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Proposal for a Unified Framework to Understand the Functional Changes That Occur in Visual Hallucinations in Parkinson's Disease

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Abstract

Visual hallucinations in Parkinson's disease are often viewed as isolated symptoms of neurodegeneration. However, growing evidence suggests that they reflect deeper disruptions in how the brain constructs perception. This paper reviews three major hypotheses—early visual dysfunction, cholinergic dysfunction, and serotonergic dysfunction—and proposes a unified framework linking these mechanisms across multiple levels of brain organization. By integrating electrophysiological, neurochemical, and network-level findings, this study argues that hallucinations arise from an imbalance between internally generated and externally driven signals. Specifically, serotonergic dysfunction, through alterations in 5-HT2A receptor density, may be a mechanistic bridge linking early sensory processing with large-scale network dynamics, including impaired transitions between brain states. Understanding these mechanisms provides insight not only into Parkinson's disease, but also into the fundamental nature of perception as an active and dynamic process. More broadly, this work highlights the importance of approaching neurodegeneration as a distributed, multilevel phenomenon, with implications for developing more integrated and effective therapeutic strategies.

Keywords: Parkinson's disease (PD), visual hallucinations, serotonin, 5-HT2A receptor, visual processing, attention networks, brain-state dynamics

1. Introduction

Parkinson's disease (PD) is a neurodegenerative disorder attributed to a dysfunction in the dopaminergic system (Lew, 2007). The primary diagnostic symptoms of PD are motor, although patients often experience a range of non-motor symptoms as well. Moreover, patients with PD may develop dementia or psychosis (Friedman, 2010). Parkinson's disease psychosis (PDP) is characterized primarily by visual hallucinations (VH).

VH can present in different forms, and they are commonly classified as elementary and complex. Elementary VH generally consists of geometric patterns, such as lines, curves, or spots (Billock & Tsou, 2012). These can also have additional features such as color or motion. Conversely, complex VH features a variety of different contents, often depicting animal or human figures in absurd or unrealistic contexts (Manford & Andermann, 1998). Patients who suffer from a psychotic disorder often encounter both elementary and complex VH, but most PD patients recall having experienced complex VH (van Ommen et al., 2023).

It was initially believed that VH and PDP were a collateral effect of dopaminergic treatments, administered to alleviate the motor symptoms of the disorder; however, some PD patients develop PDP without medications (Friedman, 2010). This suggests that PDP is a progression of PD itself. Indeed, while dopamine is the main neurotransmitter attributed to PD motor symptoms, it is believed that the disorder also displays dysfunctions in other neurotransmitter systems and in the interactions between those; many motor and non-motor symptoms of the disease have been attributed to neurotransmitters such as serotonin, acetylcholine, glutamate, and γ-aminobutyric acid (GABA) (Barone, 2010).

PD is the second most common neurodegenerative disorder worldwide, and VH has a prevalence range of 8%–40% in PD patients (Barone, 2010; Manganelli et al., 2009). Moreover, there are a variety of correlates linking VH and PD. For example, VH in PD is associated with aberrant event-related potential (ERP) components, including alterations in P1, which reflects early visual processing, and P3b, associated with later cognitive processing of stimuli. Moreover, some patients with PD and VH (PD-VH) present anomalous neural network behavior, including enhanced excitability in the default mode network and in attention-related networks, as well as longer periods in segregated brain states and fewer transitions toward integrated states. Nevertheless, the underlying mechanisms giving rise to VH are largely unknown.

A complicating factor to this phenomenon is the high occurrence of dementia in PD, known to cause cognitive impairment and dysfunctions in many neural networks, namely in attention, memory and visual perception pathways, sometimes resulting in VH itself (Gratwicke et al., 2015). Since dementia and psychosis often coexist in PD patients, it is difficult to discern the distinct effects that the two conditions have on neural networks in PD, as well as to identify their respective causal factors.

At present, there remains no unified framework explaining how neural networks contribute to VH in nondemented patients with PD. Investigating the functional changes that occur in VH in PD can offer new insights into visual processing and stimulus interpretation. Moreover, it may inform strategies for treating VH and improving patients' quality of life. This is clinically relevant as VH in PD is associated with an increased risk of dementia and mortality (Manganelli et al., 2009).

The present study reviews three existing hypotheses of VH in PD, namely early visual, cholinergic, and serotonergic, and examines how evidence from these hypotheses may interact rather than represent mutually exclusive explanations. In

particular, it proposes that serotonergic dysfunction may provide one possible integrative bridge between these abnormalities.

2. Method and Scope of the Review

This paper is a focused narrative review and theoretical synthesis. It compares evidence from electroencephalography, neuroimaging, pharmacological studies, network-control approaches, and postmortem receptor analyses (see Table 1).

Table 1: *Methods Used to Characterize Neural Dysfunction in Visual Hallucinations*

Method	What it measures	Main strength	Main limitation
Electroencephalography (EEG) and Event-Related Potentials (ERPs)	EEG records electrical activity from the scalp; ERPs isolate activity time-locked to specific stimuli or events (Fabiani et al., 2007; Beres, 2017).	Excellent temporal resolution, allowing neural responses to be tracked on the millisecond scale (Beres, 2017).	Limited ability to precisely localize the neural source of the recorded activity (Beres, 2017).
Resting-state fMRI (rsfMRI)	Measures spontaneous fluctuations in the blood-oxygen-level-dependent (BOLD) signal at rest and their correlations across brain regions, allowing functional connectivity to be estimated (Van Den Heuvel & Pol, 2010).	Allows investigation of large-scale functional networks across the brain.	BOLD is an indirect measure of neural activity and has relatively poor temporal resolution because of the delayed haemodynamic response.
Short-latency afferent inhibition (SAI)	A neurophysiological measure of sensorimotor inhibition that is used as a putative marker of central cholinergic function (Martin-Rodriguez & Mir, 2020).	Provides a non-invasive physiological measure associated with cholinergic function in humans.	It is an indirect marker rather than a direct measurement of acetylcholine, and it can be influenced by factors such as disease duration and dopaminergic medication (Martin-Rodriguez & Mir, 2020).

Network-control analysis	Uses structural and functional brain-network data to estimate transitions between brain states and the control energy required for those transitions (Zarkali et al., 2022).	Can characterize dynamic differences in integrated and segregated brain states.	The inferred transitions and energy values depend on the assumptions of the mathematical model used and do not directly measure neurotransmitter activity.
Postmortem receptor analysis	Uses receptor-binding techniques in brain tissue to quantify receptor density; Huot et al. (2010), for example, measured 5-HT2A receptor binding in cortical tissue from PD patients.	Provides direct anatomical evidence of receptor-level differences in human brain tissue.	It is cross-sectional and cannot establish whether receptor changes precede or result from VH.

Relevant studies were identified between June and August 2025 through targeted searches of PubMed, Google Scholar, and reference lists of key reviews using combinations of terms related to PD, VH, visual processing, cholinergic dysfunction, serotonin, 5-HT2A receptors, and brain-state dynamics. Studies were selected for their relevance to three recurring explanatory levels: early visual processing, cholinergic modulation, and serotonergic dysfunction. The final bibliography comprised 35 sources published between 1978 and 2024, although older sources were primarily included for methodological or conceptual background. Most cited literature (30 of 35 sources) was published between 2007 and 2024. Eight core empirical studies, published between 2008 and 2023, were selected for detailed comparison because they directly examined evidence relevant to the three explanatory models considered in this review and provided findings pertinent to the overall synthesis. Studies were prioritized when they directly examined VH, relevant electrophysiological or network abnormalities, or cholinergic/serotonergic mechanisms pertinent to the proposed framework; studies used only to define methods, neural processes, or broader PD background were treated as supporting literature rather than empirical evidence. Where findings were inconsistent with, or did not fully support, the proposed hypotheses, these were discussed explicitly rather than excluded from consideration. Because the included studies differ substantially in population, task, and measurement method, and several do not examine nondemented PD patients directly, the proposed framework should be interpreted as a testable integrative hypothesis rather than a demonstrated causal model.

3. Literature Review on Visual Hallucinations in Parkinson's Disease

Several models have been proposed to explain the functional changes associated with VH in PD. The present study focuses on three frameworks that emphasize different but potentially interacting levels of dysfunction. The first hypothesis focuses

on early visual dysfunction (Schwartzman et al., 2008; van Ommen et al., 2023), while the second and third present a broader explanation of VH involving an imbalance between external and internal stimuli and the modulation of visual and attentional networks. The distinguishing factor between the last two models lies in the type of neurotransmitters involved: the second emphasizes central cholinergic dysfunction (Manganelli et al., 2009), whereas the third focuses on serotonergic dysfunction and altered 5-HT2A receptor density (Kometer et al., 2013).

3.1. Early Visual Dysfunction

The early visual dysfunction model proposes that abnormalities in the initial processing of visual information may contribute to VH and, through disrupted communication with higher-order visual and attentional networks, lead to the misinterpretation of internally generated information as external. Evidence for this model comes from studies examining both early electrophysiological responses to visual stimuli and the interaction between primary visual processing and broader visual-attentional networks.

A study conducted by Schwartzman et al. (2008) evaluated the hypothesis that aberrant early visual ERP components reflect individuals' propensity to hallucinate. While early visual disruption had been previously associated with various psychological and neurological disorders that present VH—e.g., PD, schizophrenia—it remained unclear whether an early impairment of visual processes was a key factor in the development of VH or rather a mere outcome of the disorder itself.

To reduce the possibly confounding influence of cognitive decline, the study analyzed electroencephalogram (EEG) recordings from healthy individuals divided according to their proneness to hallucinate. Hallucination proneness was assessed using a questionnaire, after which participants completed a face-perception task involving photographs, Mooney faces, and scrambled Mooney faces (see Figure 1). The Mooney Faces Test (MFT) evaluates an individual's ability to perceive human faces in two-dimensional, black-and-white pictures, shown both upright and upside down (Silverstein et al., 2021).

During the experiment conducted by Schwartzman et al., the participants' brain activity was recorded through EEG; specifically, the study focused on ERP components that reflect early visual processes. The components analyzed were the N170, P1, and P2, respectively associated with face recognition, early extraction of information from face stimuli, and finally a higher level of visual processing involving categorization of face stimuli. P2 responses depend in part on information processed during the earlier P1 stage.

The study observed no significant differences in the N170 component, while P1 and P2 showed increased positivity in hallucination-prone individuals. The lack of significant N170 differences suggests that the observed changes in P1 and P2 were not specific to face recognition but may instead reflect abnormalities in earlier stages of visual processing. Conversely, the increased amplitude registered in P1 suggests a dysfunction in the extraction of fine information from visual stimuli, which would in turn impair the interpretation of the visual information itself, explaining the downstream effects on P2. Thus, compared with individuals with low hallucination proneness, hallucination-prone participants showed altered stimulus-evoked electrophysiological responses during early visual processing. However, because the study involved healthy participants rather than PD patients with VH, these findings support an association with hallucination susceptibility rather than establishing a PD-specific causal mechanism.

An earlier study had shown a correlation between the P1 ERP component in patients with PD and primary visual processes

(Wang et al., 2001). The association between P1 and primary visual processing in PD provides a basis for considering abnormal P1 responses as a possible marker of early visual dysfunction in PD-VH.

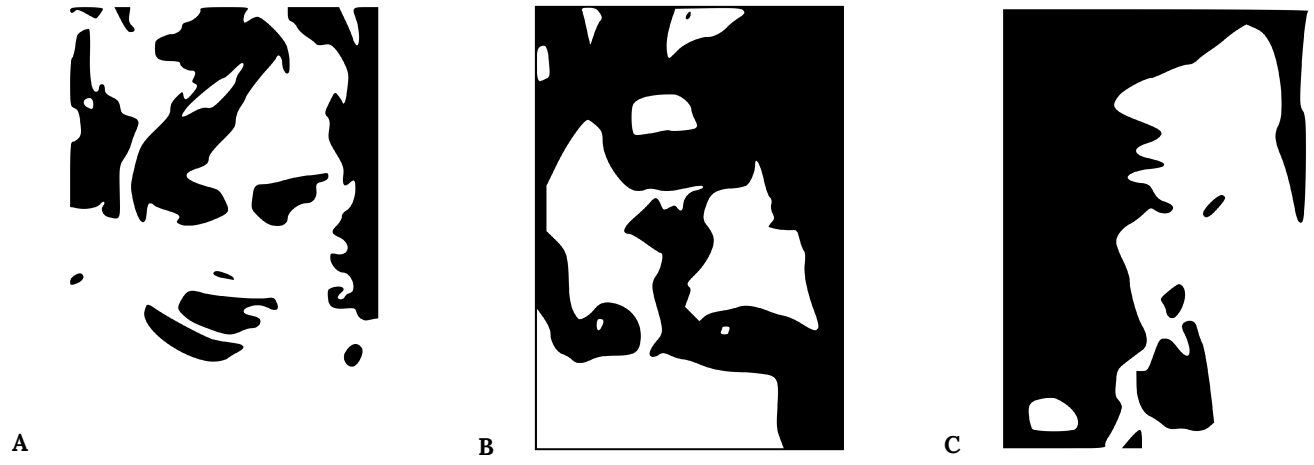


Figure 1: *Representative stimuli from the Mooney Faces Test.*

Note. The upright Mooney face (A) is typically perceived as a face, whereas the inverted Mooney face (B) is substantially more difficult to recognize due to disruption of holistic face processing. The scrambled image (C) serves as a non-face control stimulus. Stimuli were adapted from the dataset of Schwiedrzik et al. (2018) and redrawn as vector graphics for presentation clarity under CC BY 4.0 (https://creativecommons.org/licenses/by/4.0/).

Although early visual dysfunction is associated with VH in PD, existing evidence does not support a purely focal explanation. Instead, a study led by van Ommen et al. (2023) correlated impairment in primary visual processes with dysfunction in other visual-related neural networks, proposing a more holistic explanation of VH.

They aimed to evaluate a hypothesis previously postulated by Shine et al. (2014), which had explained VH as a consequence of a disconnection between the primary visual cortex, V1, and higher visual components, inducing a desynchronization between different parts of the visual network. This previous hypothesis was supported by the reduced activity registered in the primary visual cortex in patients with psychotic VH.

To evaluate the postulated model from Shine et al. and assess what areas of the brain are involved in psychotic VH, van Ommen et al. (2023) conducted an experiment on individuals experiencing psychotic hallucinations, using functional magnetic resonance imaging (fMRI) to measure brain activity during an episode of VH. The results showed a lack of activation in V1 and increased activity across the visual and attention pathways, specifically in the Dorsal Attention Network (DAN), Default Mode Network (DMN), and Ventral Attention Network (VAN). The DAN is primarily associated with externally directed attention, whereas the DMN is associated with internally oriented processing. The VAN contributes to switching between these modes of processing (Van Calster et al., 2017). The results are therefore in line with the hypothesis postulated by Shine et al. (2014), as they show that psychotic VH is not a focal pathology. Instead, it affects a wide range of

regions, namely the visual network (VIS)—excluding V1—and vision-related networks involved with memory and attention.

Based on these findings, Van Ommen et al. proposed a refinement of the model postulated by Shine et al., attributing VH in PD to the activation of a loop across the VIS—with the exception of V1—and the DAN, DMN, and VAN. Under normal circumstances, feed-forward impulses are sent from V1 to the surrounding visual network regions. This flow of information allows the stimulus to be recognized as external. In turn, a feedback inhibitory response is sent from the networks to V1. According to this model, the ability of V1 to provide feed-forward and receive feedback information from the surrounding areas is compromised in psychotic VH. This disruption may cause internally generated information to be misinterpreted as external sensory input, thereby contributing to the generation of VH.

However, since van Ommen et al. investigated VH in individuals with psychosis rather than specifically in PD patients, the extent to which this network mechanism applies directly to PD-VH remains to be established.

Additional evidence comes from Yao et al. (2014), who investigated DMN activity in resting PD patients both with VH (PD-VH) and PD patients without VH (PD-non-VH), as well as in healthy controls. Patients with symptoms of cognitive impairment and dementia were excluded. Resting-state functional magnetic resonance imaging (rsfMRI) data of the participants were acquired, and the results showed decreased activity in the DMN in PD patients compared to controls. Conversely, PD-VH patients had greater connectivity in the DMN compared to PD-non-VH. Yao et al., therefore, demonstrated that higher connectivity in the DMN is associated with VH in PD patients, relative to patients with PD without VH. Showing a correlation between aberrant DMN activity and VH in PD, these data support the model proposed by van Ommen et al.

Thus, while there are findings suggesting that early visual impairment has a role in generating VH in PD, this is treated as one level of a distributed mechanism rather than a complete explanation of the condition. Moreover, this presented model fails to include the involvement of neurotransmitters in VH in PD. To fully comprehend the underlying neurological mechanisms of VH in PD, it is necessary to adopt a more holistic approach, recognizing the role of neurotransmitters in modulating attention and vision-related networks. Consistent with the model postulated by van Ommen et al., 2023, a second hypothesis attributes VH in PD to an imbalance between external and internal stimuli associated with central cholinergic dysfunction.

3.2. Imbalance Between External and Internal Stimuli and Central Cholinergic Dysfunction

The cholinergic model proposes an association between impaired cholinergic modulation of attention and the imbalance between externally driven and internally generated information observed in VH in PD. Nevertheless, the available evidence, coming from both physiological measures of central cholinergic function and ERP studies of cognitive processing in nondemented PD patients with VH, does not fully establish the direction of this relationship. There remains disagreement whether cholinergic dysfunction directly contributes to VH, develops alongside VH as part of PD progression, or arises secondarily in association with the condition.

A 2009 study by Manganelli et al. found an association between central cholinergic dysfunction and VH in nondemented patients with PD. The research methodology included the use of short-latency afferent inhibition (SAI), a physiological measure commonly used as an indirect marker of central cholinergic function (Martin-Rodriguez & Mir, 2020). The

participants were 22 nondemented PD patients, both with and without VH (Manganelli et al., 2009). The study found that most PD-VH patients presented aberrant SAI values, unlike individuals with PD without VH. Therefore, Manganelli et al. concluded that VH in PD could be one of the factors that alter the balance of the cholinergic system. Alternatively, VH would contribute to dysfunction in the balance between cholinergic and other neurotransmitter systems. The study also confirmed the hypothesis of a correlation between cholinergic dysfunction and PD patients who are likely to develop dementia. Similarly, VH are considered to be a predictive factor of dementia. Therefore, there is a possible link between central cholinergic dysfunction and VH in PD.

However, there remains disagreement about the direction of causality, as other later studies consider cholinergic dysfunction as a causal factor of VH in PD. One of these is a study led by Chang et al. (2016).

While aberrant ERP values are considered an indicator of cognitive dysfunction in demented PD patients, their role in nondemented PD patients, both with and without VH, remained unclear. Specifically, some studies had found a correlation between PD-VH patients and impaired P3 responses, a component related to attentional allocation and cognitive processing of task-relevant stimuli (Kurita et al., 2010; Chang et al., 2016). To address this gap in the literature, Chang et al. compared ERP responses in nondemented PD patients with and without VH, and in healthy controls during a visual target-detection task. They reported that PD-VH individuals showed a prolonged latency of one subcomponent of the P3, namely the P3b component, compared with controls. Since P3b is associated with later cognitive processing and memory updating, this result was interpreted as evidence of altered cognitive processing in PD-VH and was considered compatible with central cholinergic dysfunction (Chang et al., 2016; Polich, 2003).

This study also investigated N1 and P2, two exogenous ERP responses associated with sensory and attentional processing, respectively (Chang et al., 2016; Donchin et al., 1978). Chang et al. reported no significant differences in either component across the groups. This finding does not necessarily contradict the visual-network dysfunction model described previously. These ERP components and the DAN, VAN, and DMN reflect different levels of neural processing, and there is no evidence that abnormalities in these large-scale networks must be accompanied by altered N1 or P2 responses. The Chang et al. findings therefore add evidence of altered later cognitive processing rather than excluding the involvement of visual and attentional networks.

Overall, the findings reviewed support an association between central cholinergic dysfunction and VH in PD; yet they do not establish its direction of causality. Consequently, the present framework treats cholinergic dysfunction as a potentially interacting contributor to VH rather than as a demonstrated upstream cause or downstream consequence.

3.3. Serotonergic Dysfunction, 5-HT2A Receptors, and Brain-State Dynamics

The serotonergic model proposes that altered serotonergic signaling, particularly through 5-HT2A receptors, may contribute to VH by modifying visual processing and the balance between internally and externally driven neural activity. Evidence for this model comes from pharmacological manipulation of 5-HT2A signaling, postmortem receptor analyses in PD, and studies of large-scale brain-state dynamics.

An aberrant density of 5-HT2A receptors in different cortical areas has often been connected to various forms of psychosis, including PDP (Huot, 2018), but the effect of 5-HT2A receptor activation on VH remains unclear. A study conducted by

Kometer et al. (2013) aimed to clarify this connection by investigating the effects that the activation of the 5-HT2A receptor has on parieto-occipital alpha waves.

Alpha (α) oscillations, prominent in visual cortical activity and influenced by thalamocortical interactions, have been correlated with visual processing dysfunctions and impairments (Clayton et al., 2018). Since they are strongly linked to visual processing, they are also associated with visual-related potentials (Kometer et al., 2013). The study presented here hypothesized that alpha oscillations could contribute to the generation of VH. According to their model, activation of 5-HT2A receptors, triggered by the administration of psilocybin, a serotonergic hallucinogen, would alter alpha oscillations. A second hypothesis postulated by the study concerned how early visual ERP components—specifically the P1 and N170—would be influenced by psilocybin-induced hallucinations. Thus, the investigation examined the effects of 5-HT2A activation on visual processing in healthy participants by comparing the effects of psilocybin with those observed after pretreatment with the 5-HT2A antagonist ketanserin. EEG responses were recorded during a visual perception task.

Psilocybin increased P1 amplitude over medial parieto-occipital regions, altered N170 responses, and decreased alpha power. These effects were suppressed by ketanserin pretreatment, supporting their relationship with 5-HT2A activation. Decreased alpha power was attributed to increased cortical excitability, especially in the parieto-occipital regions and visual networks, following activation of 5-HT2A receptors. A reduction in alpha power had previously been associated with increased visual attention and cortical excitability in the occipital lobe, while increased alpha oscillations might directly suppress visual processing (Clayton et al., 2018). The decreased alpha power was also related to the increase in P1 medial potential, which might represent an enhancement in early visual processing (Kometer et al., 2013). This coordinated response between the P1 and alpha power would in turn contribute to an imbalance between perception of external and internal stimuli.

Specifically, altered cortical excitability may reduce the normal distinction between neural activity driven by external visual input and activity occurring in the absence of external stimuli. This corresponds to a change in the activation of brain networks—reflected by decreased alpha oscillations and enhanced P1 medial potentials—with increased attention toward internally generated visual stimuli, rather than external stimuli. This model seems to be in line with the hypothesis proposed by van Ommen et al. (2023), in which VH arises from an altered balance between externally and internally generated signals.

Although the findings suggest that alpha power may have a role in psilocybin-induced VH, subsequent analyses showed little correlation between reduced alpha power and the occurrence of hallucinations, perhaps revealing that the reduction in alpha power triggered by psilocybin is not sufficient to induce complex VH. Moreover, the study involved psilocybin-induced perceptual alterations in healthy participants rather than in the PD population. Therefore, the study does not establish that the same mechanism causes complex VH in PD.

Finally, the psilocybin treatment produced a decrease in the N170, which is thought to play a key role in processing meaningful visual stimuli. An alteration in this component is associated with altered visual perception and may therefore reflect changes produced during psilocybin-induced hallucinations. Alternatively, the effects on the N170 could be attributed to the increased cortical excitability triggered by the activation of the 5-HT2A receptor, further contributing to an imbalance between external and internal stimuli.

Taken together, the effects of 5-HT2A activation on P1, N170, and alpha power demonstrate that serotonergic signaling can alter neural processes involved in visual perception and cortical excitability, although these findings do not establish that the same mechanism causes VH in PD.

A previous study had investigated the distribution of 5-HT2A receptors in postmortem PD patients' tissues, postulating a correlation between increased receptor density and VH in PD (Huot et al., 2010). The results showed an increased density of this receptor in tissues of PD-VH patients, compared to PD patients without VH, therefore providing PD-specific evidence supporting the involvement of 5-HT2A signaling in VH. While an existing postulate attributed the development of psychosis in PD to a polymorphism in the 5-HT2A receptor gene, a study led by Kiferle et al. (2007) refuted this hypothesis, and they concluded that aberrant levels of this receptor are not due to the genetic variations examined.

Huot et al. (2010) proposed a new explanation of the enhanced levels of 5-HT2A receptors in PD patients with psychosis, defining it as a consequence of other neurotransmitter dysfunctions in PD. PD is known to present serotonin (5-HT) dysfunctions; specifically, degeneration of serotonergic neurons in the brainstem causes a decline in neurotransmitter levels. Huot et al. proposed that this may contribute to a compensatory increase in 5-HT2A receptor density, as a biological attempt to balance the drop in 5-HT in different areas of the brain. Enhanced levels of 5-HT2A could also be attributed to the decrease in dopamine (DA) levels, which acts as a partial agonist at the receptor. These two possibilities are likely to be complementary, and this explanation would help explain the late occurrence of VH in the development of PD: the disorder disrupts the dopaminergic system more than the serotonergic system, suggesting that it takes longer for the levels of 5-HT to drop below the threshold level. According to this hypothesis, the resulting serotonergic and receptor-level changes could eventually contribute to the development of VH. This proposed compensatory increase in 5-HT2A receptor density further supports the hypothesis that dysfunction of the serotonergic system, and particularly altered 5-HT2A signaling, may have a role in VH in PD.

A possible limitation of the presented study is the interference of dementia, which was present in four out of the six PD-VH participants. Since dementia is known to cause dysfunctions in many neural networks, such as attention, memory, and visual perception (Gratwicke et al., 2015), this factor limits the reliability of the findings and makes it unclear whether they should be attributed to psychosis rather than dementia.

Additional evidence suggests an association between 5-HT2A and altered brain-state dynamics in PD-VH. Specifically, an aberrant density of this receptor was attributed to temporal dysfunctions in transitions between the segregated and integrated states of the brain in hallucinating PD patients (Zarkali et al., 2022). Integrated and segregated states are opposite methods of network organization, although they often coexist in the same functional system (Tononi et al., 1994). Integration means that the different areas of the network are interconnected and there is a fast flow of information among them; each component of the circuit is dependent on the others. Conversely, segregation happens when different regions function relatively independently of one another, with a limited amount of information flow within the whole system.

It is believed that shifts between integration and segregation are controlled by neurotransmitter systems (Zarkali et al., 2022). The study aimed to investigate how temporal dynamics in transitions between integrated and segregated states are affected in PD patients with VH, using rsfMRI.

The main difference reported was not the presence of distinct integrated or segregated states, but their temporal

dynamics: PD-VH participants spent less time in integrated states, more time in segregated states, and made fewer transitions between them than PD-non-VH participants.

The framework presented by these findings suggests that the DMN and thalamic regions have a role in VH in PD, as these are the nodes responsible for modulating the transitions between integrated and segregated states. Furthermore, the study expanded on these findings, attempting to assess the role of neurotransmitters in the alterations observed. The data gathered by Zarkali et al. provide further evidence for possible involvement of serotonergic signaling in developing VH in PD, as they found significant correlations between regional transition energy and neurotransmitter receptor-density maps, including 5-HT2A receptors.

4. Proposal for a Unified Framework

4.1. Summary of Reviewed Literature

The first hypothesis, schematized in Figure 2A, attributes VH in PD to early visual dysfunction. According to this model, an impairment in V1 would cause the activation of a loop across areas of the visual network, with increased activity in the DMN (Yao et al., 2014) and dysfunction across the DAN and VAN, resulting in the recognition of internally generated stimuli as external inputs (van Ommen et al., 2023). Aberrant P1 responses may reflect altered early visual processing, although they do not specifically localize this dysfunction to V1 (Schwartzman et al., 2008; Wang et al., 2001).

The second hypothesis focuses on the role of a cholinergic dysfunction in PD-VH patients (see Figure 2B). However, the available evidence does not establish whether this directly contributes to VH or rather develops as a consequence of the condition. Manganelli et al. (2009) postulated the presence of a link between cholinergic dysfunction and VH in PD patients reflected by aberrant SAI values; the direction of causality remains unclear. Conversely, Chang et al. (2016) observed an increased latency of the P3b component in EEGs of PD-VH patients, hypothesizing that a central cholinergic dysfunction might be an underlying cause of VH in PD. Accordingly, the unified framework does not assign a fixed causal position to cholinergic dysfunction.

Lastly, the third hypothesis, outlined in Figure 2C, suggests that an aberrant density of 5-HT2A receptors in PD-VH patients, possibly due to the dysfunction of the 5-HT system in PD (Huot et al., 2010), may be associated with altered transitions between integrated and segregated states (Zarkali et al., 2022). Moreover, pharmacological activation of 5-HT2A receptors in healthy participants resulted in increased amplitudes of the P1 potential and decreased alpha power values, providing experimental evidence that 5-HT2A activation can alter early visual processing and cortical excitability (Kometer et al., 2013). These changes may, in turn, contribute to an imbalance between the processing of external and internally generated stimuli.

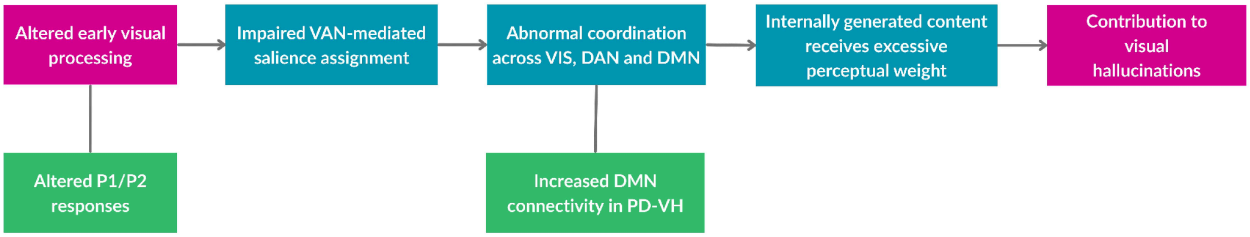
4.2. How Do These Models Relate to Each Other?

In light of the evidence considered and summarized in Table 2, this study proposes a unified framework to explain the functional changes that occur in PD-VH patients. Although the three models emphasize different mechanisms, their findings are not necessarily mutually exclusive. Instead, they may describe abnormalities occurring at different levels of the same distributed perceptual system. The following comparison therefore focuses on points of convergence while

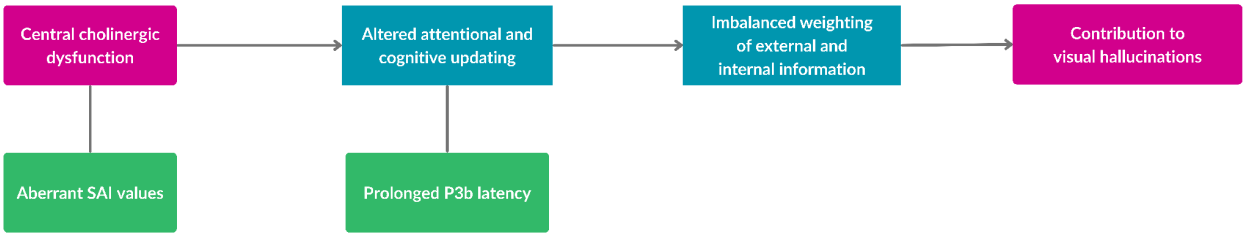
distinguishing direct empirical findings from mechanistic links proposed by the present framework.

Firstly, both the first and third models propose that an underlying mechanism of VH in PD may involve an imbalance between internally generated stimuli and external inputs, leading patients to misattribute internally generated stimuli as exogenous. While an early visual dysfunction might alter the processing of external sensory information, network-level abnormalities may describe how this information interacts abnormally with internally generated activity (Schwartzman et al., 2008; van Ommen et al., 2023). Cholinergic and serotonergic dysfunction may in turn modulate these sensory and network processes (Chang et al., 2016; Kometer et al., 2013).

A. Early visual dysfunction model



B. Cholinergic dysfunction



C. 5-HT2A receptor dysfunction model

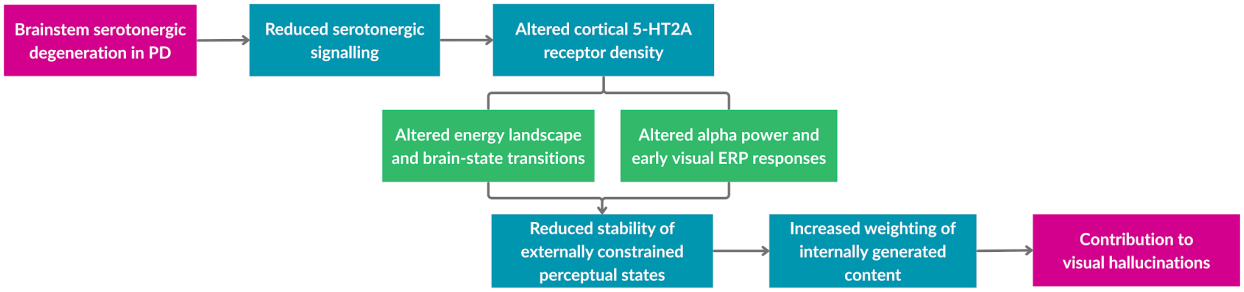


Figure 2: *Schematic representations of the three models of VH in PD. (A) Early visual dysfunction model. (B) Cholinergic dysfunction model. (C) 5-HT2A receptor dysfunction model.*

Note. Color code: key nodes in pink; intermediate mechanistic steps in blue; supporting empirical observations in green. Line code: continuous arrows indicate directional relationships proposed within each model; continuous lines without arrowheads indicate observed associations for which causal direction has not been established.

Table 2: *Summary of reviewed literature*

Hypothesis	Study	Approach	Key finding and relevance
Early visual dysfunction	Schwartzman et al. (2008)	EEG/ERP in hallucination-prone healthy participants	Higher P1/P2 positivity supports altered early visual processing but does not establish PD-specific causality.
	Yao et al. (2014)	Resting-state fMRI in PD-VH, PD-non-VH, and controls	Greater DMN connectivity in PD-VH supports involvement of internally oriented network activity.
	van Ommen et al. (2023)	fMRI during hallucinations in psychosis	Reduced V1 involvement with increased VIS/DAN/DMN/VAN activity supports a distributed network account.
Cholinergic dysfunction	Manganelli et al. (2009)	SAI in nondemented PD patients	Abnormal SAI links PD-VH to central cholinergic dysfunction; direction of causality remains unresolved.

	Chang et al. (2016)	Visual ERP task in nondemented PD groups	Prolonged P3b latency is consistent with altered cholinergic/cognitive processing in PD-VH.
Serotonergic/ 5-HT2A	Kometer et al. (2013)	Psilocybin/ketanserin EEG study in healthy adults	5-HT2A activation altered P1, N170, and alpha power, supporting serotonergic involvement in visual processing and cortical excitability.
	Huot et al. (2010)	Postmortem temporal-cortex receptor analysis	Higher 5-HT2A receptor density in PD-VH supports receptor-level involvement, though dementia is a confound.
	Zarkali et al. (2022)	Resting-state fMRI and network-control analysis	PD-VH showed fewer transitions and longer segregation, linked regionally to 5-HT2A receptor density.

Additionally, aberrant P1 values observed in hallucination-prone individuals (Schwartzman et al., 2008) may be compatible with the explanatory model proposed by van Ommen et al. (2023). Schwartzman et al. (2008) interpreted P1 abnormalities as reflecting altered early visual processing, while van Ommen et al. proposed that psychotic VH involves a dysfunction in V1, together with the DAN, DMN and VAN, resulting in an imbalance between external and internally generated stimuli. This evidence suggests that altered early visual processing reflected by P1 and dysfunction in V1 in psychotic VH may potentially represent related features of the same broader perceptual disturbance. However, Schwartzman et al. investigated healthy individuals prone to hallucinate, and P1 measurements do not specifically localize dysfunction to V1. Thus, studies examining P1 responses alongside measures of V1 activity and network function in a PD-VH population are necessary to determine whether these abnormalities are mechanistically related.

Another possible point of convergence in the reviewed literature is that the decreased alpha power reported by Kometer et al. and the altered brain-state dynamics observed by Zarkali et al. may reflect a common underlying neural dysfunction. Kometer et al. (2013) observed increased P1 amplitudes and decreased alpha power following pharmacological activation of 5-HT2A receptors, while Zarkali et al. (2022) found associations between 5-HT2A receptor-density maps and altered brain-state transitions. Specifically, the latter study found that PD-VH patients spent more time in the segregated state rather than the integrated one, compared to PD-non-VH patients. There is evidence that alpha power increases during visual integration in various areas of the brain, including the early visual cortex and dorsal visual networks (Costa et al., 2024). This raises the possibility that increased dwelling time in segregated states in PD-VH patients and the decreased alpha power observed by Kometer et al., alongside increased P1 potentials, may reflect different manifestations of a common underlying imbalance in neural integration associated with VH. Nevertheless, the findings derive from distinct

populations and methods, and measure different neurophysiological phenomena. Specifically, Kometer et al. investigated psilocybin-induced changes in healthy participants, whereas Zarkali et al. examined brain-state dynamics in patients with PD-VH. Therefore, whether these findings reflect a common underlying mechanism remains to be established through further studies combining EEG and fMRI in patients with PD-VH.

Additional support for the proposed unified framework comes from the finding that transitions between segregated and integrated states are highly influenced by the activity of the DMN and thalamic regions (Zarkali et al., 2022), thereby linking attention- and memory-related networks to altered brain-state dynamics.

In addition, increased activity in the DMN observed in PD-VH patients (Yao et al., 2014) may be related to the aberrant P3b latency reported by Chang et al. (2016). As the P3b component reflects top-down attentional processing of visual stimuli, and the DAN is involved in directing attention toward salient information (Shine et al., 2014; Van Calster et al., 2017), the abnormal P3b values reported by Chang et al. may reflect a dysfunction in visual attention allocation involving visual and attentional networks—DAN, VAN, and DMN. Since DAN activity is typically negatively correlated with DMN activity within this attentional network pathway, dysfunction involving the DAN may also be associated with the increased DMN connectivity reported by Yao et al. This raises the possibility that abnormalities in P3b activity and DMN function reflect overlapping disturbances in attentional allocation and internally oriented processing of visual information. Nevertheless, this remains a speculative cross-scale hypothesis. Future studies combining EEG and fMRI are needed to determine whether P3b abnormalities are associated with dysfunction within attentional and visual networks, particularly the DMN.

Drawing on the convergent, but not yet causally unified, evidence considered, the present study proposes a framework in which abnormalities in early visual processing, neuromodulatory function, attentional networks, and brain-state dynamics may represent interacting levels of the same distributed perceptual disturbance (see Figure 3). Serotonergic dysfunction may act as one integrative neuromodulatory factor, interacting with cholinergic and dopaminergic abnormalities rather than functioning as an isolated or necessarily upstream cause.

PD is associated with degeneration of serotonergic neurons in the brainstem, leading to reduced 5-HT levels. Huot et al. (2010) proposed that reduced serotonergic signaling may be accompanied by a compensatory increase in 5-HT2A receptor density. Altered 5-HT2A signaling may in turn be, directly or indirectly, associated with changes in visual processing, alpha oscillations, attentional-network activity, and brain-state dynamics. Cholinergic dysfunction may additionally affect attentional allocation and the balance between externally and internally generated information, although its causal position relative to VH remains unresolved. Together, these abnormalities could contribute to a perceptual system in which internally generated information is more readily misattributed as external.

Within this framework, the comparatively later involvement of the serotonergic system in PD may help explain the delayed emergence of VH, while serotonergic dysfunction may also interact with other neurotransmitter systems and contribute to the downstream cascade of visual, attentional and network abnormalities.

Van Ommen et al. (2023) proposed that the loop involving the DMN, DAN, and VAN, and contributing to VH, may originate from a dysfunction in V1. Some studies link serotonin and acetylcholine to V1 functions in animals; for instance, Shimegi et al. (2016) investigated how cholinergic and serotonergic systems modulate visual processing in monkey V1. However, the available evidence does not establish specifically where dysfunction within visual and attentional networks originates, nor

which neurotransmitter systems are most directly involved. Determining the temporal and mechanistic relationships among early visual abnormalities, altered attentional-network activity, and neuromodulatory dysfunction will require studies combining electrophysiological, neurochemical, and functional-network measures within a well-characterized PD-VH population.

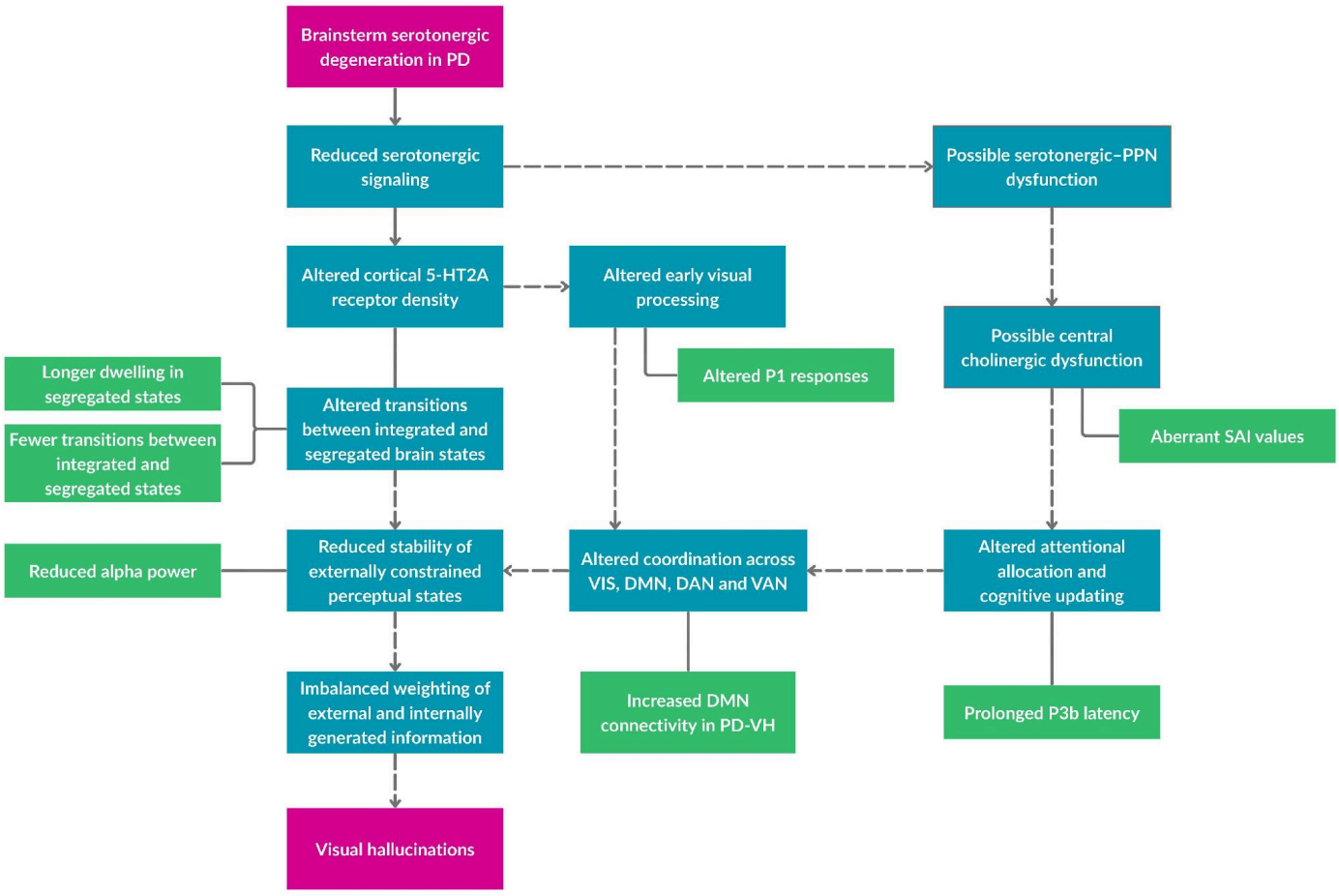


Figure 3: *Proposed unified framework for VH in PD.*

Note. Color code: key framework nodes in pink; intermediate mechanistic steps in blue; supporting empirical observations in green. Line code: continuous arrows indicate directional relationships retained from the reviewed models; continuous lines without arrowheads indicate observed associations for which causal direction has not been established; dashed arrows indicate integrative or unresolved mechanistic links proposed by the unified framework.

5. Conclusions

This review proposes that serotonergic dysfunction may play an integrative role in linking early visual abnormalities, altered neuromodulatory signaling, and large-scale network dynamics in PD with VH. By identifying testable links among abnormalities measured at different levels of the perceptual system, the proposed framework provides a basis for longitudinal and multimodal studies aimed at determining how these correlates interact, which changes occur earlier in the disease process, and how they contribute to the neural mechanisms underlying VH in PD.

6. Limitations and Future Research Directions

Accordingly, apparent convergence across studies should not be interpreted as evidence that the reported abnormalities belong to a single established causal sequence. In fact, the central limitation of the proposed framework is that it integrates evidence obtained from heterogeneous populations and methods. Some studies involve healthy participants exposed to serotonergic hallucinogens, some examine psychosis outside PD, and some include PD-VH samples affected by dementia.

Future work should therefore test the framework prospectively in carefully phenotyped, nondemented PD cohorts. The strongest design would combine receptor-sensitive imaging, EEG measures of P1/P3b and alpha power, and repeated fMRI measures of DMN–DAN–VAN coupling and integrated–segregated state transitions within the same participants. Longitudinal studies should determine whether serotonergic and cholinergic changes precede hallucination onset, while pharmacological studies should test whether modifying 5-HT2A or cholinergic signaling changes the predicted electrophysiological and network markers. These tests would distinguish correlation from causation and clarify whether the pedunculopontine nucleus (PPN) and dorsal raphe nucleus (DRN) are upstream drivers, downstream consequences, or interacting modulators of the proposed pathway.

Future studies could also investigate the role of the PPN in modulating cholinergic and serotonergic dysfunction. The PPN contains cholinergic neurons and receives serotonergic input from brainstem nuclei, including the DRN (Benarroch, 2013; Michelsen et al., 2007). Numerous studies mention the PPN in relation to VH in PD. With its cholinergic neurons, it is linked to attention networks, and it modulates visual cortical activity (Manganelli et al., 2009). Specifically, it modulates feedforward input to V1, exhibiting its crucial role in visual perception. Moreover, the neurotransmitters synthesized in the PPN modulate both the VAN and the DAN, together with other nuclei in the brainstem (Alves et al., 2022). These findings suggest that there is a connection between the PPN, attentional networks, and other structures in the brainstem, especially the DRN, and that these connections are modulated by neurotransmitters. However, the proposed role of the PPN as a link between serotonergic and cholinergic dysfunction and altered visual-attentional processing remains indirect and has not been demonstrated as a causal mechanism of VH in PD. Future studies should therefore determine whether PPN dysfunction contributes to VH by disrupting cholinergic and serotonergic modulation of visual and attentional networks.

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Her interest in perception also extends to music: she plays both violin and piano and is particularly interested in the relationships among music, memory, emotion, and cognition. She hopes to continue studying neuroscience at university and to pursue questions that connect fundamental brain mechanisms with clinically relevant problems.

Mentor Contribution Statement

The research mentor provided conceptual guidance, recommended relevant literature, discussed the interpretation of evidence, and offered feedback on the manuscript's organization and argument. The author independently conducted the literature review, developed the proposed unified framework, and wrote and revised the manuscript. The mentor did not collect or analyze original data and is not listed as a co-author.

AI Use Statement

Generative AI tools were used for limited editorial support, including assistance with identifying typographical errors and awkward phrasing, checking reference formatting, and improving the organization of flowcharts. The author independently developed the research question, selected and interpreted the literature, constructed the proposed framework, evaluated the scientific claims, and approved all final wording. AI tools were not used to generate original data or conduct data analysis. The author verified the accuracy of all substantive claims, citations, and references and accepts full responsibility for the manuscript.