Am J Transl Res 2020;12(12):7657-7669
www.ajtr.org /ISSN:1943-8141/AJTR0119848

Review Article

Neural mechanisms underlying visual and auditory processing impairments in schizophrenia: insight into the etiology and implications for tailoring preventive and therapeutic interventions

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Received August 8, 2020; Accepted October 28, 2020; Epub December 15, 2020; Published December 30, 2020

Abstract: Schizophrenia is a complex and devastating neuropsychiatric disorder with an unknown etiology. Patients with schizophrenia have a high prevalence of visual disturbances, commonly accompanied by auditory impairments. In recent review articles, the perceptual deficits of visual and auditory sensory processing have been downplayed. However, visual and auditory impairments are associated with hallucinations, which is characteristic of schizophrenia across all cultures. Despite decades of research, the common neural mechanisms underlying hallucinations remain largely unknown. In recent years, neuroimaging technologies have empowered researchers to investigate the underlying neural mechanisms. In this review article, we performed a literature search of studies that assessed visual and auditory processing impairments, along with their relationship to visual disturbances and auditory hallucinations, in schizophrenia. We proposed that the pulvinar may play a critical role. In addition, disrupted visual and auditory projections from the pulvinar to the visual and auditory cortices could be shared pathways in relation to visual disturbances and auditory hallucinations in schizophrenia. Our findings suggest that early visual and auditory processing deficits may occur before the onset of the initial psychotic episode, including hallucinations, and the full manifestation of schizophrenia. Furthermore, we discussed the directions for future studies. Our findings from this review offer unique insights into the distinct underlying neural mechanisms of schizophrenia, which may help develop tailored preventive and therapeutic interventions in the future.

Keywords: Schizophrenia, visual disturbances, visual hallucinations, auditory hallucinations, pulvinar, fMRI

Introduction

Schizophrenia is a devastating neuropsychiatric disorder that usually strikes during late adolescence or early adulthood. Patients who suffer from schizophrenia have a higher prevalence of visual and auditory impairments. In addition, auditory and visual hallucinations are more prevalent in schizophrenia than other neuropsychological disorders, such as bipolar disorder. Indeed, hallucinations are among the most common symptoms of schizophrenia across all cultures worldwide [1-8]. The perceptions formed based on experiencing unreal things are usually instructive and endangering behaviors. Thus, it is clinically and scientifically significant to pinpoint the neural mechanisms underlying auditory and visual hallucinations in schizophrenia.

After decades of research, scientists have gained valuable insight into the pathological mechanisms that drive the core features of visual and auditory perceptions in patients with schizophrenia. Notably, the majority of previous original research articles and review articles have focused on investigating roles of high order cerebral functions (e.g. cognition, memory, social functioning, and other intellectual processes) in schizophrenia. It has been pro-

posed that the optimal combination of sensory evidence with prior knowledge and beliefs may contribute to perceptions in schizophrenia [9, 10]. In addition, the hallucinations have been linked to brain-based deficits of the auditory and visual sensory processing systems [11, 12]. However, compared to high order functions in previous review articles, visual and auditory sensory processing impairments, along with associated visual and auditory perceptions, have been downplayed and undertreated in the field of schizophrenia [13-16].

Advanced neuroimaging technologies have empowered researchers to investigate the neural mechanisms underlying visual and auditory processing impairments and the core symptoms of schizophrenia. In recent years, notable progress has been made in understanding of the structural and functional alterations in specific regions of the brain, as well as the relationship between visual and auditory deficits and hallucinations in schizophrenia. In particular, the most recent findings suggest that the pulvinar may play a critical role in visual and auditory hallucinations, and that disruption of visual and auditory projections from the pulvinar through the visual and auditory cortices could be shared pathways in visual disturbances and auditory hallucinations in schizophrenia.

In this review, we conducted a comprehensive literature search and evaluation of existing studies about visual and auditory processing impairments and their connection with hallucinations in schizophrenia. We aimed to synthesize the central findings to further advance the community's understanding of the neural mechanisms involved in schizophrenia. We also discussed the clinical implications of neural mechanisms, in addition to future research directions. Understanding these mechanisms may help guide the development and tailoring of preventive and therapeutic interventions for schizophrenia in the future.

Auditory impairments and hallucinations in schizophrenia, along with the associated neural mechanisms

Auditory processing impairments in schizophrenia

The association between hearing impairments and schizophrenia has been evidenced by the findings in patients with 22q11.2 deletion syndrome (22qDS), which is a copy number variation (CNV) syndrome arising from a microdeletion of more than 30 genes spanning 1.5-3 Mb in length on chromosome 22 band q11 [17]. Individuals with 22qDS frequently suffer from hearing loss and cognitive impairments, with a large fraction of 22qDS adults having schizophrenia, for which 22qDS is considered one of the most significant risk factors for schizophrenia [17-22]. Strikingly, the lifetime prevalence of schizophrenia, together with symptoms of auditory hallucinations, is very high, up to 35% in individuals with 22q11.2DS, considerably higher than that of the general population at approximately 1% [21, 23]. Mancini *et al*. studied a cohort of 120 patients with 22q11.2DS and 110 healthy individuals [23]. During the 19-year follow-up period, spanning from childhood to adulthood, brain imaging and genetic analyses were regularly performed, and up to 35% of patients developed schizophrenia and experienced symptoms of auditory hallucinations [23]. The volume of the thalamic nuclei was smaller in the patients with 22q11.2DS than the healthy controls. Notably, the connectivity between the thalamus and brain regions associated with processing hearing and Wernicke's area was higher in 22q11.2DS patients experiencing auditory hallucinations than patients without the symptoms. Specifically, 22q11.2DS patients with auditory hallucinations had increased connectivity of the medial geniculate nucleus (MGN) and anteroventral nucleus with the auditory cortex (AC) and the Wernicke's area. More importantly, this connectivity is normal during childhood and adolescence, yet it does reach maturity by adulthood [23]. Recent findings have linked the onset of auditory hallucinations to the hyperconnectivity mentioned above, which could be attributed to the immature pattern of thalamocortical connectivity. These important novel findings may explain the auditory hallucinations in patients with 22q11.2DS, advancing our understanding of the pathophysiology of schizophrenia.

Structural and functional impairments in the central auditory system, and their correlation with auditory hallucinations in schizophrenia

Both structural and functional impairments in auditory processing are commonly found in schizophrenia, and the deficits directly contrib-

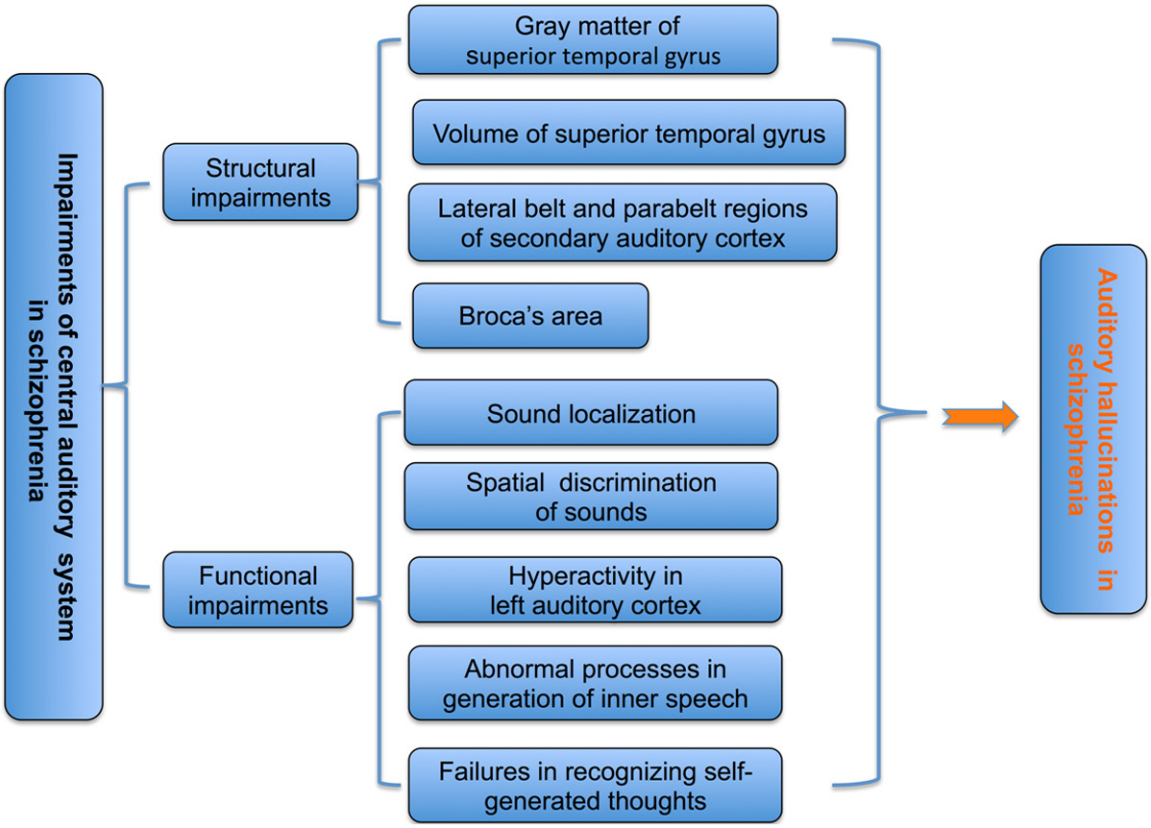

Figure 1. Schematic illustration of structural and functional impairments in the central auditory system and the correlation with auditory hallucinations in schizophrenia. Main structural and functional impairments in the central auditory processing system in patients with schizophrenia are schematically illustrated. The deficits in both structure and function of the central auditory system directly contribute to auditory hallucinations in schizophrenia patients.

ute to auditory hallucinations and diminished psychosocial function [24]. The auditory processing system consists of two subsystems, including the peripheral auditory system and the central auditory system (i.e., the cochlear nucleus to the auditory cortex). In routine hearing tests, schizophrenia patients usually show normal performance, suggesting that the peripheral auditory system is intact in schizophrenia, and that impairments in the central auditory system and possibly cortical level deficits are potentially involved in schizophrenia [25, 26].

Neuroimaging technologies [e.g. magnetic resonance imaging (MRI), functional MRI (fMRI), functional connectivity MRI (fcMRI)] have revealed both structural and functional brain alterations of the central auditory processing system in patients with schizophrenia, and the main findings were schematically illustrated in **Figure 1**.

The structure of the auditory cortex is complex, and some researchers are still uncovering new facts about the processing of auditory information. There are three primary regions in the auditory cortex, including the primary auditory cortex, secondary auditory cortex (i.e., lateral belt and parabelt regions), and the belt region. Post-mortem histology and MRI have verified structural alterations in the specific brain regions involved in auditory processing [24]. In a multisite fMRI study of auditory hallucinations in patients with schizophrenia, significant reductions in the volumes of the auditory cortex, along with alterations in the voxel-based morphometry [27-29] and functional hypersensitivity of the auditory cortex [24, 30], were detected. In recent MRI studies of schizophrenia, a significant reduction in the volume of the superior temporal gyrus (STG), a part of the secondary auditory cortex, has been widely reported in the literature. Notably, these abnormalities are often present at the initial

diagnosis or even before the onset of the neuropsychiatric disorder [31-34]. The phenomenon is specific to schizophrenia, as STG alterations have not been detected in patients with bipolar disorder who exhibit hallucinations and those individuals with chronic alcohol usage-induced hallucinations [35, 36]. Furthermore, these abnormal reductions in the volume of the STG grey matter are independent of antipsychotic medications. Interestingly, the period of adolescence, when the abnormal changes develop in the grey matter of the auditory cortex, has been well recognized as the onset of the first episode of psychosis, including auditory hallucinations, in a majority of schizophrenia cases [37-39]. These findings are particularly important, as they indicate that schizophrenia could be, at least in part, a consequence of these structural impairments in the STG grey matter of the auditory cortex [37-39]. These histological and structural findings have shown which regions should be targeted in developing potential therapeutic approaches to treat the characteristic symptoms of patients with schizophrenia.

There is increasing recognition of functional impairments in the central auditory processing system (**Figure 1**). A number of previous studies indicated abnormalities of sound localization, auditory cortical spatial localization abilities, spatial discrimination of sounds, activity of the left auditory cortex in schizophrenia patients with auditory hallucinations [24, 40-42]. A research team led by Daniel Javitt found that spatial localization impairments were correlated with auditory hallucinations in schizophrenia [40, 41]. Perrin and colleagues demonstrated that spatial localization ability is significantly diminished in patients with schizophrenia [40]. Built upon these novel findings, the research team further investigated the spatial location ability, including sound localization and spatial discrimination tasks, using location mismatch negativity (MMN). As a result, patients with schizophrenia or schizoaffective disorder exhibited significantly impaired MMN generation location compared with healthy controls [41]. These results identified a strong association between spatial localization and auditory hallucinations in schizophrenia. Still, there was no evidence of connections between AVH and other aspects of auditory dysfunction (e.g., impaired tone matching ability, MMN generation to pitch- or duration-deviants). These novel findings have also provided evidence in support of models that attribute persistent auditory hallucinations to impaired source-monitoring and may help suggest new approaches for therapeutic intervention for auditory hallucinations in patients with schizophrenia.

Potential neural mechanisms underlying the relationship between auditory processing impairments and auditory hallucinations in schizophrenia

There is limited knowledge about the neural mechanisms that cause auditory hallucinations in schizophrenia. Previous studies have reported that structural and functional abnormalities in specific sensory processing regions, such as the primary auditory cortex or Broca's area of the brain for auditory processing [43-45], including external and internal voice processing, are associated with auditory hallucinations in schizophrenia. It has been proposed that hyperactivity in the left auditory cortex, failures in recognizing self-generated thoughts, and abnormal processes involved in the generation of inner speech may contribute to auditory hallucinations [24, 40-42].

Recently, Gevonden and colleagues assessed the impact of severe hearing impairment (SHI) on dopamine function in a neuroreceptor imaging study using single-photon emission computed tomography (SPECT) in nonpsychotic individuals: SHI young adults versus normal-hearing healthy controls [46]. The participants in the SHI group and normal-hearing healthy control group underwent two SPECT scans with the dopamine D-2/3 antagonist, I-123 iodobenzamide, which is sensitive to and commonly used for detecting alterations in dopamine concentrations in the synapses of neurons. Notably, no significant differences in baseline striatal D-2/3 receptor binding were detected between the two groups, suggesting that there was no impact of hearing impairment on dopamine release [46], which may rule out the possible impact of hearing impairment on the main neuronal transmitters (e.g., dopamine) in schizophrenia. Considering the correlation between dopamine levels and psychosis, these findings also imply that alternative pathological mechanisms, rather than neurochemical theory, could account for the relationship between hearing impairment and schizophrenia symptoms.

Impairments in brain-based visual processing, visual hallucinations in schizophrenia, and neural mechanisms

Structural and functional impairments in the brain-based visual processing system in schizophrenia

Previous studies have convincingly demonstrated, in patients with schizophrenia, that visual impairments occur more frequently in patients with visual hallucinations than those patients without visual hallucinations [47, 48]. Some studies have shown that schizophrenia may impair a person's ability to process visual images from the outside world effectively. Specifically, mounting evidence has shown that patients with schizophrenia are unable to properly filter visual information at the input stage, which leads to the distortion of visual images.

Neuroimaging studies in patients with schizophrenia have shown structural and functional alterations in the brain regions that are most important for visual processing, including reduced cortical thickness in the superior parietal and parieto-occipital regions, along with decreased functional connectivity in the parietal, temporal, and left occipital lobes. A research team from the U.S. has conducted several comprehensive and seminal studies on the link between schizophrenia and dysfunctional or distorted visual perception [49-51]. Using state-of-the-art brain imaging techniques, such as fcMRI and high-field MRI, in combination with psychophysical tasks, the research team has shown that schizophrenia patients have difficulties in detecting the contours of visual targets [49-51]. It is known that defining the outer boundaries of a visual object is an essential component for visual perception in many visual processing tasks (e.g., seeing and distinguishing objects from the surrounding environment). Schallmo and colleagues performed several studies to assess the contour detection of visual targets in patients with schizophrenia, their unaffected first-degree relatives (FDRs), healthy control individuals, and patients with bipolar disorder [50, 51]. The results showed that patients with schizophrenia performed significantly weaker in detecting the contours of visual targets than their FDRs, who were unaffected by schizophrenia, and the healthy control individuals [50, 51]. Schizophrenia patients frequently exhibited abnormalities in visual contour integration and backward visual masking [52], which impedes the detection of visual target (the test stimulus) in the presence of the second stimulus (the masking stimulus) [12, 53, 54]. To generate a perception of a visual object on the basis of missing or partial visual information is defined as perceptual closure, which is commonly observed in patients with schizophrenia [55].

In addition to the detection of contours as an essential component in visual perception, the modulation of visual context, also known as the modulation of the central visual object by the presence and configuration of the surrounding environment, is considered to be another important component involved in visual perception. Several previous studies have shown that the impact of surrounding context is reduced in schizophrenia, especially surrounding suppression, a specific form of contextual modulation in which the perceived contrast of center stimuli is reduced due to a high-contrast surround or by a particularly enlarged stimulus. Contrast perception was found to be weaker in schizophrenia patients than healthy controls [56-59]. Most recently, Pokoney and colleagues evaluated the impact of perceptual contexts, such as the environment surrounding the visual target in patients with schizophrenia, their unaffected FDRs, and healthy control individuals. The patients with schizophrenia were affected to a lesser degree than the healthy controls by the environment surrounding the visual target, indicating a reduction in the influence of perceptual context [49]. These results, along with those of other studies [56-59], demonstrate the importance of accurately detecting the contours of visual targets, significantly diminished in schizophrenia [49-51]. Furthermore, the research team also investigated the neural mechanisms underlying abnormal or distorted visual perception in patients with schizophrenia. These findings from the research team [49-51], together with the earlier studies on the perception of contours in schizophrenia showing abnormalities in early visual cortical function and higher-level function in schizophrenia [53, 60-63], have suggested that the early and late stages of brain-based visual processing or high-order visual processing and cognition are involved in the dysfunctional or distorted visual perception

associated with schizophrenia. The deficits in both contour integration and surround suppression occur during later stages of visual processing and involve higher-order perceptual processing [64-66]. In a recent study, Pokoney et al. examined neural correlates of disrupted contour integration in schizophrenia, as reflected by event-related potentials (ERPs) earlier and later components [49]. ERPs are recorded in the scalp-recorded electroencephalography (EEG), allowing for a better understanding of the neural response to visual contour stimuli, deficits in neural processing, and the modulation by surrounding context. As a result, late visual (occipital P2, 190-290 ms) and cognitive (centroparietal P3, 350-650 ms) neural responses were significantly associated with the indices of contextual modulation behavior, suggesting abnormalities in visual context modulation in schizophrenia [49]. Notably, the P3 in perceptual literature is referred to as the centroparietal positivity (CPP), dependent on the subjective perceptual experience of top-down processing [67]. There is a possibility that smaller P3 amplitudes could be required for accurate detection of contours, considering the diminished surround suppression in schizophrenia [49]. It has been postulated that the P2 amplitudes might reflect visual processing at the intermediate stage in the occipital cortex, while P3 amplitudes might reflect top-down processing during the later stage and the accumulation of information in the parietal-frontal cognitive network [49]. In fact, impairments in higher-order cognitive processing are frequently observed in patients with schizophrenia [68], while deficits in visual processing and other sensory processing deficits are usually underplayed [24]. Hence, the subtle impairments of visual and auditory processing during the early stages may precede and progress into the high-order perceptual impairments as prevalently seen in patients with schizophrenia [25, 26, 69, 70]. These findings have advanced our understanding of the deficits in contour integration and abnormal modulation of visual context in schizophrenia.

Potential neural mechanisms underlying the correlation between the brain-based visual processing impairments and visual hallucinations in schizophrenia

The structural and functional impairments in the brain-based visual processing system may be involved in experiencing visual hallucinations in schizophrenia. Although there is the link between dysfunctional or distorted visual perception and schizophrenia, with the involvement of the early and late stages in the visual processing system or higher-order sensory processes impairments and cognitive processes, the question still remains about how abnormal or distorted visual perception could lead to visual hallucinations in schizophrenia and its associated mechanism.

Visual hallucinations often occurred after injuring the visual processing areas in the parietal and occipital lobes, suggesting that bottom-up impairment could result in visual hallucinations through the hyperactivation of secondary sensory cortices, which offers the perceptual content for knowledge and experience [71-73]. This over-perceptualizing may modulate associated top-down processes in the prefrontal, anterior cingulate, premotor, and cerebellar cortices, leading to a false sense of things that do not exist, and poor self-monitoring. Those affected individuals may experience and perceive the internal visual or auditory information as vivid external perceptions that are not really there. It has been hypothesized that the late stages of brain-based visual processing and cognition are involved, from which dysfunctional feedback signals carry information about prior knowledge, as well as experience about the visual target. When there are deficits in the feedback signals, the brain can fill in the gaps, leading the mind to perceive things that do not exist. Hence, brain-based visual pathology will likely predispose individuals to visual hallucinations in schizophrenia.

Based on the existing data, we propose that visual deficits are likely a cause rather than a consequence. The consistent observation of visual processing deficits in schizophrenia patients, as well as unaffected family members, also suggests that visual disturbances may represent a heritable vulnerability factor for schizophrenia. There are few studies on the pathological features of visual disturbances in patients with first-episode untreated schizophrenia experiencing visual hallucinations. Previous studies are in support of abnormal alterations in the structure and function of the visual cortex. Inspired by the findings that cathodal stimulation over the occipital cortex ameliorates visual hallucinations and that low-fre-

quency rTMS over the occipital cortex improves the symptoms of visual hallucinations [74, 75], identifying the correspondence between visual hallucinations and specific brain areas has important clinical implications.

Hypothetical pathways in relation to both visual disturbances and auditory hallucinations in schizophrenia

A large number of patients with schizophrenia also suffer from visual disturbances, and in some cases, both auditory and visual hallucinations [3, 76-78]. Thus far, the common neural circuit-level pathway of visual and auditory processing concerning both visual disturbances and auditory hallucinations in schizophrenia has not been fully understood.

Pulvinar-cortex interactions are primarily involved in vision- and attention-related functions in the visual processing processes, primarily owing to the reciprocal connections of the pulvinar with the primary and secondary visual cortices [79-83]. More recently, the lateral posterior nucleus (LP), the homolog in rodents of the pulvinar in humans, was shown to play a modulatory role in regulating the activity of the primary auditory cortex [84]. Chou and colleagues investigated, for the first time, the impact of the pulvinar/LP activity on the primary auditory cortex, suggesting that the pulvinar/LP can directly modulate the primary auditory cortex through layer 1 (L1) neurons and layers 2/3 (L2/3) pyramidal neurons. They noted that it exerts a net inhibitory effect in normal conditions. Besides, the study also showed that the pulvinar/LP is activated by signals relayed from the superior colliculus (SC), contributing to the enhancement of the primary auditory cortex (A1) processing when auditory background noise and threatening visual looming stimuli are present. As such, a multisensory bottom-up SC-pulvinar-A1 pathway plays an important role in the contextual, as well as the cross-modality modulation, of auditory cortical processing [84]. Given that the axonal projections from pulvinar/LP to primary sensory cortices usually end in layer 1 (L1), consisting of mainly L1 inhibitory neurons [85, 86], Chou and colleagues demonstrated that the pulvinar/LP exerts an overall inhibitory effect on the primary auditory cortex through L1 inhibitory neurons directly and layers 2/3 (L2/3) pyramidal neurons indirectly [84, 86-88]. On the contrary, abnormalities of the pulvinar/LP are proposed to diminish or even reverse its net inhibitory effect on the primary auditory cortex (**Figure 2**).

Interestingly, abnormalities of the pulvinar nucleus due to functional and structural alterations have been implicated in schizophrenia, including reduced metabolic rates, decreased volumes, and altered confirmations [89, 90]. These alterations in the structure and metabolic rate represent the functional impairment of the pulvinar/LP, which may alleviate the primary auditory cortex's suppression. Hence, this could eventually activate the primary auditory cortex in the absence of an external auditory stimulus, resulting in auditory hallucinations. In fact, abnormal activation of the primary auditory cortex is considered one of the possible models representing the development of auditory hallucinations through functional imaging studies [91-93]. These previous studies revealed activation of the auditory cortex and disruption of auditory-verbal network activity, which is an important working model for auditory hallucinations [91-93]. In agreement with functional imaging findings, it has been clinically noted that patients with schizophrenia who experience auditory hallucinations are more likely to have activated primary auditory cortices than patients with schizophrenia who lack auditory hallucinations. The hyperactivity is correlated with the severity of auditory hallucinations [91-93]. The etiology of combined visual disturbances and auditory hallucinations in schizophrenia remains unclear. However, based on existing literature, we propose that the pulvinar may play a critical role in the disrupted visual and hearing projections from the pulvinar to the visual and auditory cortices. This could be one of the common pathways about both visual disturbances and auditory hallucinations in schizophrenia (**Figure 2**). As such, pulvinar targeting could lead to the development of new preventive and treatment approaches for visual disturbances and auditory hallucinations in schizophrenia.

Future directions of the neural mechanisms for decoding schizophrenia, and implications

In future studies, more integrated and comprehensive models of the brain-based neurological processes associated with visual and auditory processing and hallucinations are ne-

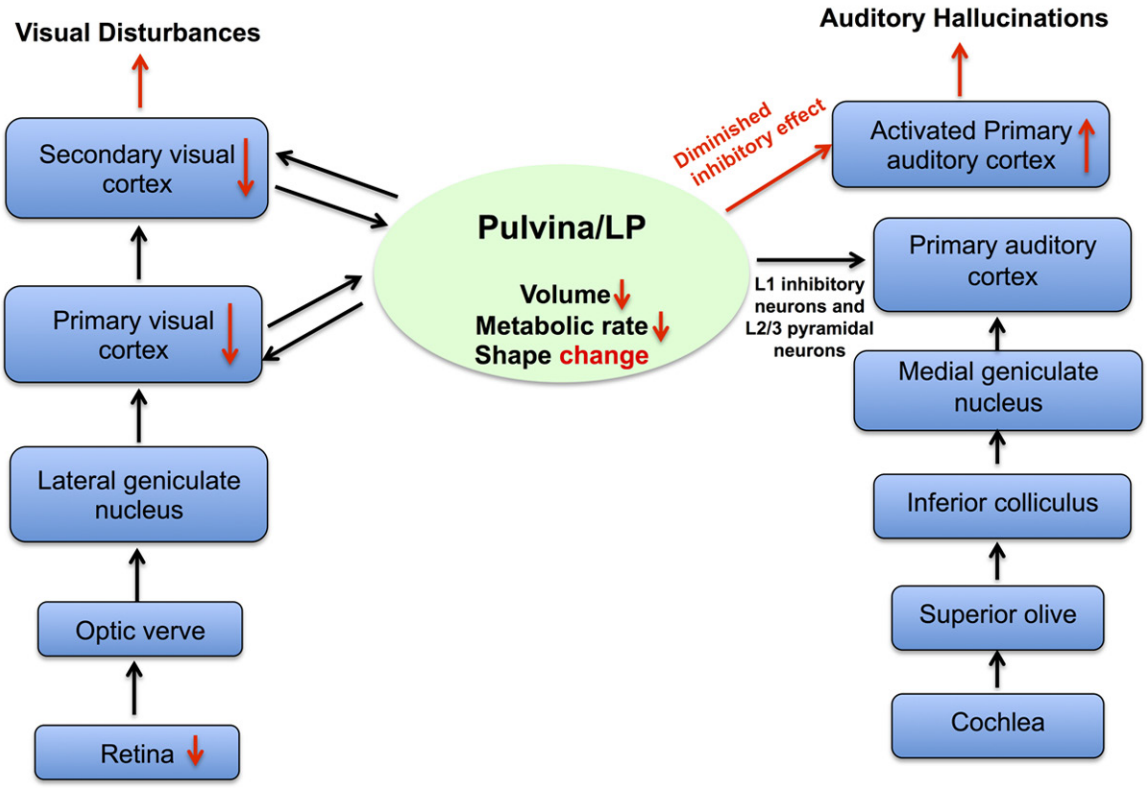

Figure 2. Hypothetical pathways in relation to both visual disturbances and auditory hallucinations in schizophrenia. The pulvinar nucleus/LP directly modulates the primary auditory cortex activity through layer 1 (L1) neurons and layers 2/3 (L2/3) pyramidal neurons, and it exerts a net inhibitory effect in normal conditions. The abnormal alterations of the pulvinar nucleus in schizophrenia include low metabolic rate, decreased volume, and conformational change, which may reduce the suppression and induce the activation of the primary auditory cortex, while also contributing to the development of auditory hallucinations. Compared with the new role of the pulvinar nucleus in the modulation of the primary auditory cortex, there are the reciprocal connections between the pulvinar and visual cortices in visual processing. These disruptions contribute to visual disturbances in schizophrenia. The pulvinar may play a critical role, and the disrupted visual and hearing projections from the pulvinar to the visual and auditory cortices could be a common pathway associated with visual disturbances and auditory hallucinations in schizophrenia. LP, the lateral posterior nucleus, is the homolog in rodents of the pulvinar in mammals.

eded, which are useful to pinpoint the origins of these impairments. In the existing models [73, 94-96], a better comparison between the cognitive and neural processes of hallucinations is also required to understand the roles of hallucinations in schizophrenia. Further endophenotypes could be applied to connect behavioral traits of the phenotypes, such as abnormalities of visual and auditory perceptions, with their respective genes. This will help identify causative factors in schizophrenia's etiology and genetic markers for visual and auditory impairments that could precede hallucinations in schizophrenia.

Visual and auditory hallucinations can be attributed to more than impairments in the visual and auditory cortex. Treatments targeting the sensory cortex in the brain may benefit the medical management of patients with schizophrenia. Some studies have reported that inhibitory brain stimulation methods, including transcranial direct current stimulation (tDCS) and low-frequency repetitive transcranial magnetic stimulation (rTMS), lead to therapeutic effects in patients with schizophrenia who experience hallucinations [97-100], and thus are expected to reduce the severity and frequency of hallucinations.

Conclusions

It has become increasingly evident that brain-based visual and auditory processing is impaired in schizophrenia patients, which contributes to the core symptoms of schizophrenia

including visual disturbances and auditory hallucinations. The pulvinar nucleus exerts an inhibitory effect on the primary auditory cortex, and lifting its suppression may contribute to the development of auditory hallucinations. Furthermore, the disrupted visual and hearing projections from the pulvinar nucleus to the visual and auditory cortices might be a common pathway underlying both visual disturbances and auditory hallucinations in schizophrenia. A better understanding of the etiology of auditory and visual cortex problems may provide insight into the development of tailored preventive and treatment strategies for schizophrenia. In addition, measurements of visual and auditory function in schizophrenia, which are currently not included in the standard assessment of patients in clinical practice, are highly recommended, as schizophrenia is likely reflected through cortical auditory and visual dysfunction.

Visual and auditory processing deficits are excellent targets for the development of future schizophrenia therapies. Distinct underlying mechanisms call for tailored therapeutics to treat individuals with the disorder in the future.

Acknowledgements

This work was supported by grants from the National Natural Science Foundation of China (81871052 to C.Z.), the Key Projects of the Natural Science Foundation of Tianjin, China (17JCZDJC35700 to C.Z.), the Projects of the Natural Science Foundation of Tianjin, China (15JCYBJC50800 to H.T.), the grant of Tianjin 131 Talents plan and the Tianjin Health Bureau Foundation (2014KR02 to C.Z.).

Disclosure of conflict of interest

None.

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