is the foreperiod task, in which participants respond to a target stimulus that is preceded by a warning signal. The interval between the warning signal and the target, known as the foreperiod, influences response preparation and reaction time. In the fixed-foreperiod paradigm, the interval remains constant within a block of trials; under these conditions, response times are typically faster for shorter intervals. In contrast, the variable-foreperiod paradigm randomly intermixes shorter and longer foreperiods, creating temporal uncertainty. Here, response times generally decrease as the interval lengthens, reflecting participants' increasing expectation that the target will appear as time elapses (the "variable foreperiod effect"). Because performance in these tasks depends on implicit temporal prediction rather than explicit duration judgments, performance in foreperiod tasks has been shown to remain relatively preserved in patients with PD (Mioni et al., 2018; Han and Proctor, 2022).

Building on this dissociation, time processing is divided into sub-second and supra-second timing. Sub-second timing, typically automatic and sensory based, involves perceptual or motor processes and is supported by the frontal operculum, cerebellum, and temporal gyri (Terao et al., 2021; Szelag et al., 2022). Conversely, supra-second timing relies more on working memory and cognitive control, engaging the frontal cortex and basal ganglia to integrate information across time. In line with this, patients with PD generally show more pronounced and consistent impairments in supra-second tasks that require attention and memory resources (Terao et al., 2021; Szelag et al., 2022). However, the presence of bradykinesia, slowness of movement, indicates that sub-second timing cannot be fully preserved. Bradykinesia reflects disturbances in the timing of motor execution affecting voluntary movement and reducing the speed of automatic or repetitive movements. This points to a degree of impairment in sub-second mechanisms as well. This pattern suggests that both sub-second and supra-second timing are affected in PD (Terao et al., 2021), with impairments in the latter domain appearing more robust across research on time processing deficits in PD.

Neuroimaging evidence supports this dissociation, showing that sub-second and supra-second timing rely on distinct neural circuits and oscillatory activity (Rammsayer et al., 2015; Meck et al., 2008; Jones and Jahanshahi, 2014), but with the presence of a certain amount of overlap. When examining results that focus on automatic task characteristics, activation is observed most consistently in the motor system.

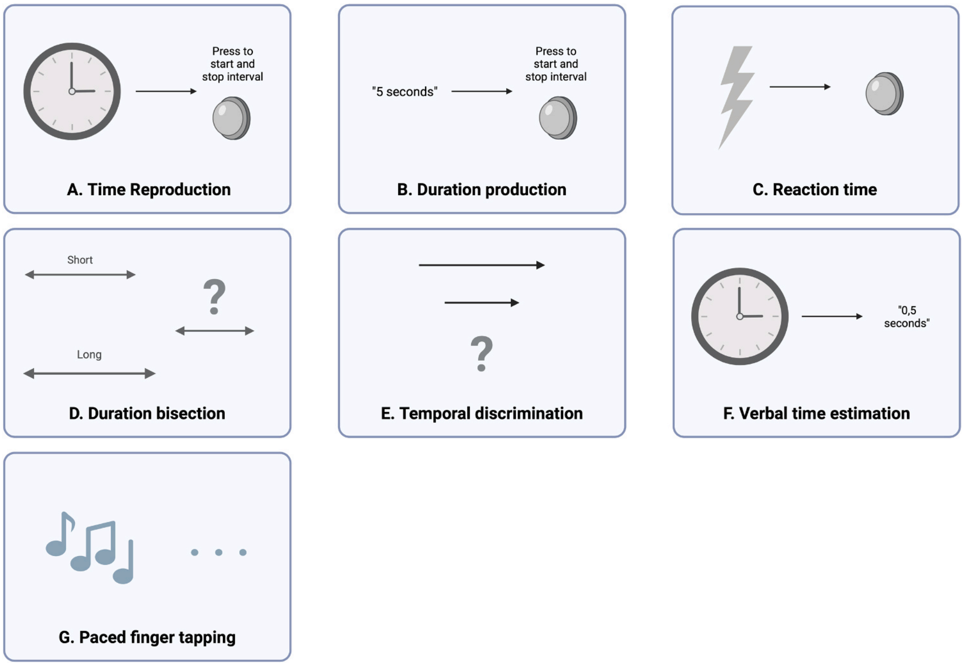

Fig. 2. Overview of tasks commonly used to assess time processing in Parkinson's disease. Each pictogram (A-G) represents a distinct timing task used in temporal processing research, capturing different aspects of time perception and motor timing. A. Time/Interval reproduction task. Participants observe a stimulus of a given duration and are then asked to reproduce that interval, typically via a button press or tap. This task assesses time estimation and reproduction, often influenced by cognitive strategies (e.g., silent counting) and dopamine levels. B. Time/Duration production task. Participants are instructed to generate specific time intervals (e.g., 2, 5, 10 s) by marking their start and end. This task evaluates the accuracy of internally generated timing. C. Simple reaction time task. Measures the speed of a participant's response to a stimulus, reflecting both motor and cognitive processing. Particularly relevant in PD to distinguish between motor slowing and cognitive delay. D. Duration bisection task. Participants judge whether a presented interval is closer to a previously learned "short" or "long" anchor. This assesses temporal discrimination and categorical decision-making. E. Temporal comparison/discrimination task. Participants compare two sequential intervals and decide which lasted longer. This task highlights subtle deficits in duration discrimination, often observed in PD. F. Verbal time estimation task. Participants verbally estimate how long an interval lasted. This task taps into internal time perception and may reveal the use of controlled versus automatic timing strategies. G. Paced finger tapping task. Participants tap in sync with regular tones and then continue tapping in their absence. Variability in tapping reveals information about motor timing and rhythmic synchronization.

The bilateral supplementary motor area (SMA) and the left sensorimotor cortex show the strongest activity, followed closely by the right cerebellum and the lateral premotor cortex (PMC). The left thalamus and basal ganglia are also engaged, while the right superior temporal gyrus is frequently active. In contrast, the prefrontal cortex and most parietal regions are only rarely recruited (Lewis and Miall, 2003).

For results emphasizing cognitive-control-related task characteristics, some overlap with the automatic system is evident, particularly in the right PMC and bilateral SMA. However, these conditions additionally engage the left cerebellum as well as prefrontal and parietal cortices, with a tendency toward greater activation in the right hemisphere (Lewis and Miall, 2003). This pattern suggests that while cognitively controlled timing can involve elements of automatic systems, it also requires the recruitment of additional brain regions. The specific contributions of these key brain regions will be discussed in more detail in Section 3.3.1.

3.2.2. *Intrinsic models and the role of working memory and attention in time processing*

Neurocomputational models provide insights into how the brain processes time. The *pacemaker-accumulator model*, a prominent extrinsic or dedicated model, conceptualizes an internal clock comprising a pacemaker that generates pulses, an accumulator that counts them, and a switch that regulates the process (Ivry and Schlerf, 2008; Hass et al., 2008). The pacemaker-accumulator model has been further elaborated in the attentional gate framework (Fig. 3). In this model, an internal pacemaker emits pulses at a certain rate, which can be modulated by physiological arousal. Increased arousal leads to a faster pacemaker, thereby producing more pulses within a given interval, whereas reduced arousal slows the pulse rate (Taatgen et al., 2007). A second component, often termed the "attentional gate", regulates how many of these pulses reach the accumulator (Taatgen et al., 2007; Branaghan and Sanchez, 2009). When attention is directed toward time, the gate is more open, allowing more pulses to pass through; when attention is diverted elsewhere, the gate narrows, and fewer pulses are transmitted. The pulses that reach the accumulator are stored in working memory and compared with reference memories of durations, providing the basis for a subjective estimate of elapsed time (Taatgen et al., 2007). The model, therefore, links temporal perception to both arousal and attentional allocation: overestimation of duration can result from heightened arousal or increased attention to time, while underestimation can occur when attention is absorbed by non-temporal demands (Taatgen et al., 2007; Branaghan and Sanchez, 2009). This model proposes the presence of a specialized neural mechanism, combining attention and working memory, suggesting that deficits in timing may result from disruptions in these cognitive systems (Ivry and Schlerf, 2008; Hass et al., 2008; Taatgen et al., 2007; Harrington and Haaland, 1999).

In contrast to models proposing dedicated timing mechanisms, intrinsic models suggest that time perception emerges from the general dynamics of neural processes or activity from a broader neural network rather than from specialized mechanisms. The *Striatal Beat Frequency (SBF) model* (Fig. 3), for instance, explains time perception through interactions of neural oscillations in the striatum, with no reliance on a pacemaker (Ivry and Schlerf, 2008; Oprisan et al., 2023; Karmarkar and Buonomano, 2007; Gu et al., 2015; Maniadakis and Trahanias, 2014b). These models argue that working memory and attention influence time perception, but within the framework of distributed neural networks rather than isolated timing mechanisms (Oprisan et al., 2023;

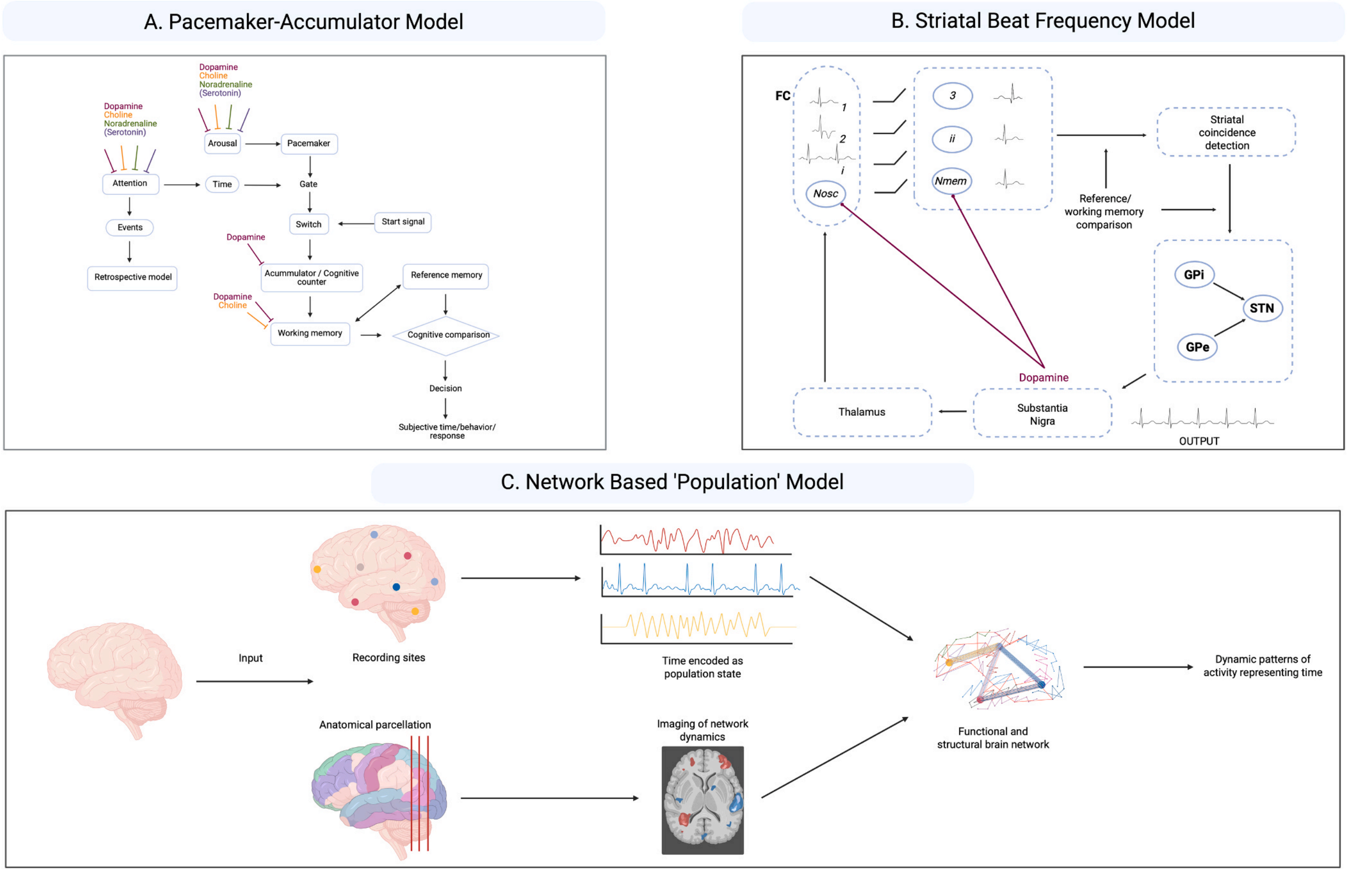

Fig. 3. Conceptual models of time processing. A. Pacemaker-accumulator model with attentional gate. A pacemaker emits pulses that pass through an attentional gate into an accumulator. The number of accumulated pulses is sorted in working memory and compared with reference memory to generate temporal judgments. The gate regulates pulse flow based on attentional allocation, while the pacemaker rate is influenced by arousal. Disruptions in attention or working memory alter pulse accumulation and comparison, leading to timing inaccuracies. B. Striatal Beat Frequency (SBF) model. Cortical neurons exhibit oscillatory activity at different frequencies, which are synchronized at the onset of an interval. The striatum detects coincident patterns of these oscillations ("beats") to encode elapsed time. Dopaminergic input from the substantia nigra modulates this process, influencing both timing accuracy and variability. Output is relayed via basal ganglia circuits (including globus pallidus interna (GPi), globus pallidus externa (GPe), and subthalamic nucleus (STN) to guide behavior. C. Network-based "Population" model. Time is represented as dynamic patterns of activity across distributed neural populations rather than a centralized clock. Temporal information emerges from evolving population states within interconnected cortical and subcortical networks. These patterns could be captured using electrophysiology or neuroimaging and reflect both structural connectivity and functional dynamics. Together, these models illustrate complementary perspectives on time processing: a centralized clock-like mechanism (A), a striatal coincidence detection system (B), and a distributed network representation of time (C).

Karmarkar and Buonomano, 2007; Gu et al., 2015; Maniadakis and Trahanias, 2014b). This network-level perspective helps explain the broader cognitive and motor disturbances in PD, implying that several brain systems are involved in the timing deficits observed. It is likely, however, that both dedicated and intrinsic timing models describe complementary mechanisms that operate under different task conditions and are differently affected in PD.

Although the SBF model has been highly influential in conceptualizing interval timing, direct empirical support for its proposed mechanism, coincidence detection of cortical oscillators in the striatum, remains limited (Paton and Buonomano, 2018). Beyond the influential SBF framework, increasing evidence shows alternative models in which time is encoded through distributed and dynamic neural activity rather than a centralized clock or oscillatory detection. *Network based "Population" model* (Fig. 3) accounts propose that temporally evolving patterns of activity across neural ensembles, known as "population clocks", encode elapsed time in both cortical and subcortical circuits, including the striatum, hippocampus, prefrontal, and parietal cortex (Bakhurin et al., 2017). In line with this, recordings from the striatum demonstrate that neuronal populations exhibit sequential firing patterns that scale with interval duration, providing a flexible population code for relative time. Striatal population activity, therefore, reflects an internal estimate of temporal position within an interval, which can be used to guide behavior into expected events (Mello et al., 2015). Similarly, cortico-striatal ensemble studies show that time can be represented through ramping activity (the consistent, gradual increase or decrease in neuronal firing rate) and learning-dependent changes in neural dynamics, indicating that timing emerges from circuit-level adaptations rather than a fixed mechanism (Emmons et al., 2020). Complementary computational approaches further suggest that striatal activity may reflect accumulation processes consistent with drift-diffusion models, where timing arises from the integration of temporal evidence across several neural populations (Bruce et al., 2025). Importantly, these models are not mutually exclusive and may operate across different tasks and timescales. Temporal processing may be inherently heterogeneous and emerges from the dynamics of distributed neural circuits rather than a single unified timing mechanism.

Humans constantly engage in temporal activities, such as timing movements, recalling events, and expressing general knowledge, some of which rely on memory, influencing our perception and, therefore, our process of timing (Ivry and Spencer, 2004). The four memory systems – episodic, semantic, working, and procedural– serve different functions in this process (Fontes et al., 2016), with working memory having the most important focus.

Working memory is valuable in time processing, especially in tasks that require planning and timing movements in various sequences (Fontes et al., 2016; Mızrak and Oberauer, 2021; Cohn-Sheehy and Ranganath, 2017; Pan and Luo, 2012; Polti et al., 2018). Studies reveal that reduced attention influences subjective time perception and working memory negatively (Ivry and Spencer, 2004; Polti et al., 2018). Research by Polti et al. (2018) examined the effects of attention and working memory load on prospective duration estimation. They showed that directing attention to the perceived duration of time lengthens it, while dividing attention with a concurrent working memory task shortens it. The degree of underestimation increased with working memory load, particularly for longer durations, indicating that working memory demands distort time perception more for extended intervals (Polti et al., 2018), a phenomenon also observed in research by Reif and colleagues (2023) (Reif et al., 2023). They found that PD patients exhibited significantly longer response times under conditions of high cognitive demand and task overlap.

Wearden et al. (2008) suggest that working memory plays a key role in time processing deficits in PD. Although PD patients generally perform well on many timing tasks, tasks that involve processing two sequential stimuli, requiring attentional and working memory resources, consistently reveal poorer performance compared to controls. Hence, the presented difficulties arise not from a fundamental timing deficit but from impairments in sequential processing, attention, and working memory (Fontes et al., 2016; Wearden et al., 2008).

In examining how the motor, perceptual, and cognitive components of time processing are affected in PD, Arroyo et al. (2021) observed general slowing of reaction time across all tasks, with pronounced impairments in perceptual discrimination and sustained alertness. Motor and attention factors explained the overall slowness, so besides motor alterations, disruptions related to alertness contribute to cognitive slowing as well.

Recent work indicates that temporal variability, rather than absolute accuracy, may represent a more robust marker of timing dysfunction in PD. For instance, increased variability in interval timing has been consistently observed and shown to correlate with cognitive impairment and executive dysfunction (Singh et al., 2021). This variability has been linked to disrupted midfrontal low-frequency (~4 Hz) activity, suggesting impaired cognitive control mechanisms are the main problem in timing performance (Singh et al., 2021). Converging evidence demonstrates that alterations in midfrontal delta/theta oscillations are strongly associated with cognitive dysfunction in PD, further elaborating on a link between temporal processing and executive control networks (Singh et al., 2023). In line with this, a recent meta-analytic (preprint) approach shows that temporal precision is consistently impaired across studies, whereas timing accuracy remains relatively preserved, reinforcing the notion that variability may be a key behavioral signature of timing deficits in PD (Weber et al., 2026).

Collectively, the evidence suggests that slowed performance in PD arises not from a fundamental timing deficit but from limited cognitive processing capacity. When attentional or working memory resources are strained, particularly during tasks involving longer intervals, increased cognitive demands, or overlapping stimuli, patients with PD experience greater difficulty maintaining accurate timing. This pattern of timing impairments in PD appears to result from deficits in attention, sequential processing, and working memory rather than a primary dysfunction of timing mechanisms (Fang et al., 2020; Torta et al., 2010; Perbal et al., 2005; Reif et al., 2023; Riesen and Schnider, 2001; Wearden et al., 2008). Consistent with this view, time processing in PD shows a dissociation between explicit (conscious, basal ganglia-dependent) and implicit (automatic) timing, with impairments primarily affecting explicit timing. This distinction parallels differences between sub-second (automatic, sensory-based) and supra-second (cognitive, memory-based) timing, with the latter being more strongly affected in PD. Theoretical models of time processing, including the pacemaker-accumulator and the Striatal Beat Frequency model, describe the important roles of attention and working memory in maintaining accurate temporal representations, but other theories on timing appear to lean towards the presence of a broader network consisting of several neural populations, cognitive functions, and structures.

3.3. *Neurophysiology of time processing in Parkinson's disease*

3.3.1. *Key brain structures*

Time processing, which ranges from milliseconds to seconds, relies on the coordinated activity of several brain regions, including the prefrontal and parietal cortices, basal ganglia, and cerebellum (Fig. 4). Together, these regions integrate sensory input, motor output, and cognitive demands, with neurotransmitters such as dopamine and acetylcholine playing a central role in supporting accurate timing (Bohnen and Albin, 2011; Terao et al., 2021; Arroyo et al., 2021; Maniadakis and Trahanias, 2014b; Ivry and Spencer, 2004; Fontes et al., 2016).

Cortical oscillations provide the neural foundation for more complex timing operations (Harrington et al., 1998). The prefrontal cortex, particularly the right dorsolateral region, is involved in time processing (Yin et al., 2019). Lesions in this area impair temporal discrimination, and evidence suggests that the right dorsolateral prefrontal cortex (DLPFC) is especially important for timing in the seconds range, where it supports the transfer and storage of temporal information in working memory during reproduction tasks (Jones et al., 2004). In contrast, the supplementary motor area (SMA) is consistently engaged in rhythm production and monitoring, with lesion and imaging studies proving its role in temporal sequencing (Jones et al., 2004). Impairments in midfrontal low-frequency oscillations (delta/theta range) have been consistently linked to deficits in cognitive control, attention, and, therefore, timing in PD, again defending the idea of impairments in cortical oscillations contributing to time processing deficits (Singh et al., 2023).

Lateralization also appears to matter. The right hemisphere is more strongly engaged during temporal perception, whereas evidence linking left hemisphere damage to impairments in duration discrimination is limited (Harrington et al., 1998). Although the left hemisphere may support related functions, such as sustained attention, it does not appear to contain specialized timing mechanisms (Harrington et al., 1998).

The parietal cortex integrates and aligns sensory and motor information, as well as external stimuli, across various time scales, and therefore plays a role in action control, spatial reference, and planning movements (Fontes et al., 2016). Transcranial magnetic stimulation (TMS) and fMRI studies show activation of the right posterior parietal and intraparietal cortices, often in cooperation with the inferior frontal gyrus, during timing tasks, supporting adaptation and estimation of duration (Fontes et al., 2016). The parietal cortex, especially the right hemisphere, participates in rhythm discrimination and in covert shifts of attention toward temporal stimuli, strengthening the idea that time perception engages attentional networks (Harrington et al., 1998). Anatomical connections between the inferior parietal cortex and the basal ganglia likely serve as pathways for relaying temporal information to core timing circuits. Even so, overlap between parietal activation during temporal and non-temporal tasks complicates interpretation, suggesting that more refined tasks are needed to tease apart these functions (Harrington et al., 1998).

The insular cortex, or insula, has emerged as a key structure in time processing, particularly in relation to subjective temporal experience. It functions as an integrative hub that combines interoceptive, emotional, and cognitive signals, thereby contributing to the internal representation of time (Zhang et al., 2024). Converging evidence from neuroimaging and meta-analyses indicates that the insula is consistently activated across a wide range of timing tasks and durations, showing its role as part of a core timing network alongside the supplementary motor area (Teghil and Wittmann, 2025). Rather than acting as a dedicated 'clock', the insula appears to encode the subjective passage of time

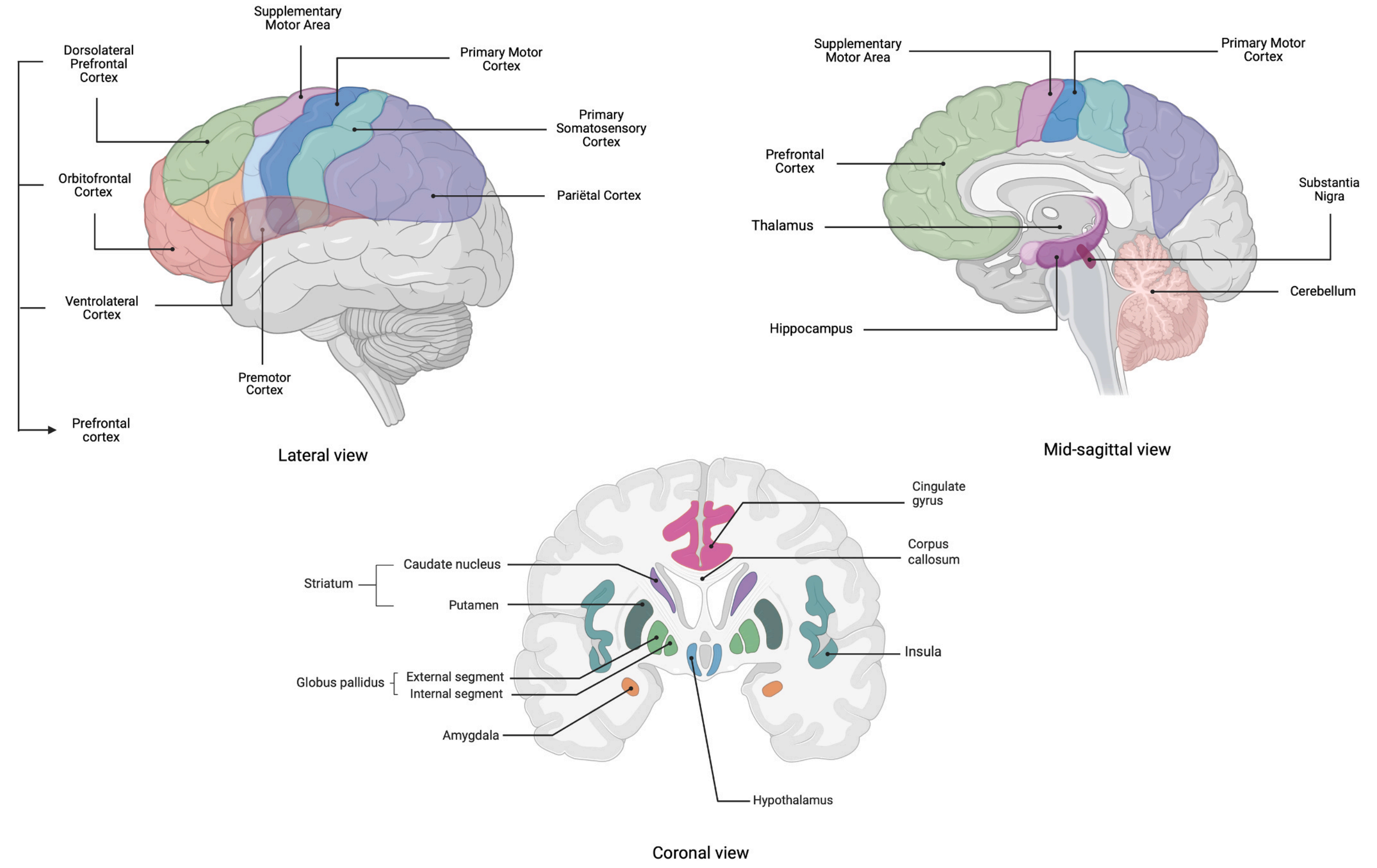

Fig. 4. Brain regions involved in time processing across anatomical perspectives (lateral, mid-sagittal, and coronal views). Time processing relies on a distributed network of cortical and subcortical structures. The prefrontal cortex supports attention, working memory, and temporal decision-making, while the supplementary motor area and premotor cortex contribute to motor timing and internally generated rhythms. The parietal cortex integrates sensory and temporal information, with the right parietal regions particularly involved in duration estimation and discrimination. Subcortically, the basal ganglia, especially the striatum, play a central role in interval timing, with dopaminergic input modulating temporal precision. The cerebellum contributes to fine temporal control and motor coordination, particularly for sub-second timing. The hippocampus is involved in supra-second timing and the temporal organization of memory, whereas the insula is associated with interoceptive awareness and subjective time perception. These regions interact through cortico-striatal and cortico-cerebellar circuits, forming an integrated system for temporal processing that is disrupted in Parkinson's disease.

through the accumulation and integration of bodily signals, linking temporal perception to interoceptive awareness and emotional state (Teghil and Wittmann, 2025). In PD, this role may be disrupted: reduced insular activation during encoding of supra-second durations have been reported in patients in the L-Dopa OFF state (Harrington et al., 2011), potentially based on impaired integration of internal signals during temporal processing. Together, these findings position the insula as a linking hub between bodily states and the experience and estimation of time, particularly for cognitively mediated and supra-second intervals.

The basal ganglia and cerebellum form two areas that support different but complementary aspects of time processing. Evidence from lesion, pharmacological, and imaging studies indicates that the basal ganglia regulate both motor and cognitive timing across millisecond-to-second intervals. Dopaminergic modulation within striatal circuits influences the perceived passage of time: agonists accelerate the internal clock, whereas antagonists slow it down, producing predictable shifts in interval judgments (Koch et al., 2009). In PD, degeneration of the nigrostriatal pathway and related cortical projections leads to variable timing performance, particularly when tasks require cognitive control, working memory, or attention, indicating that basal ganglia dysfunction most strongly affects longer, cognitively mediated intervals rather than short automatic ones (Koch et al., 2009). The basal ganglia appear to act as a threshold or gating system that regulates when motor or cognitive responses are initiated (Ivry and Spencer, 2004). Through reciprocal connections with the supplementary motor and prefrontal cortices, these circuits stabilize rhythmic behavior and sequence timing by ensuring that actions or updates occur only once neural activity crosses a specific activation threshold (Ivry and Spencer, 2004; Harrington et al., 1998). In this framework, dopamine adjusts the sensitivity of these thresholds, explaining how depleted dopamine levels delay initiation and distort the perception of elapsed time in PD. Evidence from deep brain stimulation affirms the involvement of basal ganglia circuits in interval timing. For example, modulation of subthalamic nucleus activity has been shown to alter timing performance in a frequency-dependent manner, indicating that intact oscillatory activity within cortico-basal ganglia loops contributes to better temporal processing (Wojtecki et al., 2011).

The cerebellum, by contrast, ensures temporal precision on the sub-second scale and refines the fine-grained structure of movement (Koch et al., 2009; Ivry and Spencer, 2004; Mannarelli et al., 2023; Claassen et al., 2013). Cerebellar lesions increase variability in both motor and perceptual timing, even when no overt movement is required, showing its role in encoding the duration of discrete events (Koch et al., 2009; Mannarelli et al., 2023). Neurophysiological and stimulation studies indicate that the lateral cerebellum governs the timing of successive, event-based movements such as finger tapping, whereas continuous movements relying on ongoing sensory feedback are less affected (Claassen et al., 2013). This aligns with the cerebellum's role in predictive control and sensorimotor integration: adjusting movement parameters and anticipating temporal contingencies (Mannarelli et al., 2023). Moreover, cerebellar-prefrontal loops contribute to attentional and executive aspects of timing, contributing to the idea that the cerebellum participates not only in automatic temporal encoding but also in the cognitive regulation of timing through its connections with the prefrontal cortex (Mannarelli et al., 2023). The cerebellum further contributes to temporal precision by means of coordinated processing across the cerebellar cortex and deep cerebellar nuclei. Purkinje cells, the sole output neurons of the cerebellar cortex, appear to play a role in learned temporal control, as experimental and computational work suggests that they can acquire and express precisely timed pauses in firing that match expected stimulus intervals (Majoral et al., 2020; Ricci et al., 2023). Transient changes in Purkinje cell intrinsic excitability during an early post-learning window are necessary for successful consolidation of cerebellum-dependent motor memory (Seo et al., 2025). Granule cells and parallel fiber activity may provide temporally patterned input to this process, although whether timing is primarily represented in upstream granule-cell population dynamics or within Purkinje cells themselves remains debated (Ricci et al., 2023). Downstream, the deep cerebellar nuclei transform convergent Purkinje-cell inhibition into temporally precise output, allowing cerebellar signals to shape the fine timing of movement with millisecond-level precision (Gauck and Jaeger, 2000; Gilbert and Rasmussen, 2025). Together, basal ganglia and cerebellar systems interact with cortical networks to form a distributed timing architecture: the cerebellum refines the precision of short, event-based intervals, while the basal ganglia coordinate longer, internally generated or attention-dependent durations (Koch et al., 2009; Ivry and Spencer, 2004; Harrington et al., 1998; Mannarelli et al., 2023; Claassen et al., 2013). Disturbance within either circuit produces characteristic patterns of temporal error (variability and lag with cerebellar dysfunction and hastening or underestimation with basal ganglia impairment), illustrating their complementary roles in maintaining accurate temporal control.

The striatum is important within cortico-striatal circuits modulated by dopamine. Neurophysiological studies show that striatal neuronal populations dynamically encode elapsed time, with activity patterns adapting to task demands and interval ranges (Rolando et al., 2024). These representations are flexible rather than fixed, referring to the view that the striatum contributes to temporal prediction and action timing through distributed population activity rather than a single clock mechanism.

Beyond these circuits, the hippocampus also contributes to time processing (Fontes et al., 2016). Traditionally associated with episodic memory, the hippocampus represents the temporal context of events and integrates sequences across time, encoding temporal organization through “time cells” that track time intervals during tasks (Fontes et al., 2016). Neuroimaging and lesion studies demonstrate hippocampal engagement during supra-second timing tasks, especially those involving attention, working memory, and sustained estimation (Maniadakis and Trahanias, 2014b; Banquet et al., 2021; Eichenbaum, 2017; Lee et al., 2020; Howard and Eichenbaum, 2015). The hippocampus interacts with regions such as the entorhinal cortex and CA1–3 fields. Cognitive functions are important for constructing internal representations of duration and for distinguishing between different moments in time, providing support for the role of the hippocampus in informing a temporal framework for organizing experiences (Maniadakis and Trahanias, 2014b; Banquet et al., 2021; Eichenbaum, 2017; Lee et al., 2020; Howard and Eichenbaum, 2015). In PD, hippocampal involvement may help explain timing impairments that cannot be fully accounted for by basal ganglia dysfunction alone. Cognitive timing tasks that rely on memory for duration or retrospective estimation are disrupted in PD. Structural and functional changes in the hippocampus and its circuits could affect the ability to maintain and retrieve temporal information (Maniadakis and Trahanias, 2014b; Banquet et al., 2021; Eichenbaum, 2017; Lee et al., 2020; Howard and Eichenbaum, 2015). This supports the view of time processing as a distributed function, with interactions between striatal, prefrontal, and hippocampal systems. Such a perspective may also clarify why dopamine replacement improves performance on motor-based timing tasks, but has limited benefit for cognitively driven timing processes (Maniadakis and Trahanias, 2014b; Banquet et al., 2021; Eichenbaum, 2017; Lee et al., 2020; Howard and Eichenbaum, 2015).

Slowness reflects brain-specific mechanisms rather than a unitary deficit (Jahanshahi et al., 1993). demonstrated that delayed responses are not unique to PD and occur in Huntington’s disease (HD) and diseases affecting cerebellar function. However, the mechanisms behind this slowing differ across disorders: in PD, slowness is linked to reduced agonist muscle bursts; in cerebellar disease, to abnormal antagonist timing; and in HD to impaired buildup of muscle activity (Jahanshahi et al., 1993). This suggests that while behavioral slowness is broadly observed across conditions affecting brain function, the underlying neural components – and thus the role of different brain areas – are disorder-specific. The extent to which dopamine modulates these region-dependent mechanisms is considered in a later section.

Although this review primarily focused on behavioral, cognitive, and functional mechanisms of temporal dysfunction in PD, structural neuroimaging findings may also argue for a network-level account. MRI studies in PD, particularly in relation to cognitive decline, have reported gray-matter alterations in frontal, temporal, parietal, hippocampal, and subcortical regions, as well as white-matter and structural-connectivity abnormalities involving large-scale cortico-subcortical networks (Hanganu and Monchi, 2016; Sun et al., 2026; Zarkali et al., 2024; Schröter et al., 2025). These findings are relevant because temporal processing is thought to depend on distributed interactions among basal ganglia-frontal circuits, the cerebellum, hippocampal memory-related regions, and parietal associative areas (Su et al., 2022). In line with this, recent neuroimaging studies further demonstrate that cognitive decline in PD is associated with alterations of dynamics and spontaneous neuronal activity in large-scale brain networks, including the default mode and executive control networks, reflecting disrupted integration of neural activity across distributed systems (Fiorenzato et al., 2024). However, direct structural imaging studies specifically examining time perception in PD remain limited. Therefore, structural correlates were not a primary focus of the present review, but they likely form an important part of the broader neurobiological framework underlying impaired temporal processing in PD.

To summarize, time processing relies on a distributed but closely integrated network that spans cortical, subcortical, and cerebellar regions. The prefrontal and parietal cortices provide top-down control, the basal ganglia support temporal organization and the initiation of timed actions, and the cerebellum ensures precision and rhythmic consistency. The hippocampus contributes to the temporal structure of memory and links timing to broader cognitive functions. Communication among these regions, through cortico-striatal, cortico-cerebellar, and hippocampal pathways, enables the brain to align perception, cognition, and movement across different temporal networks. Disruption within any of these circuits, as seen in PD, can therefore alter both motor and cognitive aspects of timing, depending on which networks are most affected (Koch et al., 2009; Ivry and Spencer, 2004; Harrington et al., 1998).

3.3.2. *The role of dopamine in time processing*

PD is characterized by the progressive degeneration of neuromelanin-containing dopaminergic neurons in the substantia nigra pars compacta, resulting in dopamine depletion within the striatum and disruption of basal ganglia circuits. This disruption contributes not only to motor symptoms such as bradykinesia but also to impairments in time processing (Haining and Achat-Mendes, 2017; Blandini et al., 2000). These timing deficits are especially pronounced during tasks involving internally generated movements, suggesting that bradykinesia may partly originate from broader temporal dysfunction.

Experimental studies elaborate on the role of dopamine in timing (Fig. 5). Dopamine depletion has been associated with increased temporal uncertainty and more cautious responses during timing tasks (Tomassini et al., 2019). Other studies report systematic distortions in

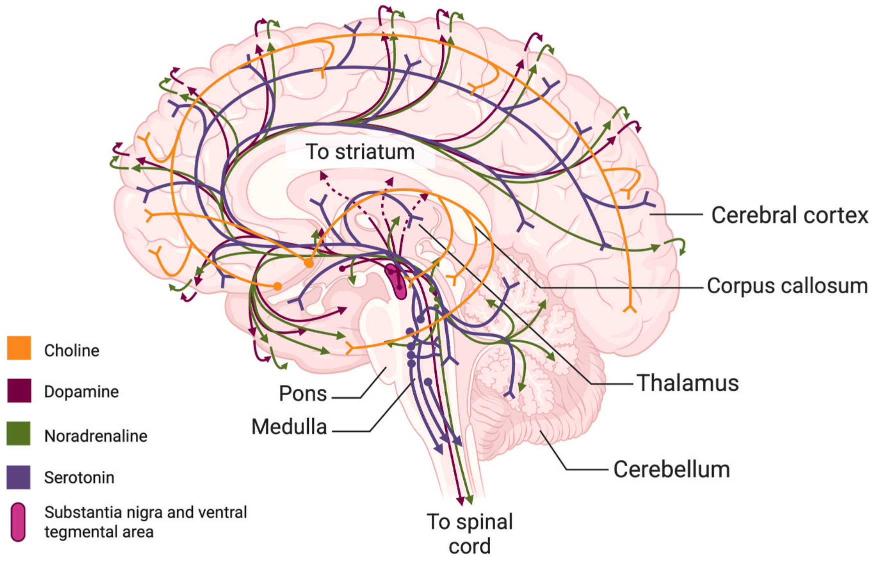

Fig. 5. Distribution of key neurotransmitter systems involved in time processing in the human brain. Major neuromodulatory systems, including dopamine, noradrenaline, serotonin, and acetylcholine, project widely from brainstem and basal forebrain nuclei to cortical and subcortical regions. Dopaminergic pathways partly originating from the substantia nigra densely innervate the striatum and cortex, playing a central role in interval timing, reward prediction, and temporal precision. Noradrenergic projections modulate arousal and attention, thereby influencing pacemaker rate and temporal variability. Serotonergic pathways contribute to mood, impulsivity, and temporal decision-making. Cholinergic projections from the basal forebrain support attention and working memory, both critical for accurate time perception. The widespread and overlapping distribution of these neurotransmitter systems highlights their integrative role in modulating cortical-subcortical timing networks. Dysregulation of these systems, particularly dopaminergic depletion in Parkinson's disease, contributes to the timing deficits observed across cognitive and motor domains.

interval estimation, including overestimation of short intervals and slower timing mechanisms (Terao et al., 2021; Parker et al., 2013).Animal studies continue on this relationship, showing that blocking dopaminergic transmission slows response time, consistent with a decelerated internal clock.

Behavioral studies in PD also suggest alterations in interval timing mechanisms. Patients often underestimate longer intervals while overproducing shorter durations (Terao et al., 2021; Coull et al., 2012; Magalhães et al., 2018; Fung et al., 2021; Jones and Jahanshahi, 2011). These patterns likely depend on task demands and cognitive context rather than a uniform change in clock speed (Terao et al., 2021; Honma et al., 2016; Malapani et al., 1998; Riesen and Schnider, 2001; Jones and Jahanshahi, 2011; Smith et al., 2007; Miyawaki, 2022). Dopaminergic dysfunction appears to interact with attentional and working memory processes that influence temporal estimation and decision-making Wearden et al. (2008).

Honma et al. (2016) observed that reduced striatal dopamine transporter availability was associated with underestimation of intervals longer than 10 s during production tasks, possibly reflecting impaired internal counting strategies. Similar findings have been reported by Parker et al. (2013) and Terao et al. (2021), who reported overestimation of short intervals and underestimation of longer ones. Variability in timing performance was especially pronounced in production and reproduction tasks that place greater demands on cognitive resources.

Dopaminergic therapy can partially improve timing performance, particularly in short-interval or motor-based tasks. For example, levodopa has been shown to reduce timing variability and improve interval production (Malapani et al., 1998; Merchant et al., 2008a). Dopamine replacement may also normalize the timing of repetitive sequential movements by reducing inter-response interval variability and improving reaction and movement times through basal ganglia-thalamocortical circuits Pastor et al. (1992). Together, these findings imply that dopaminergic signaling contributes to both central timekeeping and motor implementation processes.

Collectively, these findings give the impression that timing distortions in PD, such as overestimation of short intervals and underestimation of longer intervals, likely arise from interactions between dopaminergic dysfunction and cognitive processes such as attention and working memory. Reduced attentional engagement may limit the accumulation of temporal information, whereas altered dopaminergic modulation can influence pacemaker dynamics and temporal estimation.

3.3.3. *Limitations of dopaminergic therapy*

Although levodopa provides substantial relief for motor symptoms in PD, its effects on time processing are selective and context dependent. There is a dissociation between improvements in motor timing and persistent deficits in cognitive or perceptual timing, making dopamine's contribution to time processing strongly dependent on task demands and timescale.

Koch et al. (2008) showed that sub-second timing, typically automatic and supported by cerebellar circuits, remains largely intact in PD, whereas supra-second timing, requiring attention, working memory, and cognitive control, is markedly impaired. These findings support a dual-system model of time perception, with a cerebellar-based system mediating short automatic intervals, and a dopamine-sensitive frontostriatal system governing longer, cognitively mediated durations (Koch et al., 2008), though partial overlap between these systems may explain motor timing disturbances such as bradykinesia. Importantly, dopaminergic therapy did not reliably improve supra-second timing and in some cases worsened performance (Koch et al., 2008).

Koch et al. (2009) reported that auditory timing deficits, including impaired gap detection and interval discrimination, persisted despite dopaminergic therapy, suggesting cortical or attentional contributions Bernardinis et al. (2019) and Harrington et al. (2011) similarly found that while sub-second perceptual timing was generally preserved, subtle impairments emerged around the one-second threshold and persisted as striatal dysfunction despite therapy and dopamine levels being pharmacologically restored (Elsinger et al., 2003). found reduced accuracy and greater variability on a paced finger-tapping task in PD, accompanied by decreased activation in the sensorimotor cortex, cerebellum, and frontostriatal circuits. Dopamine therapy normalized much of this neural activity, but precise motor timing deficits persisted, suggesting a specific impairment in temporal control rather than generalized motor slowing (Elsinger et al., 2003). In line with this, Jones et al. (2011) showed that chronically treated PD patients exhibit hastened tapping at very short intervals (250 ms), particularly during the continuation phase, resembling motor festination, whereas de novo patients and controls tap slightly behind the beat. At longer intervals, accuracy declines across all groups, while variability becomes the more prominent deficit, especially in de novo patients at 2000 ms. Acute dopaminergic state ("ON" vs. "OFF") did not significantly alter performance, suggesting that long-term medication effects or disease progression, rather than immediate levodopa state, shape these timing patterns.

Neuroimaging studies converge on the idea that dopamine alters activation patterns without consistently restoring behavior (Dušek et al., 2012; Avanzino et al., 2016; Coull et al., 2011). Dušek et al. (2012) found that dopamine replacement therapy increases precuneus activity during reproduction of longer intervals, whereas activity declined in the dopamine-deprived state. Because the precuneus is involved in episodic memory retrieval and belongs to the default mode network, this pattern was interpreted as compensatory recruitment of mnemonic resources rather than restoration of an internal clock mechanism. Although this modulation was associated with a trend toward improved temporal reproduction, timing deficits persisted (Dušek et al., 2012). The complexity of dopaminergic effects is further illustrated by the "dopamine overdose" hypothesis described by Jones et al. (2008), in which dopamine therapy improved motor symptoms but disrupted cognitive timing performance, such as time production, arguing domain-specific benefits alongside impairment in other aspects of timing (DiMarco et al., 2023). similarly found no significant group-level differences in interval timing between medicated and unmedicated PD states but reported that impulse control disorder and depression influenced task performance, emphasizing the importance of individual variability in timing deficits and their neurochemical correlates (DiMarco et al., 2023). Zhang et al. (2016) have the same view on this, concluding that timing deficits in PD are not solely attributable to distortions in temporal perception. Altered decision-making processes, including premature responding and reduced sensitivity to temporal information, also contribute to performance deficits in timing tasks.

Timing abnormalities in PD have been attributed to a dopamine-modulated internal clock, with deficits particularly evident in time estimation, reproduction, and synchronization-continuation tasks involving internally generated movements (Harrington and Haaland, 1999). However, time deficits may not be unique to PD but may also occur in healthy aging and HD (Jahanshahi et al., 1993; Wild-Wall et al., 2008). In a time estimation task, young participants outperformed all other groups, whereas older controls, PD patients, and HD patients showed no significant differences despite differing degrees of dopaminergic and frontostriatal disruption. This proposes that even subtle frontostriatal dysfunction, including that associated with aging, may impair time processing. Notably, although all older groups performed worse at baseline compared to young participants, they demonstrated similar improvements across repeated trials, indicating preserved learning capacity despite impaired temporal processing (Wild-Wall et al., 2008).

Overall, dopaminergic therapy provides symptomatic relief, but its benefits on time processing are limited and highly task dependent. Levodopa tends to improve short-interval, motor-based tasks but has little effect, and may even increase variability, in cognitively demanding supra-second timing tasks. A similar pattern emerges when considering non-motor symptoms in late-stage PD (Rosqvist et al., 2018). Even in patients with minimal motor benefit, dopaminergic therapy alleviates certain neuropsychiatric symptoms such as mood and apathy, while sleep, cognition, and autonomic function remain largely unaffected (Rosqvist et al., 2018). This broader clinical profile mirrors the partial improvements observed in timing tasks, suggesting that dopamine can influence timing performance but cannot fully restore it due to the multisystem nature of PD.

3.3.4. Multiscale and compensatory models of time processing

While dopaminergic dysfunction of the basal ganglia is central to temporal deficits in PD, these impairments involve other connected brain regions as well. Multiscale and compensatory models of time processing help explain the variability in timing behavior observed in PD by presenting neural systems that operate at different temporal scales and by recognizing the brain's ability to recruit alternative circuits when primary systems are compromised.

Koch et al. (2009) proposed a four-tiered framework of time processing, dividing time perception into microseconds, milliseconds, seconds, and circadian rhythms, each supported by separate but overlapping neural systems. Millisecond-level timing, essential for motor control and sensorimotor synchronization, is largely attributed to cerebellar function, while timing in the seconds range relies more heavily on the basal ganglia, particularly the striatum. Longer, consciously estimated intervals additionally involve the posterior cerebellum and prefrontal cortex. This can explain that PD-related timing deficits may vary depending on the temporal scale of the task and the specific circuits involved (Koch et al., 2009).

Support for this multiscale and compensatory organization comes from studies demonstrating differential impairments depending on task demands. For instance, Behroozmand and Johari (2019) examined how patients with PD respond to temporally predictable versus unpredictable sensory cues during both speech and hand movement tasks. Strikingly, motor timing deficits were only observed when stimuli followed a predictable temporal structure. Compared to controls, PD patients showed slower initiation and cessation responses under predictable conditions. In contrast, no significant differences emerged when stimuli were presented in an unpredictable sequence. This suggests that predictive timing, thought to rely on dopaminergic frontostriatal loops and internal forward models, is impaired in PD, whereas more reactive, stimulus-driven timing mechanisms may remain relatively intact. These reactive processes likely involve cerebellar and parietal regions, which are less dependent on dopamine and may serve a compensatory role (Behroozmand and Johari, 2019). The study also found similar timing profiles across speech and hand movements, implicating shared substrates in the cortico-basal ganglia network, including the pre-supplementary motor area and inferior frontal gyrus (Behroozmand and Johari, 2019).

Additional evidence for compensatory recruitment comes from neuroimaging studies, which show both hypoactivation and hyperactivation in PD, depending on task type and context. Hypoactivation is frequently observed in the striatum, particularly in the caudate nucleus, and in the cerebellum during predictive timing tasks, especially in early-stage or off-medication patients, supporting the idea that cerebellar contributions to internally generated timing in PD are limited regarding predictive timing tasks. Reduced activity is also noted in the sensorimotor cortex and medial premotor areas during motor timing tasks. At the same time, the cerebellum shows hyperactivation during non-predictive or externally paced tasks, such as synchronization-continuation tapping, where it may act as a compensatory system to support impaired basal ganglia output. This apparent inconsistency suggests that cerebellar involvement in PD is task- and context-dependent, influenced by medication state and timing demands (Bareš et al., 2019; Vikene et al., 2019). As Bareš et al. (2019) point out, the cerebellum not only facilitates motor coordination but also contributes to precision timing and temporal integration across both cognitive and motor domains. Thus, while the cerebellum shows reduced engagement in predictive timing, it is a candidate for compensating in other timing contexts (Bareš et al., 2019).

In rhythm perception tasks, PD patients show hyperactivation in cortical regions such as the planum temporale and inferior parietal lobule, likely due to an increased perceived complexity of rhythmic input resulting from basal ganglia dysfunction (Jones and Jahanshahi, 2011). Additionally, Breska and Ivry (2018) proposed a dissociation between cerebellar-based isolated interval timing and basal ganglia-based rhythmic timing, challenging the notion of a unified timing mechanism. Building on this, Cameron et al. (2016) observed that dopaminergic medication selectively improved performance on beat-based rhythms while impairing non-beat-based rhythm discrimination in PD. This suggests that dopamine does not uniformly enhance timing abilities but may bias the system toward certain rhythmic structures, further supporting the idea of specialized and differentially modulated timing networks.

Altogether, a multiscale and compensatory perspective shows that when dopamine-dependent striatal timing mechanisms are disrupted in PD, other brain regions, including the cerebellum, prefrontal cortex, and parietal areas, can support timing behavior, particularly in tasks that rely on perceptual or reactive timing. The nature and structure of the timing task, whether sub-second or supra-second, implicit or explicit, discrete or continuous, shapes which circuits are engaged and how compensation unfolds. This framework provides an understanding of the heterogeneity of timing deficits in PD.

3.3.5. *Beyond dopamine*

While dopaminergic dysfunction is central to the motor and cognitive manifestations of PD, converging evidence indicates that other neurotransmitter systems contribute to timing deficits. Executive functions – attention, working memory, planning, and cognitive flexibility – are foundational for accurate time processing, because they support the monitoring, updating, and comparison of internal time representations (Kudlicka et al., 2011; Dirnberger and Jahanshahi, 2013).

The “dopamine overdose” hypothesis helps explain why dopaminergic therapy yields mixed cognitive outcomes: while dopamine replacement can improve functions mediated by dorsal striatal-dorsolateral frontostriatal circuits (e.g., set shifting, planning), it may concurrently overstimulate relatively spared ventral striatal-orbitofrontal/limbic circuits, producing deficits in reversal learning, reward-based decision-making, and probabilistic classification. Because these executive operations scaffold temporal judgments, such differential medication effects translate into variable performance across time paradigms (Jones et al., 2008; Bohnen and Albin, 2011; Dirnberger and Jahanshahi, 2013).

PD is not a purely dopaminergic disorder. Multiple neurotransmitter systems (Fig. 5) interact to shape both motor and non-motor features, and many converge on executive operations that govern timekeeping (Barone, 2010). Cholinergic degeneration in basal ganglia forebrain projections to prefrontal and temporal cortices correlates with poorer attention, set shifting, and working memory; anticholinergic challenge selectively worsens these processes, whereas cholinesterase inhibition can improve cognition in PD dementia. Beyond cognition, cholinergic loss contributes to apathy, depression, autonomic dysregulation, and sleep problems – states that further erode attentional engagement and motivational drive during timing tasks (Barone, 2010).

Experimental animal studies provide additional insight (Zhang et al., 2019). demonstrated that muscarinic receptor blockade with scopolamine in mice degraded fixed interval timing performance, flattening temporal response curves and increasing premature responding. These effects occurred without altering dopaminergic ramping activity in the medial frontal cortex, suggesting that cholinergic dysfunction specifically disrupts attentional and salience-processing components of timing rather than the clock-like dynamics often attributed to dopamine. In PD, degeneration of basal forebrain cholinergic projections is therefore likely to exacerbate supra-second timing deficits by impairing top-down attentional modulation and stimulus gating, even when dopaminergic therapy improves motor symptoms. This supports the notion of dissociable but interacting neuromodulating contributions: dopamine directly influences an internal clock and memory component, whereas acetylcholine biases preparatory attention and impulse control during temporal estimation.

Serotonergic alterations in PD influence both non-motor symptoms and higher cognitive processes relevant to time processing. Serotonergic changes (notably in cortex and basal ganglia) are mainly linked to mood, anxiety, sleep, pain, and inhibitory control, with receptor-specific effects on dopamine release that can destabilize striatal signaling. Stimulation of the serotonergic system has been associated with improvements in anxiety, depression, constipation, and pain, whereas reduced serotonergic transmission leads to deficits in cognition, executive function, and increased fatigue. Because executive control, attention, and sustained cognitive engagement are essential for accurate time processing, serotonergic degeneration in PD may indirectly compromise time processing by weakening these higher order functions (Barone, 2010).

Noradrenergic degeneration of the locus coeruleus impairs arousal and sustained attention, with receptor-specific consequences, thereby influencing timing tasks (Barone, 2010). Glutamatergic hyperactivity within basal ganglia circuits contributes to motor symptoms and excitotoxic stress; modulation can improve motor signs and, in some contexts, executive performance and mood. GABAergic pathways that shape basal ganglia output become dysregulated with dopamine loss and long-term therapy, variably affecting non-motor symptoms (pain, sleep) that impinge on attentional resources. Adenosine A2A receptors, antagonistic to D2 signaling and interacting with glutamatergic inputs, modulate indirect pathway function and likely influence sleep and cognition (Barone, 2010).

3.3.6. *Genetics and time processing in Parkinson’s disease*

Genetic variability may also contribute to heterogeneity in temporal dysfunction in PD, although direct evidence linking specific variants to interval timing deficits remains limited. Studies about PD genetics indicate that both rare pathogenic variants and common risk variants, including single-nucleotide polymorphisms and cumulative polygenic risk, can influence disease susceptibility, age at onset, cognitive impairment, progression, and treatment response (Reed et al., 2019; Funayama et al., 2023). Variants in genes such as *SNCA*, *LRRK2*, *GBA1*, *MAPT*, *PRKN*, and *PINK1* may therefore modulate the neural and cognitive systems relevant to temporal processing (Reed et al., 2019; Funayama et al., 2023). In addition, polymorphisms in circadian clock genes, including *ARNTL*, *PER1*, and *CLOCK*, have been associated with distinct PD phenotypes, motor fluctuations, and sleep-related disturbances (Yalçin et al., 2025), suggesting that alterations in biological timing systems may further contribute to symptom heterogeneity. Although these findings do not permit definitive conclusions regarding time processing specifically, they support the possibility that underlying genetic variability partly shapes the diversity of motor and cognitive timing abnormalities observed in PD. Future studies examining associations between PD-related genetic variants and specific timing phenotypes may therefore help provide a more comprehensive neurobiological framework for temporal dysfunction in PD.

3.4. *Additional insights into temporal cognition and Parkinson’s disease*

Beyond core timing deficits in PD, recent research has identified distinct modulators of temporal cognition, including executive demands and sensory integration. This section briefly mentions emerging mechanisms and therapeutic possibilities that expand our knowledge of timing in PD.

3.4.1. *Temporal cueing, cognitive control, and therapeutic interventions*

Recent studies have expanded our understanding of how PD affects temporal cognition, particularly concerning task complexity, medication status, and broader cognitive functions (Zokaei et al., 2021). investigated temporal orienting – the ability to use time-based cues to guide action – and found that while PD patients could use these cues to improve motor response times comparably to healthy controls, they struggled with perceptual discrimination in the presence of distractors.

Dopaminergic medication reversed this deficit, suggesting a role of the nigrostriatal system in managing tasks that require both temporal cueing and inhibition of distractors, altogether suggesting that PD-related impairments go beyond motor symptoms, including executive functions (Zokaei et al., 2021).

Expanding on this association between timing and executive function, (Honma et al., 2021) demonstrated that duration feedback training improved accuracy and reaction times in PD patients performing GO/NO-GO and Stroop tasks. They show an accuracy-reaction time trade-off, with training enhancing response inhibition and cognitive control, implicating the potential of duration-based cognitive interventions in PD rehabilitation; however, further research is needed to assess their long-term benefits (Honma et al., 2021).

Research on the interaction between motor timing and gait disorders in PD provides possible therapeutic avenues (Bella et al., 2015). demonstrated that auditory cueing could improve gait synchronization in PD patients, suggesting its value for enhancing motor timing. This sensorimotor timing intervention could also extend beyond motor tasks, offering therapeutic potential for non-motor benefits, such as cognitive impairments, through individualized rehabilitation approaches (Bella et al., 2015).

Complementary to this possible therapeutic option, Su et al. (2022) conducted a large-scale online study and explored how time processing measures might serve as diagnostic tools. The study resulted in patients exhibiting impaired sensitivity in distinguishing stimulus durations in both auditory and visual domains. Although the online methodology allowed for a larger sample size, variability in participant environments introduced limitations (Su et al., 2022). Nevertheless, the study demonstrated that web-based timing assessments can effectively capture subtle cognitive deficits in PD at scale. Importantly, such cross-modal perceptual disruptions, likely reflecting basal ganglia and cortical timing dysfunction, may represent early biomarkers of cognitive decline and disease progression (Su et al., 2022).

3.4.2. Sensory timing

Most timing tasks described thus far, such as tapping, interval production, and reproduction tasks, require overt motor output, making it difficult to separate perceptual from motor contributions to timing performance. To isolate the sensory component of time processing, some studies have used motor-independent tasks such as temporal order judgment (TOJ) tasks (da Silva et al., 2015; Nishikawa et al., 2015). TOJ tasks, which require participants to determine which of two closely spaced sensory stimuli occurred first, provide a means of isolating perceptual timing from motor execution, since accuracy, rather than response speed, is evaluated. This distinction is particularly relevant in PD, where bradykinesia could otherwise confound interpretations of timing performance.

Da Silva et al. (2015) addressed this by comparing PD patients on dopaminergic medication with healthy elderly and young controls. They found that although PD participants showed slower improvement in detecting temporal intervals, their overall TOJ accuracy remained comparable to that of controls. This indicates that temporal sequencing of sensory events is largely preserved, while processing efficiency and sensory integration are slowed, consistent with a perceptual rather than a motor deficit.

Nishikawa et al. (2015) examined tactile TOJ across young, elderly, and PD participants and similarly found that aging, but not PD, reduced temporal resolution. PD-specific effects were limited to delayed response initiation, reinforcing that dopaminergic loss does not directly impair the perceptual timing mechanism itself. Taken together, these findings argue that PD-related dopaminergic degeneration amplifies age-related slowing in sensory integration without fundamentally disrupting the ability to sequence sensory events in time. TOJ performance, therefore, appears to rely more on cortical sensory networks, particularly parietal and temporal regions involved in attentional modulation, than on basal ganglia circuits alone. The preservation of TOJ accuracy despite motor slowness supports the view that timing disturbances in PD stem primarily from sensory-cognitive inefficiency rather than from motor dysfunction per se (da Silva et al., 2015; Nishikawa et al., 2015).

3.5. Distinguishing bradyphrenia from bradykinesia: mechanisms and markers

Bradyphrenia, or cognitive slowing, is a well-recognized phenomenon in PD that occurs alongside motor slowness (bradykinesia) but constitutes a distinct cognitive impairment (Mayeux et al., 1987). described bradyphrenia as a slowing of cognitive processing, characterized by deficits in attention, concentration, and mental initiative, rather than memory loss associated with dementia. Although early theories linked bradyphrenia to dopamine deficiency in parallel with bradykinesia, subsequent research suggests a stronger association with noradrenergic dysfunction. Elevated levels of norepinephrine metabolites have been correlated with impairments in vigilance through attention, arousal, and alertness, demonstrating a neurochemical origin of bradyphrenia beyond purely dopaminergic or motor mechanisms (Barone, 2010).

Vlagsma et al. (2016) further clarified the distinction between bradyphrenia and bradykinesia, noting that although they co-occur, they represent separate constructs. Reaction time (RT) studies can conflate mental and motor slowing, but more refined analyses differentiate between central cognitive processing (involving the prefrontal and motor cortices) and peripheral motor execution (Vlagsma et al., 2016). While overall reaction times are typically prolonged in PD, the outcome is influenced by motor slowness. Tasks isolating cognitive execution times show that there are deficiencies primarily from this field, supporting the notion that bradyphrenia reflects a central cognitive deficit rather than being a mere extension of motor dysfunction. Furthermore, bradyphrenia can impair performance on speed-based neuropsychological tasks yet often leaves global cognitive test outcomes relatively unaffected. This dissociation reinforces the specificity of bradyphrenia as a central processing deficit rather than a marker of general cognitive decline (Vlagsma et al., 2016).

In line with this distinction, Jahanshahi et al. (1992) showed that patients with PD are slower than controls in both simple and choice RT tasks, with a greater deficit in the latter. Importantly, this slowing could not be explained by fatigue or reduced arousal but was linked to delayed response initiation and difficulties in making full use of advanced information within short preparatory intervals. While motor execution times were uniformly slower, they were unaffected by pre-cueing, suggesting intact motor programming. These findings support the view that bradyphrenia reflects slowed central processing and initiation, whereas bradykinesia is more closely tied to motor output. Together, such RT paradigms demonstrate that the two forms of slowness are separate yet interacting features of PD.

Electrophysiological evidence further refines this distinction (Low et al., 2002). used lateralized readiness potentials to separate premotor from motor contributions to RT slowing. They found that PD patients showed delayed premotor processing across conditions, even at early disease stages, while motor execution was only prolonged during complex sequential responses. This pattern suggests that bradyphrenia arises from slowed central processes – particularly response selection and initiation – whereas bradykinesia manifests when motor demands increase, consistent with the clinical picture of sequential movement difficulties in PD.

In contrast to reaction time tasks, which integrate cognitive and motor elements, inspection time (IT) tasks isolate early-stage perceptual processing, eliminating the need for motor output (Johnson et al., 2004). demonstrated that PD patients show significantly prolonged IT compared to controls, indicating a perceptual processing deficit consistent with bradyphrenia. This slowing is mainly independent of motor impairment, as IT performance does not correlate with motor severity measures such as the Unified Parkinson’s Disease Rating Scale

(UPDRS), and is partially unresponsive to dopaminergic therapies like levodopa. These findings open alternative therapeutic avenues, such as targeting nicotinic acetylcholine receptors (nAChRs), which may be more effective in treating bradyphrenia (Johnson et al., 2004).

The underlying mechanisms of movement slowness in PD – whether due to deficits in perception, motor programming, or movement initiation – remain under investigation (Carlsen et al., 2013). employed an RT paradigm involving a startling acoustic stimulus (SAS) to bypass cortical initiation pathways and elicit pre-programmed motor responses via subcortical circuits. The SAS markedly reduced RT in PD patients, suggesting that motor programming remains intact, but that initiation processes are delayed (Carlsen et al., 2013). This delay in motor initiation may parallel the slowed cognitive initiation observed in bradyphrenia, supporting a shared underlying deficit in the initiation of action, whether motor or cognitive (Carlsen et al., 2013).

In summary, bradyphrenia in PD represents a distinct cognitive domain of slowing that is mechanistically and pharmacologically separate from bradykinesia. While standard dopaminergic treatments and motor assessments do not capture or improve bradyphrenia, timing tasks such as inspection time offer clearer insight into its nature and point toward non-dopaminergic therapeutic targets.

4. Discussion

This review shows that time processing deficits in PD arise from cognitive and neurophysiological dysfunction (Poewe et al., 2017; Berardelli et al., 2001). Across diverse tasks, patients consistently demonstrate impairments in explicit timing, particularly in tasks that rely on supra-second estimation, working memory, and attentional control. In contrast, implicit timing, sub-second perception, and reactive forms of timing are often relatively preserved, suggesting that PD affects distinct timing systems differently (Terao et al., 2021; Mioni et al., 2018; Szelag et al., 2022; Ivry and Spencer, 2004; Fontes et al., 2016; Mızrak and Oberauer, 2021; Cohn-Sheehy and Ranganath, 2017; Pan and Luo, 2012; Polti et al., 2018; Wearden et al., 2008). This dissociation is supported by neuroimaging evidence showing separable but overlapping networks for automatic versus cognitively controlled timing (Szelag et al., 2022; Rammsayer et al., 2015; Meck et al., 2008; Jones and Jahanshahi, 2014). The evidence gives rise to novel perspectives. Time processing deficits are unlikely to arise solely from dysfunction of an isolated internal timing mechanism, but rather from impairments in how temporal information is integrated and translated into behavior (Merchant et al., 2008b; Narayanan, 2016). Many experimental timing tasks involve not only the estimation of temporal intervals, but also decision-making, memory, and motor execution processes, in other words, the multifactorial nature of temporal performance (Merchant et al., 2008b). This is supported by findings that subgroups of patients exhibit increased variability specifically related to timing, while other cognitive processes remain relatively preserved, suggesting a dissociation between core temporal mechanisms and task-related demands (Merchant et al., 2008b). At the neural level, temporal control of action has been associated with ramping activity (time-dependent ramping) in prefrontal regions, reflecting a gradual build-up of activity over time that supports anticipation and movement initiation (Narayanan, 2016). Such dynamics resemble accumulation-to-bound processes described in the drift-diffusion model, in which information is integrated over time until a response threshold is reached. Although not a model of time perception itself, this framework provides a useful way to distinguish whether observed impairments reflect deficits in temporal representation, altered decision thresholds, or changes in non-decision processes such as motor execution. From this perspective, timing impairments in PD may emerge from disrupted integration of temporal information within fronto-striatal networks, rather than from a single dysfunctional timing mechanism (Merchant et al., 2008b; Narayanan, 2016).

A central theme across studies is the role of the basal ganglia and their dopaminergic modulation. The basal ganglia serve as a core timing hub, integrating sensory, cognitive, and motor information to regulate time processing and movement initiation (Koch et al., 2009; Ivry and Spencer, 2004; Harrington et al., 1998). Dopamine depletion disrupts these frontostriatal circuits that are critical for supra-second and internally generated timing, leading to overestimation or underestimation of intervals depending on task demands (Terao et al., 2021; Honma et al., 2016; Coull et al., 2012; Tomassini et al., 2019; Parker et al., 2013; Magalhães et al., 2018; Fung et al., 2021; Jones and Jahanshahi, 2011). Dopaminergic therapy improves performance in short-interval and motor-based tasks, but its benefits do not generalize to cognitively demanding tasks and may even increase variability, consistent with the "dopamine overdose" hypothesis (Jones et al., 2008; Bohnen and Albin, 2011; Dirnberger and Jahanshahi, 2013). This task- and context-specific pattern, regarding temporal variability, impaired cognitive control, and altered decision-making processes and attention (Singh et al., 2021, 2023; Zhang et al., 2016), shows that PD-related timing deficits cannot be reduced to a uniformly slowed internal clock. Rather, they reflect a dynamic interaction between dopaminergic modulation, cognitive load, and compensatory recruitment of additional neural circuits.

Beyond the basal ganglia, multiple other brain regions and structures emerge as contributors to temporal cognition in PD. The cerebellum appears particularly important for millisecond-level timing and for compensating when striatal circuits are impaired, especially in externally paced or reactive tasks (Koch et al., 2009; Ivry and Spencer, 2004; Mannarelli et al., 2023; Claassen et al., 2013). The prefrontal and parietal cortices support attention and working memory, processes that are consistently implicated in PD-related timing deficits (Fontes et al., 2016; Yin et al., 2019). The hippocampus contributes to supra-second estimation and the temporal organization of memory, which may help explain deficits that cannot be accounted for by dopamine deficiency alone (Maniadakis and Trahanias, 2014b; Fontes et al., 2016; Banquet et al., 2021; Eichenbaum, 2017; Lee et al., 2020; Howard and Eichenbaum, 2015). These findings align with multiscale and compensatory models of timing, in which different brain systems operate across temporal ranges and can partly substitute for one another when primary circuits are compromised.

The reviewed literature also points out that timing deficits are not purely a dopaminergic disorder. Alterations in cholinergic, serotonergic, noradrenergic, and glutamatergic systems affect executive functions and arousal, indirectly shaping temporal cognition (Barone, 2010). This broader neurochemical landscape explains why levodopa alone provides only partial improvements and why individual differences in comorbid symptoms, such as depression or impulse control disorders, influence timing performance.

Bradyphrenia and bradykinesia illustrate how timing impairments manifest across cognitive and motor domains. Although both forms of slowness co-occur, they represent distinct phenomena: bradyphrenia reflects slowed central processing and impaired attentional engagement, whereas bradykinesia reflects deficits in movement initiation and execution (Jahanshahi et al., 1992; Mayeux et al., 1987; Vlagsma et al., 2016; Low et al., 2002). RT and IT tasks, alongside electrophysiological measures, help to disentangle these mechanisms. Importantly, bradyphrenia appears relatively insensitive to dopaminergic therapy, pointing toward non-dopaminergic contributions and highlighting the need for alternative therapeutic targets (Johnson et al., 2004; Carlsen et al., 2013).

Despite consistent findings, several limitations should be noted. Most studies employ small sample sizes, single-task, or cross-sectional designs, limiting generalizability. The heterogeneity of tasks complicates comparisons across studies, as different tasks probe different aspects of timing. Furthermore, the lack of standardized protocols for assessing temporal cognition in PD constrains comparability, interpretation, and hinders the development of clinical applications.

Future research should focus on longitudinal studies to track the evolution of timing deficits across disease stages, multimodal imaging approaches to clarify network-level mechanisms and compensatory recruitment, and pharmacological or neuromodulating interventions targeting non-dopaminergic systems. Greater methodological consistency across studies is needed, as the wide variety of experimental paradigms currently used to assess timing makes it difficult to compare findings across studies and identify specific mechanisms in timing deficits in PD. Standardized task batteries that probe explicit and implicit timing, sub-second and supra-second intervals, and both motor and cognitive timing would facilitate more direct comparisons across studies. In addition, experimental tasks that more clearly separate motor execution from perceptual or cognitive timing components may help clarify how temporal processing deficits relate to clinical phenomena such as bradykinesia and bradyphrenia, and, put together, broaden our view on this multimodal network-level mechanism.

5. Conclusions

This review demonstrates that time processing deficits in PD are not the result of a single dysfunctional mechanism but rather reflect distributed impairments across dopaminergic and non-dopaminergic networks. Explicit timing, particularly in the supra-second range, is consistently disrupted, while implicit and sub-second timing often remains relatively preserved. Besides this dissociation, attention, working memory, and compensatory recruitment of cortical and cerebellar circuits contribute to time processing alongside basal ganglia dysfunction in PD.

Together, these findings position temporal cognition as a sensitive marker of both motor and cognitive alterations in PD. They also emphasize that dopaminergic therapy, while essential for motor symptoms, provides only partial relief for timing deficits. A more complete understanding of time processing in PD will be key to designing targeted diagnostic tools and personalized therapeutic strategies that address both the motor and cognitive dimensions of the disease.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2026.106719.

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