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Resting-State EEG and MEG Correlates of Auditory Hallucinations in Adults With Schizophrenia: A Systematic Review

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Abstract

Auditory hallucinations (AH) are reported by 60–75% of people diagnosed with schizophrenia. They have been linked to a range of cortical structural and functional changes. We systematically reviewed electroencephalogram (EEG) and magnetoencephalography (MEG) resting-state studies of adults with schizophrenia experiencing auditory hallucinations (verbal and/or nonverbal). After searching for relevant studies using the PubMed and Web of Science databases, we applied the PRISMA method to exclude duplicates and studies not matching our inclusion criteria. The selection process yielded 16 studies (8 EEG, 5 MEG, 2 qEEG-LORETA, and 1 EEG-fMRI). Results suggest that both EEG frequency changes and altered intra- and interhemispheric coherence play a role in the generation or perception of AH. Also, while overactivity of the auditory cortex and disruption of normal activity in speech-related areas have been proposed, MEG research suggests that distinct symptoms in schizophrenia may be related to different types of brain alterations and also that different cortical regions may be involved in different types of AH. More research in younger populations is needed and follow up studies should evaluate the effects of targeted interventions during the occurrence of hallucinatory episodes.

Keywords: auditory hallucinations; EEG; MEG; resting-state schizophrenia

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Introduction

The clinical profile of schizophrenia includes positive (e.g., hallucinations, delusions), negative (e.g., apathy, thought, and speech impairment), cognitive (e.g., attention and memory deficits), affective (e.g., depression) and psychomotor (e.g., catatonia) symptoms. Although auditory hallucinations (AH; the perception of sounds in the absence of auditory stimuli) also occur in both other diagnoses and individuals with no psychiatric history (McCarthy-Jones, 2012, 2017), they are reported by 60–75% of people diagnosed with schizophrenia (Lecrubier et al., 2007; Shinn et al., 2012).

Accumulating evidence indicates that distinct symptoms of schizophrenia are associated with both structural and functional neural changes, as detected by a variety of noninvasive imaging techniques (Hu et al., 2017). These studies challenge the idea of a single specific underlying biological mechanism involved in the manifestation of psychotic symptoms, while generally supporting the hypothesis of schizophrenia and related psychotic diagnoses as disconnection syndromes (Andreasen, 1997; Friston, 1999; McGuire & Frith, 1996). According to this hypothesis, the symptomatology of people diagnosed with schizophrenia is strongly associated with altered structural and/or functional communication between distant groups of brain regions that synergistically work as networks. As connectivity between the brain networks regulating resting state have been widely found to be altered in people diagnosed with

schizophrenia (Li et al., 2015; Zhou et al., 2015), the investigation of functional changes associated with spontaneously generated symptoms may shed light on their aetiology. Indeed, people diagnosed with schizophrenia who experience AH have several alterations to the resting state of their brain (Alderson-Day et al., 2016).

Methods such as electroencephalography (EEG) and magnetoencephalography (MEG) have been employed in attempts to identify the physiological processes closely associated with the experience of AH. Relative to other neuroimaging techniques, both EEG and MEG offer excellent temporal resolution (on a millisecond timescale) and allow for tracking brain activity through the scalp, with EEG measuring the electrical signals produced by groups of neurons, and MEG the associated magnetic field. MEG offers greater spatial resolution than EEG as magnetic fields are not significantly distorted, while the propagation of electrical signals related to neuronal activity is affected by the skull (Cohen & Cuffin, 1983). On the other hand, EEG is more accessible to a large number of investigators given its relatively lower cost and can easily be combined with functional magnetic resonance imaging (fMRI), in the attempt of revealing the binding mechanisms between different resting-state networks (Jann et al., 2010; Laufs et al., 2006; Mantini et al., 2007).

EEG studies have produced a significant amount of data and uncovered a range of electrophysiological changes associated with AH (Ford et al., 2012), which might reflect or provide information on the spatial characteristics of the neuronal networks involved (Uhlhaas et al., 2008), in line with the functional dysconnectivity hypothesis of schizophrenia (Andreasen et al., 1998; Friston, 1999).

In an attempt to identify more specific EEG patterns reflecting the perception of AH and a more consistent relationship between hallucinatory episodes and brain topography, a number of studies have also investigated resting-state EEG microstates (Kindler et al., 2011), subsecond time epochs with quasi-stable field topography that are thought to reflect transiently stable distributed neural networks. Microstates are separated by rapid changes of scalp field topography and, since different scalp fields are likely to reflect the unique activation of a neural network, it has been proposed that different microstates correspond to different brain functions (Lehmann et al., 2010). In this context, a specific class of microstates with a fronto-central distribution has been found to be consistently shorter in patients with schizophrenia when compared to healthy controls (Kikuchi et al., 2007; Koenig et al., 1999).

Finally, a significant contribution to the exploration of the underlying neural generators of abnormal EEG activity in schizophrenia is offered by low-resolution electromagnetic tomography (LORETA) EEG studies. In drug-naive symptomatic people diagnosed with schizophrenia, LORETA studies have revealed increased delta activity in the fronto-temporal region and decreased theta/alpha activity in the fronto-temporo-limbic area when compared with normal controls (Mientus et al., 2002; Pascual-Marqui et al., 1999). Beta frequency bands, however, have shown inconsistent differences in schizophrenia. For example, while Pascual-Marqui et al. (1999) reported increased beta activity mainly in right temporal areas, Mientus et al. (2002) found a trend for decreased beta activity.

In this context, the aim of the present systematic review was to discuss the main findings in resting-state EEG (including EEG-fMRI studies) and MEG research in people diagnosed with schizophrenia reporting AH and/or auditory verbal hallucinations (AVH), in an effort to offer an overview of the main frequency-specific changes that have been shown to be closely linked to such experiences. In particular, we sought to determine (a) whether the generation or perception of AH is linked to a specific pattern of altered frequency band oscillations; and (b) whether different types of AH (i.e., acousmata vs. AVH) reflect distinct EEG change patterns and/or originate in separate brain regions. The results will be discussed within the framework of the dysconnectivity hypothesis of schizophrenia, attempting to integrate different methods of investigation and data analysis.

Methods

A search was carried out in the PubMed and Web of Science online databases without date restrictions for English-language articles containing the terms: “(eeg OR qeeg OR electroencephalogram OR eegfmri OR eeg-fmri OR meg OR magnetoencephalogram OR megfmri OR meg-fmri) AND (auditory verbal hallucinat* OR auditory hallucinat* OR verbal hallucinat* OR (hear* voice*) OR (hallucinat* AND voice*)) AND (schizophr* OR psychosis)”.

We looked for EEG, MEG, and EEG/MEG-fMRI studies in adults diagnosed with schizophrenia-spectrum disorders, who experienced AH, during

resting state. We excluded reviews, book chapters, meeting and conference abstracts, editorial material, and publications in languages other than English. We also excluded studies where AH were not specifically mentioned.

The search was originally concluded on January 20, 2021, and then updated on September 20, 2022. The updated search returned 236 results from PubMed and 252 from Web of Science. We then applied the PRISMA method to exclude duplicates and studies not matching our inclusion criteria (Figure 1). The selection process yielded 16 articles. Of these, 12 studies employed EEG only, 8 MEG only, 2 qEEG-LORETA, and 1 EEG-fMRI. A descriptive summary of the papers included is presented in Table 1.

Figure 1. *PRISMA Flow Diagram for Systematic Review.*

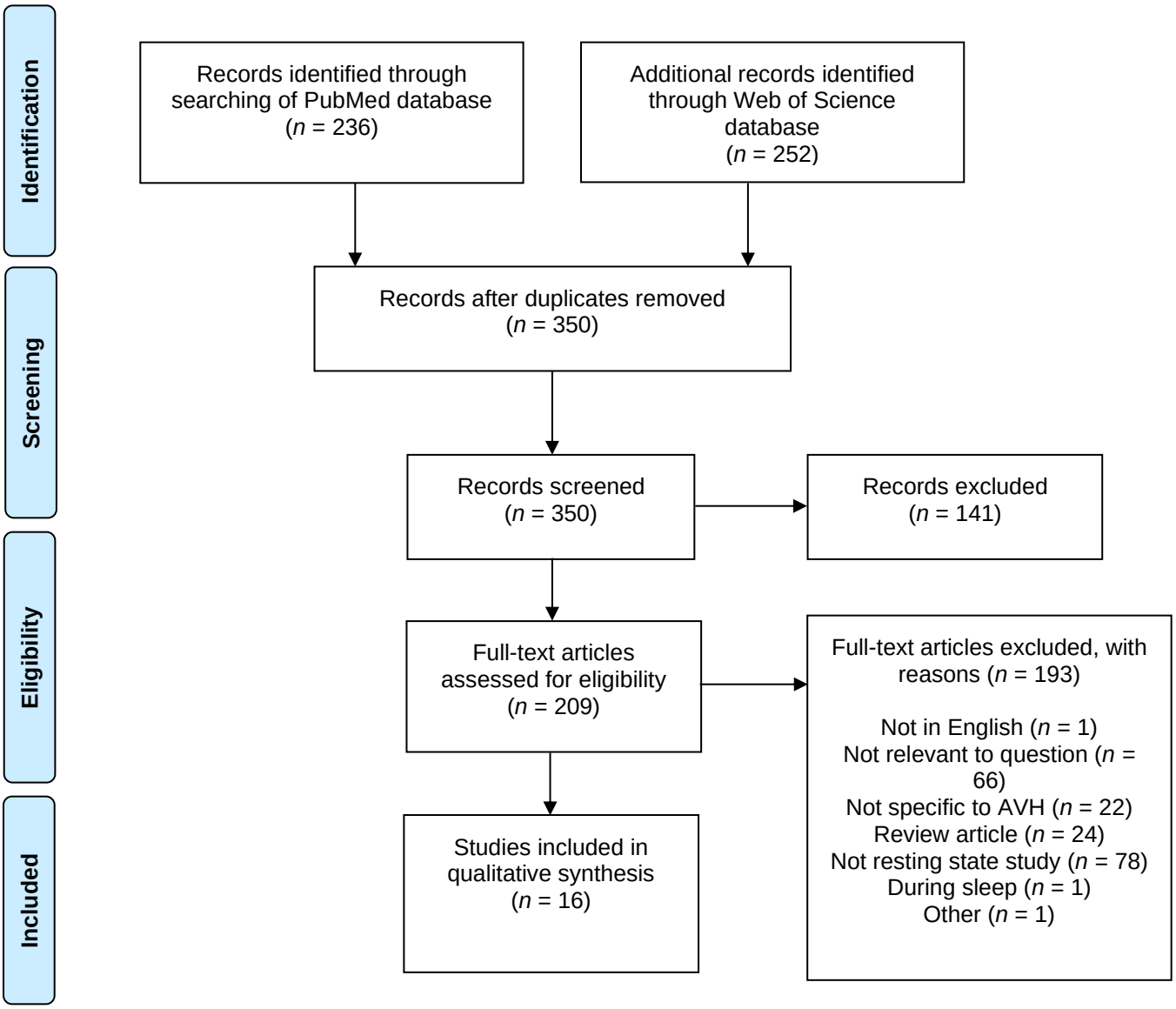

Table 1
Summary of the Reviewed Studies

Study	Population	Methods	Design	Results
EEG Studies				
Arora et al., 2021	SZ with AH (*n* = 12; 8 males, mean age = 44.25 years, *SE* = 3.16 years). SZ without AH (*n* = 12; 10 males, mean age = 45.67 years, *SE* = 3.16 years). HC (*n* = 12; 6 males, mean age = 39.75 years, *SE* = 3.16 years).	Spontaneous EEG.	Between-groups cross-sectional design.	Alpha activity was lower in patients with SZ and AH compared to patients with SZ and no AH in frontal areas bilaterally. Beta activity was lower in patients with SZ and AH when compared to patients with SZ and no AH. AH severity negatively correlated with alpha activity at F3, F4, P3, and P4. AH severity also negatively correlated with beta activity in the parietal area bilaterally.
Kindler et al., 2011	SZ with AH (*n* = 9; 6 males; mean age = 35.2 ± 11.7 years).	EEG (resting state/patients were asked to listen and attend to the voices and indicate the beginnings and endings by a button press).	Within-subjects design; experience of AH signaled by button press.	AVH were associated with shortening of Class D microstate.
Ahn et al., 2019	SZ with AH (*n* = 8).	qEEG measured during resting state, before/after transcranial alternating current stimulation.	tACS delivered and ΔEEG related to ΔAH.	Decrease of AH was associated with an increase in alpha-oscillation power in the left temporoparietal/frontal region.
Sritharan et al., 2005	SZ with AH (*n* = 7; 3 males, mean age = 31.9 years, *SD* = 4.9 years).	Spontaneous EEG.	Within-subjects design; experience of AH signaled by button press.	Increase in coherence between the left and right superior temporal cortices during AH vs. non-AH states.
Lee et al., 2008	SZ with AH (*n* = 25; 11 males, mean age = 39.2 years, *SD* = 6.8 years). SZ without AH (*n* = 23; 10 males, mean age = 38.5 years, *SD* = 7.1 years).	Spontaneous EEG.	Between-groups cross-sectional design.	SZ with AH patients had increased gamma frequency D2 in Fp2 (right prefrontal cortex) and decreased beta frequency D2 in the left parietal region.

Table 1
Summary of the Reviewed Studies

Study	Population	Methods	Design	Results
Angelopoulos et al., 2011	SZ with AH (*n* = 8; 4 males, mean age = 36 years, *SD* = 7 years). SZ without AH (*n* = 7; 3 males, mean age = 30 years, *SD* = 9 years). HC (*n* = 16; 8 males, mean age = 31 years, *SD* = 6 years).	Spontaneous EEG.	Mixed design. Between group cross-sectional analysis and within-subject design; experience of AH signaled by button press.	Increased phase-coupling of alpha band in patients with SZ and AH, distributed intra- and interhemispherically in the anterior brain areas. Increase of alpha-band synchrony in SZ patients with AH, compared to both SZ patients without AH and HC at the T7–T8 electrode pair interhemispherically.
Van Lutterveld, Koops, et al., 2012	SZ with AH (*n* = 24; 17 males, mean age = 41 years, *SD* = 14 years).	Spontaneous EEG.	rTMS delivered and ΔEEG related to ΔAH.	No correlations between changes in whole-head alpha-band or theta-band power and changes in AH.
Zheng et al., 2017	SZ with AH - medicated (*n* = 20; 12 males, mean age = 21.75 years, *SD* = 4.7 years). SZ with AH – unmedicated (*n* = 12; mean age = 21.21 years, *SD* = 6.72 years). HC (*n* = 22; 7 males, mean age = 22.91 years, *SD* = 6.91 years).	EEG (eyes focusing on a white cross on a black background on a computer screen).	Between groups cross-sectional design.	When the two SZ groups were compared, greater activities were found in RMFG (alpha and beta band) and LSTG (alpha and beta band) in the nonmedicated group when compared with the medicated group.
MEG Studies				
Ishii et al., 2000	SZ with AH (*n* = 1; one male, age 28 years).	Spontaneous MEG recordings.	Within-subjects design; experience of AH signaled by button press.	Increase in theta-band activity during experiences of AH. When AH reduced at follow-up, significant theta-band activity in the left superior temporal cortex was no longer detected.
Ropohl et al., 2004	SZ with AH (*n* = 1; male, age 33 years). HC (*n* = 13; all male, mean age = 31.3 ± 4.7 years).	Spontaneous MEG recordings.	Between groups cross-sectional design.	Increase of fast activity (12.5–30 Hz) in the left superior temporal cortex during AH in patients, relative to HC.

Table 1
Summary of the Reviewed Studies

Study	Population	Methods	Design	Results
Reulbach et al., 2007	SZ (*n* = 16; 9 males, mean age = 33 years, *SD* = 2.8 years) of whom 8 had AH. HC (*n* = 8; 4 males, mean age = 35 years, *SD* = 8.2 years).	Spontaneous MEG recordings.	Between groups cross-sectional design; experience of AH signaled by button press.	Greater number of dipoles in the fast frequency range (12.5–30 Hz) in SZ patients with AH when compared with SZ patients without AH. AH per se were associated with a concentration in dipoles on the left superior temporal gyrus and parts of the dorsolateral prefrontal cortex. AH involving commands were associated with a concentration of dipoles in parts of the left dorsolateral prefrontal cortex.
Sperling et al., 1996	SZ with AH (*n* = 3; 2 males). HC (*n* = 3; 2 males).	Spontaneous MEG recordings.	Between groups cross-sectional design.	Slow activity dipoles (2–6 Hz) were not increased in patients with SZ and AH over both hemispheres compared with HC. Patients with SZ and AH had greater fast frequency (12.5–30 Hz) activity in the temporal region of the left and right hemispheres.
Van Lutterveld, Hillebrand, et al., 2012	SZ with AH (*n* = 12; 8 males, age range 26–62 years).	Spontaneous MEG recordings.	Within-subjects design; experience of AH signaled by button press.	Experience of AH was associated with a decrease in beta-band power in the left temporal cortex and a decrease in alpha-band power in the right inferior frontal gyrus. The onset of AH was associated with a reduced theta-band power in the right hippocampus.
qEEG-LORETA Studies				
Lee et al., 2006	SZ with AH (*n* = 25; 11 males, mean age = 39.2 years, *SD* = 6.8 years). SZ without AH (*n* = 23; 10 males, mean age = 38.5 years, *SD* = 7.1 years).	qEEG/LORETA (eyes closed/eyes open).	Between groups cross-sectional design.	Gamma (30–50 Hz) and beta (2 and 3) frequencies were correlated in SZ with AH, but not in SZ without AH. Patients with SZ and AH had significant increase of beta 1 (13–18 Hz) power in the left inferior parietal lobule and in beta 2 power (19–21 Hz) in the left medial frontal gyrus when compared with patients with SZ and no AH.

Table 1
Summary of the Reviewed Studies

Study	Population	Methods	Design	Results
Horacek et al., 2007	SZ with AH (*n* = 12; 7 males, mean age = 34.4 years, *SD* = 9.1 years).	LORETA (eyes closed).	Within-subjects design; rTMS delivered and ΔEEG related to ΔAH.	Symptom improvement was associated with bilateral increase of current density in the delta power (anterior cingulate cortex), beta 1 and beta 3 power (temporal lobe) as well as beta 2 power (middle temporal and inferior parietal lobule).
EEG-fMRI Studies				
Hare et al., 2017	SZ with AH but no VH (*n* = 42; 32 males, mean age = 37.8 years, *SD* = 11.9 years). SZ with AH and VH (*n* = 40; 30 males, mean age = 37.2 years, *SD* =11.3 years). SZ with no hallucinations (*n* = 61; 44 males, mean age = 40.2 years, *SD* = 11.8 years). HC (*n* = 155; 110 males, mean age = 37.8 years, *SD* = 11.3 years).	Simultaneous EEG/fMRI.	Between-groups cross-sectional design.	Some evidence that AH were associated with decreased mean amplitude of low-frequency fluctuations (ALFF) across the posterior brain regions (cuneus and lingual gyrus). Also, ALFF was increased in the right inferior frontal gyrus and in part of the inferior temporal lobe.

Note. AH = auditory hallucinations; D2 = correlation dimension; EEG = electroencephalogram; fMRI = functional magnetic resonance imaging; HC = healthy controls; LORETA = low-resolution brain electromagnetic tomography; MEG = magnetoeletroencelography; qEEG = quantitative electroencephalogram; rTMS = repetitive transcranial magnetic stimulation; SZ = schizophrenia; tACS = transcranial alternating current stimulation; VH = visual hallucinations.

Results

The studies reviewed included a range of designs. Three studies compared people with schizophrenia with AH to healthy controls (Sperling et al., 1996; Reulbach et al., 2007; Ropohl et al., 2004). It is not possible to determine from such designs what activity is specific to AH and what is either related to schizophrenia per se or the use of medications.

Comparing People With Schizophrenia With and Without AH

Two studies compared people with schizophrenia with and without AH (Lee et al., 2008; Lee et al., 2006). Assuming that these patient groups do not differ on any factors except the presence of AH (a questionable assumption), such designs have the potential to identify neural activity associated with trait AH specifically. The most recent of these studies found that AH were associated with increased (more chaotic) gamma frequency correlation dimension in the right prefrontal cortex (Fp2) and decreased (less chaotic) beta frequency correlation dimension in the left parietal cortex (P3) region (Lee et al., 2008). The authors speculate that the more chaotic integration of gamma frequency information in the prefrontal cortex could represent difficulties differentiating internally and externally

generated sensory inputs, hence representing a misattribution process. In an earlier study, Lee et al. (2006) found that trait AH were associated with increased beta 1 and beta 2 frequency amplitude in the left inferior parietal lobule and the left medial frontal gyrus.

Comparing Healthy Controls and Schizophrenia Patients With and Without AH

Four studies compared neural activity between three groups: people with schizophrenia with and without AH, as well as healthy controls (Angelopoulos et al., 2011; Arora et al., 2021; Hare et al., 2017; Zheng et al., 2017). The study by Arora et al. (2021) found decreased beta-band activity to be associated with trait AH. This finding mirrored that of Lee et al. (2006) noted above. Arora et al. (2021) also examined correlations between AH severity and EEG activity, reporting that higher AH severity was associated with lower parietal beta activity. Arora et al. (2021) also reported that alpha activity was lower in people with schizophrenia with AH compared to those without AH in frontal areas bilaterally. Again, severity of AH was negatively correlated with alpha activity at frontal and parietal sites. Although Zheng et al. (2017) reported findings that could be interpreted as an increase in alpha-band activity being associated with AH, because these findings involved comparing a medicated and unmedicated group of patients such a conclusion may be unreliable. Angelopoulos et al. (2011) reported increased alpha-band phase-coupling to be associated with AH, both intra- and interhemispherically in the anterior brain areas, as well as an increase in alpha-band synchrony at the T7–T8 electrode pair interhemispherically.

Finally, in a multisite EEG-fMRI study that was able to differentiate between neural activity associated with auditory and visual hallucinations in schizophrenia, Hare et al. (2017) found some evidence that AH were associated with decreased mean amplitude of low-frequency fluctuations (ALFF) across posterior brain regions (cuneus and lingual regions) as well as increased ALFF in the right inferior frontal gyrus and part of the inferior temporal lobe.

State Experiences of AH

Five studies examined neural activity associated with state experiences of AH (Ishii et al., 2000; Kindler et al., 2011; Reulbach et al., 2007; Sritharan et al., 2005; Van Lutterveld, Hillebrand et al., 2012). These studies asked patients to press a button to signal experiences of AH and compared the activity during the presence of AH to periods when no AH were present.

Kindler et al. (2011) found AH to be associated with a shorter duration of Class D microstates in a fronto-central location. In terms of coherence, Sritharan et al. (2005) found increased coherence in the alpha frequency band between the left and right superior temporal cortices during AH. In terms of power, Van Lutterveld, Hillebrand, et al. (2012) found that experiencing AH was associated with (a) a decrease in alpha-band power in the right inferior frontal gyrus, (b) a decrease in beta-band power in the left temporal cortex, and (c) reduced theta-band power in the right hippocampus. In contrast, to this finding of reduced theta band, Ishii et al. (2000) found that experiences of AH were associated with increased theta-band activity during experiences of AH. When AH reduced at follow-up, significant theta-band activity in left superior temporal cortex was no longer detected, suggesting a causal role for increased theta-band activity in AH. Finally, Reulbach et al. (2007) reported that experiences of AH were associated with elevations of dipoles and dipole density maxima in the beta frequency range.

EEG Changes Related to AH Changes After Neurostimulation

Three studies examined how changes in EEG resulting from neurostimulation treatments correlated with changes in AH (Ahn et al., 2019; Horacek et al., 2007; Van Lutterveld, Koops, et al., 2012). The earliest of these studies found that improvements in AH resulting from rTMS were associated with bilateral increase of current density in the delta band (anterior cingulate cortex), beta 1 and beta 3 bands (temporal lobe) as well as the beta 2 band (middle temporal and in the inferior parietal lobule; Horacek et al., 2007). In contrast, Van Lutterveld, Koops, et al. (2012) found no correlations between changes in whole-brain alpha-band or theta-band power and changes in AH after rTMS. Finally, Ahn et al. (2019) found that a decrease in AH after transcranial alternating current stimulation (tACS) was associated with an increase in alpha power.

Discussion

Due to the use of a wide range of designs and forms of EEG analysis (e.g., power, coherence, microstates), there are very few reliable well-replicated findings in this area. The first takeaway from this review is hence the need for the creation of well-powered replicated studies that create a clearer picture of the nature of EEG changes associated with AH. Nevertheless, the disparate range of

findings do appear to often point in the same direction, particularly when we consider how the findings from intervention studies align with the findings from cross-sectional studies.

In terms of alpha-band power, the work of Arora et al. (2021) has provided evidence of lower alpha activity in frontal areas bilaterally being associated with both trait and state AH. Similarly, Van Lutterveld, Hillebrand, et al. (2012) found state experiences of AH were associated with a decrease in alpha-band power (specifically in the right inferior frontal gyrus). This suggests that improvement in AH should be associated with increased alpha power, which is what Ahn et al. (2019) found using tACS. However, it was notable that other studies had not found such changes in alpha-band power to be associated with neurostimulation-induced AH improvements (Horacek et al., 2007; Van Lutterveld, Koops, et al., 2012). The evidence of increased phase-coupling of alpha-band activity distributed intra- and interhemispherically, along with the finding indicating an increase in the frequency of AVH during an eyes-closed versus eyes-open task (Angelopoulos et al., 2011) also supports the assumption of an alpha-oscillation role in AVH generation.

Such findings are consistent with antipsychotic-related improvements in people diagnosed with schizophrenia being associated with enhancement of alpha oscillations (Jin et al., 1995). The meaning of such alpha-band changes is not clear. Although alpha oscillations have been shown to reflect idling (Pfurtscheller et al., 1996) or the active inhibition of task-unspecific brain circuits (Busch & Herrmann, 2003), there is also evidence for a positive correlation of the alpha amplitude with short-term memory and working-memory load (Jensen et al., 2002), as well as task difficulty (Sauseng et al., 2005). Notably though, Ahn et al. (2019) suggest an important "organizing role of alpha oscillations, which, when enhanced by stimulation, enable improvement of other, impaired network dynamics in other frequency bands" (p. 134).

In terms of beta activity, two studies found decreased beta-band activity to be associated with trait AH (Arora et al., 2021; Lee et al., 2006). Decreased beta-band activity was also associated with more severe AVH (Arora et al., 2021) and experience of AH (Van Lutterveld, Hillebrand, et al., 2012). Lee et al. (2008) also point to less chaotic beta frequency correlation dimension in the left parietal cortex region as associated with AH. Consistent with these findings, improvements in AH resulting from rTMS were associated with increases in beta-band activity in a range of temporal and parietal sites (Horacek et al., 2007). Another interesting finding was that the beta frequency power D2 in the left parietal brain region was lower in patients with AH when compared with nonhallucinating patients. It is known that beta oscillations follow periods of synchronous gamma activity (Kopell et al., 2000; Traub et al., 1999). Similarly, transitions from gamma to beta oscillations can be detected following the presentation of novel auditory stimuli (Haenschel et al., 2000), suggesting that beta oscillations might reflect the encoding of auditory stimuli.

In terms of the meaning or role of these beta-band alterations, beta oscillations are argued to be involved in signalling the novelty or salience of a stimulus (see Van Lutterveld, Hillebrand, et al., 2012). Alternatively, the link between decreased beta-band power and AH has been proposed to result from impaired corollary discharge signalling between frontal and temporal regions (see Van Lutterveld, Hillebrand, et al., 2012). There is hence the need to better establish the precise role of such alterations in beta-frequency power in AH.

Although, Van Lutterveld, Hillebrand, et al. (2012) found experiencing AH was associated with reduced theta-band power (in the right hippocampus), Ishii et al. (2000) found that experiences of AH were associated with increased theta-band activity. Other studies have failed to find alterations of theta power to be associated with AH, finding such changes to relate to schizophrenia per se instead (Arora et al., 2021). There is hence a need for a clearer picture of the role (if any) of theta-band changes in relation to AH.

There is also evidence for a direct association between phase synchronization or EEG signal coherence in frontal, temporal, central with parietal brain regions, and positive/negative symptoms of schizophrenia (Bob et al., 2008). Of note is the research suggesting that functional changes in left fronto-temporo-parietal resting-state language processing and speech monitoring networks, could be associated with the generation of AVHs (Wolf et al., 2011). Other resting-state EEG research, however, has revealed both decreases and increases in coherence associated with schizophrenia (Jalili et al., 2007) and the data analysis method used (i.e., multivariate synchronization analysis) should be further explored.

A potentially important finding is that the gamma frequency D2 in the right prefrontal cortex is higher in patients with AH. Gamma oscillations (30–50 Hz) are thought to play an important role in high-level cognitive processing (Tallon-Baudry & Bertrand, 1999). It has also been proposed that gamma activity is related to or reflects coherent object representations, perhaps mediating internally driven representations and memory storage (Allen et al., 2005). Previous research also indicates that the prefrontal cortex is implicated in the processes that allow a healthy person to distinguish between endogenous sensations from those induced by external influences (Frith, 1996). Thus, it is possible that a chaotic integration of gamma frequency information in the prefrontal cortex in patients with AH reflects the reduced ability to discriminate internal from external sensory inputs. In this context, Kindler et al. (2011) suggest that the reduction of specific microstate duration in schizophrenia may impair the correction of an erroneous misattribution of self-generated inner speech to external sources. However, it is also possible that increased D2 in the right prefrontal region of the brain is only a consequence of emotion processing during the perception of AH (George et al., 1996), which typically bear a derogatory and hostile tone.

Clearly, EEG studies pointing at alterations in the temporal lobe are consistent with the wider research literature on the localization of neural changes associated with AH (Jardri et al., 2011). Similarly, treatment studies aimed at reducing neural activity in the left superior temporal gyrus, through neurofeedback training (Orlov et al., 2018) and neurostimulation (Giesel et al., 2012) have reported reductions in AVH, although larger studies are needed to establish if genuine therapeutic effects are present (Guttesen et al., 2021). Understanding the specific type of activity associated with the presence of AH in this region could inform the future design of such studies.

Some of the studies reported here suggest the importance of distinguishing between AH and specifically AVH. In their MEG study on patients with both acousmata (unspecific noises) and AVH, Reulbach et al. (2007) identified the left superior temporal lobe and the left dorsolateral prefrontal cortex as the neural correlates of increased activity in the fast frequency range. However, within this population, patients hearing acousmata showed only a concentration of dipoles in the left superior temporal gyrus, while patients with AVH showed combined and confluent localization of dipole maxima in the left superior temporal gyrus and parts of the left dorsolateral prefrontal cortex. These results suggest a differential role of separate regions in the generation or perception of different types of AH (cf. Jones, 2010) and might also add to previous findings relating subsyndromes of symptoms to distinct cerebral flow patterns (Liddle et al., 1992) and metabolic changes (Cleghorn et al., 1992). They might also support and extend previous multimodal research, including MEG and MRI studies, suggesting the differential roles of the dorsolateral prefrontal cortex and superior temporal gyrus in AH (e.g., Kawaguchi et al., 2005; Shergill et al., 2000)

All the studies included in the present review were carried out in adult participants. Longitudinal studies in younger populations indicate that differential structural or functional and neurochemical changes take place at different stages of the illness, from the prodromal phase to the phase when the first psychotic episodes start to manifest (Keefe, 2014; Pantelis et al., 2005; Wood et al., 2011). This suggests that the profile for the abnormalities discussed in the present review could vary considerably during the illness progression and that the evidence gathered from investigations in adults should stimulate similar investigations with younger populations.

Also, most of the studies reviewed here included medicated patients, which raises the question of whether and to what extent the changes in the parameters reported were genuine consequences of the illness, drug effects, or a mixture of both (Koch et al., 2016). The differences found by Zheng et al. (2017) between nonmedicated and medicated patients suggest that more EEG research should investigate AH-related changes in first episode or drug naïve participants, with follow-up studies evaluating the effects of therapeutic interventions on selected measures. In this context, studies involving the administration of antipsychotic drugs and also other nonpharmacological interventions (e.g., neurofeedback training) should be considered.

In conclusion, at rest, frequency-specific changes in EEG activity may differentially contribute to the experience of AH in people diagnosed with schizophrenia. Whilst the results of studies in relation to alpha- and beta-band activity appear to line up well, there is still the need for large, well-powered and replicated studies in this area. Such work should take into account the potential to identify separate neural correlates for unspecific noises (acousmata) versus AVH, recruit participants at a range of stages of illness, and make use of both longitudinal and intervention studies.

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Declaration of Interest

All authors declare no conflicts of interest.

Author Contribution

Dr. Amico and Dr. McCarthy-Jones undertook the systematic review process. All authors contributed to the writing of the manuscript.

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