The Neural and Computational Mechanisms of Hallucinations

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Abstract

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Full Text

Preamble

Neural and Computational Mechanisms of Hallucinations

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Abstract

Hallucinations are perceptual experiences that occur in the absence of external sensory stimuli, commonly observed across various psychiatric and neurological disorders, with severity correlating closely with disease progression. Research indicates that hallucinations are intimately associated with functional abnormalities in brain regions including the prefrontal cortex, insula, striatum, and hippocampus. Hallucinations arise from an imbalance between sensory signals and prior expectations, leading to aberrant integration of prediction error signals and the misattribution of internal signals as external stimuli. This article systematically reviews research paradigms for studying hallucinations, examines the neural circuits underlying hallucination generation, and elucidates the mechanisms of abnormal prediction error integration. Finally, future research directions are discussed, including establishing cross-modal standardized assessment systems, exploring closed-loop neurofeedback, and developing individualized intervention frameworks using targeted neuromodulation techniques.

Keywords: hallucinations, conditioned hallucinations, neural circuits, predictive coding, neuromodulation

1. Introduction

People sometimes hear voices that are not actually present or suddenly see images and lights that do not exist in reality—these are hallucinations. Hallucinations refer to subjective perceptions of objects that do not exist in objective reality and are closely associated with various psychiatric and neurological conditions (Corlett et al., 2019; Horga & Abi-Dargham, 2019; Waters & Fernyhough, 2017). As a core symptom of schizophrenia, hallucinations are experienced by approximately 60–80% of patients (Andreasen & Flaum, 1991; Sommer et al., 2012). Furthermore, hallucinations play a significant role in numerous other disorders, including depression and Parkinson’ s disease, representing a critical clinical feature that cannot be overlooked. In patients with depression, for instance, hallucination symptoms correlate closely with depressive and anxiety symptoms and may increase the risk of self-harm and suicidal behavior (Bornheimer et al., 2019; Toyohara et al., 2022; Yates et al., 2019). Additionally, the presence of hallucinations can impede patient recovery and further exacerbate impairments in psychological and social functioning (Knight et al., 2020). Although Parkinson’ s disease is characterized primarily by motor dysfunction, up to 60% of patients frequently experience hallucinations (Diederich et al., 2009). As the disease progresses, the frequency and severity of hallucinations increase significantly, adding to patients’ psychological burden and further reducing their quality of life (Lenka et al., 2019; Powell et al., 2022). Psychiatric disorders with hallucinations as a primary symptom account for 32.4% of disability-adjusted life years (ranking first) and 13.0% of years of life lost due to premature death (ranking second), imposing enormous burdens on human health, society, and the economy (Vigo et al., 2016).

However, hallucination research faces numerous challenges due to their unpredictable and uncontrollable nature, coupled with reliance on self-report measures (Rogers et al., 2021). Capturing neural data during spontaneous hallucinations is contingent upon multiple factors, including hallucination frequency, patient mental state, cooperation levels, and reporting accuracy. To address these issues, recent studies have introduced conditioned hallucinations (CHs), which associate objective stimuli with subjective experiences through experimental manipulation, providing a novel approach for exploring the neural mechanisms of hallucinations (Kafadar et al., 2022; Powers et al., 2017; Schmack et al., 2021; Yun et al., 2023). Within the Bayesian theoretical framework, perception is conceptualized as an inferential process based on prior beliefs and the reliability of sensory evidence. Recent research indicates that the weighting of priors versus sensory input—particularly an increased reliance on prior beliefs—constitutes a key mechanism for inducing hallucinations (Alderson-Day et al., 2017; Bansal et al., 2022; Cassidy et al., 2018; Corlett et al., 2019; Horga & Abi-Dargham, 2019; Kafadar et al., 2022; Powers et al., 2017, 2025; Schmack et al., 2021), with hallucination severity showing significant positive correlation with individual tendencies to rely on prior information (Bansal et al., 2022; Kafadar et al., 2022). Building on these findings, recent research has shifted focus toward exploring neural activity patterns associated with prior beliefs to identify potential biomarkers for hallucinations (Cassidy et al., 2018; Powers et al., 2017).

This article begins by systematically examining the heterogeneous behavioral manifestations of hallucinations and innovative developments in measurement paradigms, elaborating on the perceptual characteristics of multimodal hallucinations and their disease-specific expression patterns in psychiatric and neurological disorders. Subsequently, we review the neural circuit mechanisms underlying hallucination generation. Based on predictive coding theory, we elucidate the dynamic imbalance mechanism between reduced sensory signal weighting and excessive prior belief dominance, revealing the computational mechanisms of hallucination formation. Finally, we systematically evaluate the regulatory effects of transcranial magnetic/electrical stimulation on thalamocortical connections and the intervention efficacy of deep brain stimulation on hippocampal activity, while discussing the potential clinical value of closed-loop neurofeedback technology for real-time correction of prediction errors and symptom alleviation.

2.1 Behavioral Manifestations of Hallucinations

Human exploration of hallucinations dates back thousands of years (Rogers et al., 2021), with hallucinations typically defined as erroneous perceptual experiences occurring without corresponding external sensory stimulation (Corlett et al., 2019; Kafadar et al., 2022; Leptourgos et al., 2022; Powers et al., 2017; Rogers et al., 2021; Schmack et al., 2021). Hallucinations exhibit substantial heterogeneity in their presentation, occurring in either single sensory modalities (such as auditory or visual) or involving multiple modalities simultaneously. Their content ranges from simple flashes or sounds to complex scenes or dialogues, from ordinary everyday experiences to bizarre or terrifying imagery, with individuals showing significant variation in their awareness of these experiences' reality (Honig et al., 1998; Rogers et al., 2021; Waters & Fernyhough, 2017).

Hallucinations represent common symptoms across numerous psychiatric and neurological disorders, though the predominant modality varies by disease type (Naasan et al., 2021). For example, patients with schizophrenia may experience hallucinations in any sensory modality, with approximately 70% reporting auditory hallucinations, particularly auditory verbal hallucinations (AVH) (Andreasen & Flaum, 1991; Mo et al., 2024; Sommer et al., 2012), while about 27% report visual hallucinations (VH) (Waters et al., 2014). In contrast, patients with Parkinson' s disease and dementia with Lewy bodies more frequently exhibit visual hallucinations, with incidence rates of 28.2% and 61.8% respectively, whereas auditory hallucinations occur less frequently at 8.9% and 30.8% (Eversfield & Orton, 2019). Notably, hallucinations are not limited to pathological states; approximately 50% of healthy individuals may experience hallucinations under specific circumstances, such as during significant stress or emotional setbacks (Honig et al., 1998). In many cultures, hallucinations are even regarded as sacred experiences with profound spiritual significance (Rogers et al., 2021). However, when hallucinations are pathologically sustained, they severely disrupt cognitive function, induce distress and shame (Waters et al., 2014), and increase the risk of self-harm and suicide (Bornheimer et al., 2019; Toyohara et al., 2022; Yates et al., 2019).

In recent years, researchers have increasingly focused on the constructive nature of perception, recognizing that perception is not merely passive reception of external sensory information but rather an integrative process based on current sensory input and prior expectations (Corlett et al., 2019; Horga & Abi-Dargham, 2019). In other words, perceptual formation depends on the integration of bottom-up external stimuli and top-down internal expectations. Although this mechanism is efficient for information processing, it is also prone to errors. For instance, while waiting in a noisy park, an individual might misinterpret ambiguous background noise as someone calling their name. The likelihood of this misperception depends on internal expectations: if they are anticipating meeting a friend, this high expectation increases the probability of interpreting the noise as a signal (Figure 1 [Figure 1: see original paper]). This perceptual bias resembles the mechanism of hallucination generation, wherein excessive influence of prior expectations leads individuals to perceive non-existent stimuli as real. Researchers have further proposed that the distinction between veridical perception, illusion, and hallucination lies in the degree to which internally generated perceptual experiences are constrained by external sensory information: veridical perception is maximally constrained, illusion is moderately constrained, and hallucination is minimally constrained by external information (Rogers et al., 2021; Waters et al., 2014).

Figure 1. Example of perceptual decision-making with and without expectation bias. (a) No-expectation scenario: A person in a park hears an ambiguous sound (external objective stimulus) and must decide whether they heard their name ("yes" or "no" decision), with no additional expectation influencing the decision. (b) Expectation scenario: If the person had arranged to meet a friend there (contextual cue), they are more likely to make a "yes" decision, i.e., more readily believe they heard their name. (c, d) Perceptual decision processes. (c) In the no-expectation condition, the decision criterion (marked by vertical dashed line) remains neutral, with relatively low hit and false alarm rates. (d) In the expectation condition, the decision criterion shifts toward more liberal signal acceptance, resulting in higher hit rates but also increased false alarms.

2.2 Research Paradigms for Hallucinations

Due to their unpredictable and subjective nature, hallucinations present significant methodological challenges. Traditional research has primarily focused on two aspects: state capture and trait characteristics (Zmigrod et al., 2016). In recent years, innovative paradigms such as intracranial electrical stimulation (iES) and laboratory-induced conditioned hallucinations have provided new avenues for deeply investigating the neural mechanisms of hallucinations.

2.2.1 State Capture Studies

State capture studies reveal the immediate neural mechanisms of hallucination generation by recording brain activity in real-time during hallucination episodes. For example, participants mark the onset and offset of hallucinations via button press, allowing researchers to compare brain activity between hallucination and non-hallucination periods. Studies have found that auditory hallucinations involve significant co-activation of frontotemporal language networks and medial temporal memory hubs, suggesting abnormal integration of inner speech generation and memory retrieval (Jardri et al., 2011), whereas visual hallucinations elicit prominent activation in higher-order visual processing regions including the lateral occipital complex, middle temporal gyrus, and superior temporal sulcus (van Ommen et al., 2023). Notably, both modalities are accompanied by abnormal activity in the insula—a central hub for consciousness monitoring—and the dorsolateral prefrontal cortex (DLPFC), a key region in self-regulation networks, indicating that hallucinations represent a multidimensional dysregulation product of perception, memory, and self-monitoring systems (Zmigrod et al., 2016). However, capturing hallucination states in laboratory settings is extremely difficult, and many studies rely on case analyses with small sample sizes, limiting the generalizability of findings.

2.2.2 Trait Studies

Trait studies reveal hallucination mechanisms by comparing brain structure and activity (at rest or during tasks) between individuals with and without hallucination symptoms. Research has shown that the cerebellum is closely related to the mechanisms and treatment efficacy of auditory hallucinations (Chen et al., 2019). Further studies suggest the existence of a common hallucination functional network involving the cerebellar vermis and superior temporal sulcus (Kim et al., 2021). Auditory information processing in patients with auditory hallucinations exhibits dual deficits: on one hand, excessive enhancement of auditory gating-related potentials such as P50, N100, and P200 reflects failure of primary sensory filtering mechanisms (Thoma et al., 2017); on the other hand, reduced mismatch negativity amplitude indicates impaired cortical monitoring of information deviance (Perrin et al., 2018). Visual hallucinations in schizophrenia patients are associated with hyperconnectivity between the amygdala and visual cortex, suggesting abnormal integration of emotion and perception (Ford et al., 2015). Although trait analysis methods reveal stable neural markers through large-sample cohorts and provide targets for early intervention, they cannot distinguish whether these neural features represent causes or consequences of hallucinations.

2.2.3 Intracranial Electrical Stimulation

In recent years, intracranial electrical stimulation has provided a unique perspective for hallucination research. By applying invasive electrical stimulation to awake patients with brain disorders, such as drug-resistant epilepsy, researchers can induce hallucinatory experiences and directly localize the critical brain networks underlying hallucinations (Siddiqi et al., 2022). For instance, stimulating the auditory association cortex in the temporal lobe can elicit complex verbal auditory hallucinations (Jaroszynski et al., 2022), while stimulating primary visual cortex induces simple phosphenes such as patterns or gratings (Li et al., 2022; Mégevand et al., 2014). Direct stimulation of the superior temporal sulcus leads to insular activity abnormalities and induces auditory hallucinations such as singing voices (Jaroszynski et al., 2022), whereas direct stimulation of the insula, particularly the central sulcus, causes patients to report olfactory and gustatory hallucinations (Li et al., 2023; Mazzola et al., 2017). Through precise stimulation of specific brain regions, researchers can repeatedly induce identical perceptual experiences, thereby directly exploring causal relationships between brain regions and hallucinatory behaviors (Siddiqi et al., 2022).

2.2.4 Conditioned Hallucinations

Inspired by the phenomenology of hallucinations, researchers have recently proposed behavioral methods to objectively study them. As previously noted, hallucinations represent subjectively compelling misperceptions that share similarities with veridical perception in subjective experience. Consequently, researchers operationally define hallucinations as "confident false alarms" and quantify them through sensory detection tasks (Rogers et al., 2021). For example, in an auditory detection task, individuals must determine whether a target signal is present in background noise and rate their confidence level. Studies have demonstrated that experimentally induced conditioned hallucinations share highly similar neural mechanisms with spontaneous hallucinations, making them a valuable translational model for investigating hallucination symptoms (Kafadar et al., 2022; Powers et al., 2017; Schmack et al., 2021).

The incidence of conditioned hallucinations shows significant correlation with the severity of hallucinations outside the laboratory, and this correlation is stronger than that of other psychopathological dimensions (Benrimoh et al., 2024; Kafadar et al., 2022; Powers et al., 2017; Schmack et al., 2021). Moreover, as a state-sensitive marker of hallucination susceptibility (Benrimoh et al., 2024; Kafadar et al., 2022), conditioned hallucinations provide an ideal target for intervention research. Notably, the neural mechanisms exhibited by individuals—both psychiatric patients and healthy subjects—during conditioned hallucinations are highly similar to those of spontaneous hallucinations in psychotic patients (Jardri et al., 2011; Zmigrod et al., 2016). Therefore, conditioned hallucinations are emerging as a highly promising translational model that not only facilitates exploration of hallucination neural mechanisms but also provides crucial theoretical foundations and research directions for identifying novel therapeutic targets for schizophrenia and other psychiatric disorders (Matamales, 2021; Schmack et al., 2021).

3.1 Bottom-Up and Top-Down Processing

Perception arises from the integration of bottom-up external sensory stimuli and top-down internal expectations. When the precision of sensory evidence decreases or the weighting of prior expectations increases, the relative importance of prior information in perceptual processing is significantly enhanced, providing a potential neural basis for hallucinations. Numerous neuroimaging and computational psychiatry studies have confirmed that distorted bottom-up sensory signals and excessive top-down predictive signals constitute the core pathological mechanism of hallucination generation (Ffytche et al., 1998; Horga et al., 2014; Jardri et al., 2011; Powers et al., 2017). During hallucinations, activation in relevant sensory cortices increases significantly, providing specific content for the hallucinatory experience. For example, the auditory cortex of patients with auditory hallucinations shows abnormal hyperactivity during hallucination episodes, particularly in lateral temporal regions including Heschl’ s gyrus, planum temporale, and superior temporal gyrus (Hamilton et al., 2021). Furthermore, even during non-hallucination states, individuals with hallucination symptoms exhibit significant alterations in baseline activity of sensory cortices (Diederen et al., 2010; Horga et al., 2014), suggesting that their perceptual systems may harbor inherent neural activity biases. Even in the absence of external auditory stimulation, the auditory cortex of patients with auditory hallucinations maintains elevated background activity levels, which may predispose the brain to misinterpret internally generated signals as external sounds, thereby facilitating hallucination occurrence.

Auditory hallucination generation is closely associated with increased activation of the insular cortex (Zmigrod et al., 2016). Intracranial electrical stimulation studies have further confirmed that direct stimulation of the insula not only induces auditory hallucinations but can also trigger olfactory and gustatory hallucinations (Jaroszynski et al., 2022; Li et al., 2023; Mazzola et al., 2017). However, visual hallucinations are not accompanied by significant insular activation, which may reflect inherent differences between hallucinations of different sensory modalities or methodological limitations (Zmigrod et al., 2016). Additionally, research has found that individuals with hallucinations exhibit stronger prior expectations for signal occurrence during perceptual tasks, and these expectations are directly associated with increased insular activation (Powers et al., 2017), suggesting that the insula may play a critical role in integrating prior expectations with sensory information.

Top-down processing in hallucinations also involves coordinated activity across multiple higher-order brain regions. The prefrontal cortex (PFC) plays a key role in regulating the integration of prior expectations and sensory evidence. Anatomical studies indicate that the PFC modulates auditory association cortex through two projection pathways: a direct pathway from PFC to auditory association cortex responsible for rapid sensory signal modulation, and an indirect pathway via PFC-basal ganglia-thalamus-auditory association cortex multisynaptic connections, with the striatum as a hub mediating abnormally enhanced dopaminergic transmission that ultimately leads to excessive sensory signal integration (Horga & Abi-Dargham, 2019). The ventromedial prefrontal cortex (VMPFC) acts as a "neural switch," with its activity intensity dynamically regulating hallucination onset and termination. When VMPFC activation increases, the weighting of prior beliefs rises and reliance on external sensory evidence decreases, causing internal memory fragments to be misidentified as veridical perceptions; conversely, when inhibitory VMPFC regulation dominates, the balance between endogenous and exogenous information is restored (Hugdahl et al., 2023). This region may influence the distinction between internally generated information (such as memory fragments) and external sensory input by modulating the balance between excitatory and inhibitory neural activity (Jardri et al., 2016; Steinmann et al., 2019). The role of the striatum in hallucinations is also noteworthy. Research has found that dopamine release in the tail of the striatum is closely related to perceptual expectations, with elevated activity predicting high-confidence false alarms (i.e., conditioned hallucinations), while dopamine release in the ventral striatum encodes reward expectation; together, these mechanisms drive hallucination generation through abnormal enhancement of prior weighting (Schmack et al., 2021).

The anterior cingulate cortex (ACC) is closely associated with error monitoring and conflict processing, and its abnormal activity may prevent effective suppression of intrusive irrelevant memories (Ebitz & Hayden, 2016; Kolling et al., 2016), thereby inducing hallucinations. The temporoparietal junction (TPJ), serving as a hub for multimodal information integration, is involved in maintaining the sense of agency, and its dysfunction may cause individuals to misattribute internally generated voices to external sources (Salgado-Pineda et al., 2022). Functional deficits in the supplementary motor area (SMA) may lead to erroneous processing of endogenous language signals, facilitating the generation of linguistic content in hallucinations; this mechanism is manifested as significantly reduced SMA activation in patients with auditory hallucinations (Allen et al., 2008). The cerebellum participates in predicting sensory consequences of self-generated actions (such as speech), and its functional abnormalities may cause mismatches between predicted signals and actual feedback. In auditory verbal hallucinations, this prediction error is thought to prompt the brain to misidentify internal speech as external, while disrupted connectivity in the cerebellum-thalamus-cortex circuit further impairs the precision of sensorimotor integration (Pinheiro et al., 2020). These brain regions collectively constitute a complex neural network that modulates hallucination occurrence and expression by regulating perceptual information processing and integration (Figure 2 [Figure 2: see original paper]).

Figure 2. Schematic diagram of neural circuits involved in hallucination generation. The relative positions and neural circuits of brain regions associated with hallucination production. Cortico-basal ganglia-thalamo-cortical loop: Orange lines indicate feedforward circuits from auditory association areas to prefrontal cortex and striatum; blue lines indicate feedback circuits, involving a direct pathway from lateral prefrontal cortex to auditory association areas (solid line) and an indirect pathway via basal ganglia-thalamo-cortical loops (dashed line).

3.2 Role of the Hippocampus and Parahippocampus

Phenomenological studies of hallucinations indicate that hallucinatory experiences are often closely related to individual memory content. For example, 39% of patients report that their auditory hallucinations represent replays of past experiences, while 55% state that the themes of auditory hallucinations are related to previous memory content (McCarthy-Jones et al., 2014). These findings suggest that hallucination generation may be intimately linked to abnormal memory processes. Further research has shown that individuals with hallucination symptoms experience significant difficulties in identifying memory sources, such as distinguishing between self-generated and externally input events (Brébion et al., 2000). Moreover, compared to individuals without hallucinations, those with hallucinations are more prone to false alarms regarding distractors from previous tasks, indicating an inability to effectively suppress task-irrelevant information (Michie et al., 2005). This tendency to misattribute internal memories to external sources may represent an important mechanism underlying hallucination occurrence (Frith & Done, 1989).

The hippocampus and parahippocampus, which play central roles in memory retrieval and integration, are also crucial in hallucination generation. Research has found that individuals with Lewy body disease who experience hallucinations show significantly higher Lewy body density in the parahippocampal cortex compared to those without hallucinations (Harding et al., 2002). Neuroimaging studies have further revealed that prior to hallucination onset, the parahippocampal cortex undergoes transient deactivation, subsequently triggering activation of bilateral temporal language areas involved in speech perception and initiating spontaneous re-experiencing of memory fragments—that is, hallucination generation (Diederen et al., 2010). Hippocampal activity is associated not only with auditory (Jardri et al., 2011; Perez-Rando et al., 2023) and visual hallucinations (Behrendt, 2016) but also with multisensory hallucinations (Lefebvre et al., 2016). A real-time capture study of auditory hallucinations found that hippocampal theta band power (4–10 Hz) transiently decreases before patients report hallucinations (Van Lutterveld et al., 2012). Theta oscillations are considered a key mechanism by which the hippocampus coordinates distributed network activity, analogous to a conductor in an orchestra, temporarily sequencing perceptual, motor, and memory information through synchronized neural activity across different brain regions (Buzsáki, 2002; Lisman & Buzsaki, 2008). Researchers have further discovered that altered hippocampal neural oscillations drive abnormal network functional connectivity in patients with hallucinations (Hare et al., 2018). Therefore, abnormal activity in the hippocampus and parahippocampus may trigger intrusive re-experiencing of memory fragments, bringing them into conscious awareness and resulting in hallucination generation (Jardri et al., 2013). These findings not only deepen our understanding of hallucination neural mechanisms but also suggest that future research should further investigate the causal role of the hippocampal-parahippocampal network in hallucination formation and evaluate intervention strategies targeting functional abnormalities in this network to improve clinical management of hallucination-related symptoms.

4 Computational Mechanisms of Hallucinations

The recent emergence of computational psychiatry has provided novel perspectives for understanding the computational and neural mechanisms underlying hallucinations. By constructing mathematical models and computational frameworks, researchers can simulate potential abnormalities in brain information processing, thereby revealing underlying mechanisms of hallucination generation (Friston, 2005; Powers et al., 2017; Sheldon et al., 2022; Sterzer et al., 2018). Previous researchers simulated excessive pruning of computational units in speech perception networks, resulting in hallucination-like phenomena, demonstrating the critical role of cortical disinhibition in hallucination generation (Hoffman & Dobscha, 1989).

Bayesian models hold significant value for understanding hallucination symptoms (Friston, 2005; Powers et al., 2016). Within this computational framework, perception is conceptualized as a predictive coding process based on sensory evidence and prior beliefs, aiming to provide a probabilistically optimal explanation of the underlying causes of sensory events. This approach has proven robust in simulating various perceptual neural information processing mechanisms (Alais & Burr, 2004). The core principle of Bayesian models is that the brain continuously updates the weighting between prior beliefs and sensory evidence to generate optimal estimates of the external world. Under normal circumstances, this mechanism efficiently integrates ambiguous sensory information, such as speech comprehension in noisy environments. However, in pathological states, abnormal enhancement of prior weighting or decreased sensory precision may cause the perceptual system to generate inferences that deviate from actual input, thereby inducing hallucinations (Cassidy et al., 2018).

Research demonstrates that abnormal weighting of prior beliefs plays a critical role in hallucination generation. Prior beliefs adjust perceptual system response biases to minimize errors—when individuals encounter uncertain stimuli, the system preferentially selects perceptual hypotheses that better match prior probability distributions (Summerfield & De Lange, 2014). While this strategy reduces overall prediction error, it leads to erroneous confirmation of expected options (Horga & Abi-Dargham, 2019). When sensory input reliability decreases, the brain may compensate for uncertain sensory evidence by enhancing prior weighting, forming a "hallucination as perceptual compensation" mechanism (Benrimoh et al., 2019, 2024). Notably, this perceptual optimization mechanism may incur adaptive costs under specific conditions: when systematic perceptual biases exist, such as sensory signal attenuation or noise interference, optimal inference manifests as simultaneous increases in both hit rates and false alarm rates. Temporally, prior beliefs driving perceptual inferences may derive from either accumulation of short-term sensory experience or stable patterns encoded by long-term implicit memory.

The relative weighting of priors depends on the ratio between their precision and sensory evidence precision: when sensory input precision decreases absolutely (e.g., sensory signal attenuation) or prior precision increases absolutely (e.g., overconsolidated expectations), the dominance of priors in perceptual inference is enhanced. This phenomenon of "relative prior hyper-precision" is particularly pronounced in patients with hallucinations. Empirical studies have shown that clinical populations with hallucination symptoms and individuals at high risk for psychotic disorders exhibit abnormally increased prior weighting during perceptual decision-making (Kafadar et al., 2020; Powers et al., 2017).

The dopamine system plays an important role in modulating prior weighting. Research suggests that the mesolimbic dopamine pathway may influence prior weight encoding by regulating prediction error signals (Cassidy et al., 2018). For example, dopaminergic hyperactivity may enhance reliance on prior beliefs, causing the perceptual system to preferentially "fill in" uncertain sensory input with internal models (Benrimoh et al., 2018, 2019). Dopamine receptor agonists can induce hallucination-like experiences in healthy individuals, whereas dopamine antagonists can alleviate hallucination symptoms in psychiatric patients (Cassidy et al., 2018; Schmack et al., 2021). For instance, amphetamine-induced conditioned hallucinations manifest as significant perceptual biases in individuals during sensory tasks, and these biases are closely related to increased striatal dopamine release (Cassidy et al., 2018). Further research has found that prestimulus striatal dopamine release can predict conditioned hallucinations, while dopamine release in the tail of the striatum is significantly correlated with perceptual biases that lead to increased conditioned hallucinations (Schmack et al., 2021). These findings collectively point to the critical role of the striatal dopamine system in modulating prior weighting, providing important experimental evidence for understanding the neurochemical mechanisms of hallucination generation.

In recent years, the Hierarchical Gaussian Filter (HGF) model, as a general Bayesian framework, has provided new research tools for understanding hallucinations. The HGF model combines reinforcement learning (RL) with probabilistic optimal principles, enabling simulation of individual learning processes under different uncertainty conditions, such as environmental fluctuations and perceptual uncertainty (Kafadar et al., 2022; Powers et al., 2017; Sheldon et al., 2022). The advantage of this model lies in its ability to simultaneously consider multiple parameters including prior beliefs, sensory evidence, and learning rates, thereby more comprehensively revealing the neural mechanisms of hallucinations. Although current research has primarily focused on the role of relative prior hyper-precision in hallucination generation, other parameter distortions in the HGF model (such as learning rate, belief stability, etc.) may also importantly influence hallucination generation (Sheldon et al., 2022). For example, some individuals with hallucinations may experience hallucinations across multiple sensory modalities due to low learning rates, whereas others may develop modality-specific hallucinations due to abnormal relative prior hyper-precision in a particular sensory modality. By integrating the HGF model with other neuromodulation techniques, future research may enable finer differentiation of hallucination subtypes and provide theoretical foundations for personalized treatment. Particularly, combining computational model parameters with neuromodulation target selection may open new avenues for developing more precise intervention strategies.

5 Neuromodulation Interventions for Hallucinations

Hallucination interventions primarily include pharmacological and non-pharmacological approaches (Sommer et al., 2012). Although pharmacological treatments have achieved some success in alleviating hallucination symptoms, up to 30% of schizophrenia patients show poor medication response, manifesting as treatment-resistant auditory hallucinations (Shergill et al., 1998). Consequently, researchers have gradually turned their attention to non-pharmacological treatments such as cognitive-behavioral therapy (CBT), adjunctive electroconvulsive therapy (ECT), and non-invasive brain stimulation techniques including transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) (Jiang et al., 2022; Marzouk et al., 2020; Sommer et al., 2012). These methods offer new possibilities for hallucination treatment by directly or indirectly modulating brain neural activity.

The left temporoparietal junction (TPJ) is the most common target for TMS treatment of hallucinations. Early small-sample studies suggested that TMS stimulation of the left TPJ produced moderate to good therapeutic effects on hallucination symptoms (Hoffman et al., 1999). However, large-scale clinical trials in recent years have shown that these positive effects are not stable and may even disappear completely in some studies (Slotema et al., 2011). Additionally, researchers have attempted to target frontal regions, particularly the dorsolateral prefrontal cortex, but treatment effects have similarly shown significant instability (Marzouk et al., 2020). These inconsistent results may be related to stimulation frequency, individual patient differences, and heterogeneity of hallucination subtypes. For example, different low-frequency stimulations, such as 1 Hz and 10 Hz, show different efficacies, suggesting that stimulation parameter selection requires further optimization (Marzouk et al., 2020; Xie et al., 2022). Recent studies indicate that image-guided repetitive TMS (rTMS) can safely and effectively alleviate auditory verbal hallucinations in schizophrenia patients, with its efficacy significantly correlated with electric field strength in individualized auditory hallucination brain networks (Hua et al., 2024), suggesting that precise optimization of electric field parameters may be key to enhancing rTMS intervention effects.

Addressing the instability of TMS treatment effects, researchers have proposed that hallucination treatment requires individualized design (Klirova et al., 2013) and computational modeling (Huys et al., 2016). The core of individualized treatment lies in selecting optimal stimulation targets and parameters based on patients' clinical symptoms, neuroimaging data, and computational model predictions. For example, functional magnetic resonance imaging (fMRI) or electroencephalography (EEG) can precisely identify abnormal neural activity regions or frequency bands in patients' brains related to hallucinations, thereby optimizing TMS target localization and intervention frequency bands. Additionally, computational modeling can help predict the modulatory effects of different stimulation parameters on patients' neural circuits, providing important references for developing personalized treatment plans (Huys et al., 2016).

Beyond TMS, other non-pharmacological treatments have also shown potential in hallucination intervention. CBT helps alleviate auditory hallucination symptoms by assisting patients in identifying and correcting cognitive errors related to hallucinations (Shukla et al., 2021). ECT modulates brain neurotransmitter levels and neural network activity by inducing brief seizures, demonstrating significant efficacy in some treatment-resistant hallucination patients (Nieuwdorp et al., 2015).

Despite progress in non-pharmacological treatments for hallucinations, numerous challenges remain. First, treatment responses vary significantly across patients, possibly related to heterogeneity of hallucination subtypes and individual differences in neural circuits. Second, existing research has primarily focused on evaluating the efficacy of single intervention methods, with few studies investigating the effects of combined approaches. Furthermore, how to integrate computational modeling with clinical practice to achieve truly individualized treatment remains a key focus for future research.

6 Future Research Perspectives

Although significant progress has been made in understanding the neural mechanisms of hallucinations in recent years, many unanswered questions remain regarding the specific neural circuits involved and their interaction mechanisms (Horga & Abi-Dargham, 2019; Sheldon et al., 2022). Below, we outline future research directions and challenges from four dimensions.

First, current research has identified multiple brain regions related to hallucinations, such as the prefrontal cortex, temporoparietal junction, insula, hippocampus, and parahippocampus (Allen et al., 2008; Cassidy et al., 2018; Hugdahl et al., 2023). However, the specific functions of these regions in hallucination generation and how they interact to induce hallucinations remain unclear. Current studies predominantly employ single-region analysis methods while neglecting the distributed network patterns involved in hallucinations. Future research should integrate multimodal neuroimaging techniques to analyze dynamic interaction mechanisms among these brain regions from a whole-network perspective, with particular focus on whether hallucinations of different modalities depend on the same or different neural networks, to construct a more complete neural circuit model of hallucinations.

Second, cross-modal comparative research remains limited by experimental paradigm differences and urgently requires breakthroughs in three aspects: (1) developing standardized multimodal induction paradigms to enable comparative studies of multimodal hallucinations within a unified framework; (2) adopting a Bayesian predictive coding framework to quantitatively analyze prediction error signals across different modalities; and (3) establishing cross-diagnostic cohorts to systematically examine interactions between disease-specific (e.g., schizophrenia, Parkinson' s disease) and modality-specific (e.g., auditory, visual) effects. This integrated research strategy will help reveal the core neural and computational mechanisms underlying hallucination generation.

Third, fMRI remains the primary tool for studying hallucination-related brain activity, but its limited temporal and spatial resolution leaves the information encoding rules and circuit transmission mechanisms underlying hallucination generation unclear. For example, it remains uncertain how oscillatory patterns in cortico-basal ganglia-thalamic circuits affect information representation during hallucinations, or what the pathways of abnormal information flow between different brain regions are. Addressing these challenges requires establishing a multimodal technology fusion platform: spatially, integrating 7T ultra-high-field MRI (Viessmann & Polimeni, 2021) with optogenetic imaging to achieve cross-scale observation from whole-brain networks to cell populations; temporally, combining intracranial EEG with calcium signal fiber photometry to capture millisecond-level neural activity features. Notably, such technological integration must be combined with computational psychiatry methods to decode the mapping relationships between neural activity patterns and clinical symptoms through deep learning algorithms.

Fourth, existing research is largely based on correlational analyses, lacking direct causal evidence for the role of specific brain regions or neural circuits in hallucination generation. Current causal intervention research faces two major challenges: the lack of precise hallucination biomarkers to guide target selection, and the difficulty in timing interventions due to the intermittent nature of hallucination episodes. Future research strategies may include: establishing individualized target prediction models based on computational psychiatry (Huys et al., 2016) to identify neural activity features closely related to hallucination symptoms; applying closed-loop neuromodulation technology (Fried, 2023; Widge et al., 2018) to implement precise interventions during symptom onset; and combining Bayesian causal inference models (Kafadar et al., 2022) to differentiate between necessary and sufficient causal relationships. Additionally, integrating causal intervention techniques (such as intracranial electrical stimulation) with behavioral experiments can further verify the causal roles of specific brain regions or neural circuits in hallucination formation. This strategy holds promise for enhancing the precision of hallucination interventions and providing scientific foundations for personalized treatment.

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