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Data Availability
The data associated with this publication are available at:
https://github.com/JeremyManwaring/Schziophrenia_Speech_Analysis

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Functional Analysis of Auditory Verbal Hallucinations and Speech-Network Activation in Schizophrenia

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Abstract

The study by Soler-Vidal et al. (2022) examined the relationship between auditory verbal hallucinations (AVH) and reduced auditory cortex activation in schizophrenia. We extended an analysis focusing on auditory-language activation, regional effects, connectivity, and clinical correlations, replicating and extending their results. Patients with and without auditory verbal hallucinations were compared using functional magnetic resonance imaging (fMRI) while participants listened to words, sentences, and reversed speech (n=71). Auditory perception activated a shared language and auditory network including the bilateral superior temporal cortex, inferior/lateral prefrontal cortex, inferior parietal cortex, and supplementary motor area. Reduced activation in the left primary auditory cortex was observed, a lower-order region involved in processing basic sounds. Our additional exploratory findings suggest that auditory verbal hallucinations may be associated with altered activation in higher-order temporal-language regions, particularly the left middle temporal gyrus and superior temporal sulcus, and hallucination severity correlating with functional activation in the right posterior superior temporal gyrus. Overall, this indicates that auditory verbal hallucinations do not just include early sound processing, but include both early auditory processing and speech processing in higher-order regions, with abnormalities observed in distinct, yet interconnected cortical regions.

Keywords: auditory verbal hallucinations; schizophrenia; fMRI; speech perception; replication; functional neuroimaging

Introduction

Schizophrenia is a psychiatric disorder that affects perception, cognition, and thought organization. Its symptoms are commonly divided into positive symptoms, such as hallucinations and delusions, and negative symptoms, such as reduced emotional expression, diminished motivation, and restricted speech. [2] Auditory verbal hallucinations are a particularly important positive symptom because they involve the perception of voices or speech-like sounds in the absence of an external source. These experiences can distort perceived reality and may interfere with communication, attention, and daily functioning [4]. Broader theories of psychosis suggest that hallucinations may emerge from disrupted connectivity, impaired temporal and agency processing, and altered interactions between bottom-up sensory input and top-down expectations [2, 3].

Human perception and language comprehension are often described through predictive coding. In this framework, the brain continuously anticipates sensory input and updates those predictions when actual sensory signals differ from expectations. Perception therefore depends on the interaction between bottom-up sensory evidence and top-down predictions. In the auditory system, this balance supports rapid sound recognition and efficient speech comprehension. Predictive-coding accounts of psychosis include competing stronger-prior and imprecise-prior explanations and should not be treated as a single settled mechanism [3].

Speech processing depends on a distributed network of auditory, temporal, frontal, parietal, and attentional systems. The primary auditory cortex supports early sound processing, while surrounding secondary association areas contribute to phonetic and speech analysis. Broca's and Wernicke's areas are traditionally associated with language production and comprehension, but broader temporal, parietal, hippocampal, and executive-control networks are also needed for attention, learning, and meaning [5].

In schizophrenia, structural and functional changes in the central nervous system may disrupt this system. Prior work has reported gray-matter differences in the superior temporal gyrus and primary auditory regions and altered communication among auditory and frontal systems [6, 4]. These changes may contribute to hallucinations by making internally generated speech feel as though it comes from an external source [2]. One explanation is that hallucinations arise from disrupted communication between brain regions that initiate actions and regions that process sensory feedback, producing deficits in temporal coordination and self-monitoring [4].

These symptoms may also reflect an imbalance between bottom-up and top-down processing. When top-down expectations, beliefs, or biases are weighted too strongly, ambiguous or internally generated sensory experiences may be misinterpreted as real external events [7]. Bayesian models of brain function describe perception as a process in which prior beliefs are combined with incoming sensory information to reduce prediction errors. Alterations in this process may increase sensory mismatch, prediction error, and disorganized processing [3]. Together, these factors may weaken the brain networks responsible for generating accurate predictions about the world, creating conditions under which auditory verbal hallucinations can occur.

Soler-Vidal et al. (2022) used fMRI to examine auditory cortex activation in schizophrenia patients with and without AVH. The present paper analyzes that dataset in order to evaluate the reproducibility of the original findings and to examine how perceptual and cognitive processes may interact during speech perception.

Methods

Participants

The original study recruited patients with schizophrenia and healthy controls. After exclusions and group matching, the final sample included patients with auditory verbal hallucinations (AVH+; $n = 23$), patients without auditory verbal hallucinations (AVH−; $n = 23$), and healthy controls ($n = 25$). Exclusion criteria were the following, younger than 18 or older than 65, history of brain trauma or neurological disease, alcohol or substance abuse/dependence within the previous 12 months, estimated IQ below 70, excessive head motion, failure to complete the scan or task, and failure to meet group-specific symptom criteria [1].

Patients were diagnosed using the Structured Clinical Interview for DSM Disorders (SCID). The AVH+ group included patients who experienced frequent auditory verbal hallucinations, while the AVH− group included patients who had been free of auditory hallucinations for at least six months. Hallucination severity was assessed using the auditory hallucinations subscale of the Psychotic Symptom Rating Scales (PSYRATS-H) [8]. To qualify for the AVH+ group, participants were required to experience auditory verbal hallucinations at least once per day.

Premorbid IQ was estimated using the Word Accentuation Test (Test de Acentuación de Palabras; TAP), and current IQ was assessed using four subtests from the Wechsler Adult Intelligence Scale, Third Edition (WAIS-III). Healthy controls also completed the SCID and IQ assessments so that participants with psychiatric symptoms or other exclusionary characteristics could be removed from the control group.

This study was a secondary analysis of deidentified public data and involved no new participant recruitment. The original protocol was approved by the Hospital Sisters Research Ethics Committee, and all participants provided written informed consent [1].

Task Design

Participants completed speech perception tasks while undergoing brain imaging. Imaging was performed with a 3T Philips Ingenia MRI scanner. Functional brain activity was measured using $T2^*$-weighted echo-planar imaging (EPI), and high-resolution anatomical images were acquired using fast field echo (FFE) scanning.

The speech perception task included three conditions: spoken words, spoken sentences, and unintelligible reversed speech. Each condition included six 26-second blocks, separated by 2-second intervals. After every three active blocks, participants heard 26 seconds of white noise. In the word condition, participants heard unrelated words. In the sentence and reversed-speech conditions, participants heard eight sentences or reversed-speech stimuli, respectively. Stimuli were presented through MRI-compatible headphones, and visual materials were presented through MRI-compatible goggles. After scanning, participants completed a questionnaire assessing attention to the task and hallucination frequency during scanning [1].

Original Image Preprocessing and Analysis

Preprocessing in the original study was conducted using the FEAT module in FSL (FMRIB Software Library) [9]. Steps included removing the first 10 seconds of each run, motion correction, coregistration, normalization, and spatial filtering with a Gaussian kernel. Participants were excluded if absolute movement exceeded 3.0 mm or if average movement exceeded 0.3 mm.

The original statistical analysis used first-level general linear models (GLMs) to compare words, sentences, and reversed speech with the white-noise baseline. At the group level, mixed-effects GLM analyses generated mean activation maps and group comparisons. Age, sex, and premorbid IQ were included as covariates. Cluster-level statistical significance was assessed using Gaussian random field methods with a cluster-forming threshold of $z > 2.3$ and corrected cluster significance of $p < .05$ [1].

Table 1: Demographic and clinical characteristics by group.

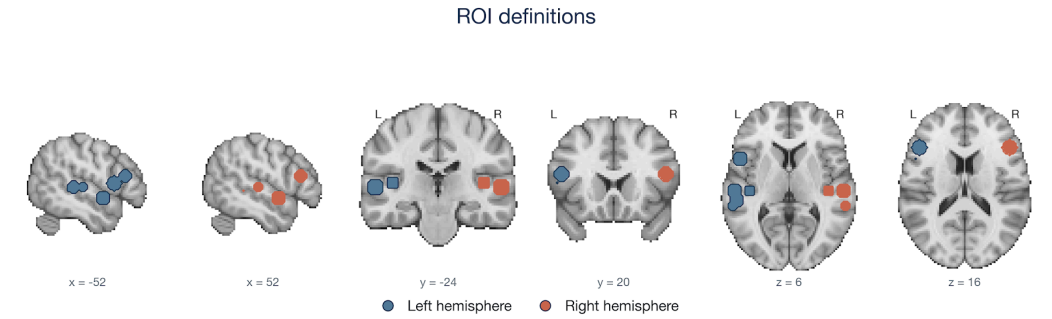
Measure	Healthy controls	AVH−	AVH+
n	25	23	23
Age (years)	39.8 ± 14.1	45.0 ± 7.4	40.1 ± 13.0
Sex (M/F)	18/7	16/7	20/3
Premorbid IQ (TAP)	101.2 ± 9.9	101.3 ± 8.9	98.4 ± 9.6
PSYRATS-H	N/A	0 [0–0]	24.9 ± 7.3 [11–39]
Framewise displacement (mm)	0.16 ± 0.07	0.18 ± 0.09	0.19 ± 0.08
> 20% high-motion	2 (8%)	2 (9%)	0 (0%)

Replication Methods

Anatomical and functional fMRI data were sourced from OpenNeuro dataset ds004302, (v. 1.0.0) including 71 participants: 25 controls, 23 patients without auditory verbal hallucinations (AVH−), and 23 patients with auditory verbal hallucinations (AVH+) [10]. Processed BOLD data replicated the original analyses to assess reproducibility. First-level general linear modeling was performed with Nilearn using fMRIPrep derivatives [11, 12]. Task regressors were convolved with the SPM hemodynamic response function, with AR(1) noise modeling, drift correction, high-pass filtering (0.01 Hz), and 6 mm full-width at half-maximum (FWHM) spatial smoothing. Motion, white-matter, and cerebrospinal-fluid confounds were included.

Seven first-level contrasts were analyzed: words > baseline, sentences > baseline, reversed speech > baseline, words > reversed speech, sentences > reversed speech, speech > reversed speech, and words > sentences. Group-level analyses compared healthy controls, AVH− patients, and AVH+ patients using second-level GLMs and ROI analyses extracted from 12 speech- and language-related ROIs across the left and right hemispheres. Corrected extraction used 8 mm cortical spheres and 6 mm Heschl's-gyrus spheres. Several left temporal spheres overlapped and were therefore not anatomically independent.

Three-group ROI differences were tested with Welch's ANOVA, with Benjamini–Hochberg false-discovery-rate (FDR) correction across 12 ROIs within each contrast. Pairwise comparisons were treated as exploratory and used Games–Howell tests. A post hoc covariate-adjusted sensitivity analysis compared AVH− and AVH+ patients in the L-MTG and L-STS while controlling for age, premorbid IQ, sex, and mean framewise displacement. These ROIs were selected after review of the exploratory results and were not treated as a preregistered confirmatory family.



Left hemisphere

ROI	x	y	z	r (mm)
L STG Posterior	-58	-22	4	8
L STG Anterior	-54	0	-8	8
L MTG	-60	-38	0	8
L IFG Triangularis	-48	24	16	8
L IFG Opercularis	-52	12	8	8
L STS	-56	-32	4	8
L Heschl	-42	-22	8	6

Right hemisphere

ROI	x	y	z	r (mm)
R STG Posterior	58	-22	4	8
R STG Anterior	54	0	-8	8
R MTG	60	-38	0	8
R IFG	48	24	16	8
R Heschl	42	-22	8	6

Figure 1: Speech- and language-related regions of interest using ROI locations

PSYRATS-H severity was related to ROI activation using Pearson correlations and partial correlations controlling for age and IQ. Partial-correlation significance used n-k-2 degrees of freedom, where $k = 2$ covariates, and FDR correction was applied across 12 ROIs within each contrast. Whole-brain subgroup inference used 10,000 two-sided permutations, a voxelwise cluster-forming threshold of $p < .001$, and maximum-cluster-size family-wise error correction at $p < .05$. ROI-to-ROI functional connectivity, hemisphere laterality, and multivariate pattern analysis (MVPA) were treated as exploratory. MVPA used shuffled five-fold cross-validation and a 100-permutation significance test.

Results

Demographic and Clinical Characteristics

The final sample included 71 participants: 25 healthy controls, 23 AVH− patients, and 23 AVH+ patients. The sample was primarily male across all three groups: healthy controls included 18 men and 7 women (72%), the AVH− group included 16 men and 7 women (70%), and the AVH+ group included 20 men and 3 women (87%). Sex distribution did not differ significantly across groups, $\chi^2(2) = 2.26$, $p = .323$.

Age did not differ significantly across groups, Welch's $F(2,41.97) = 2.00$, $p = .148$. AVH− patients were 5.2 years older than controls and 4.9 years older than AVH+ patients, while AVH+ patients and controls were nearly identical in age.

IQ was similar across groups. Healthy controls had a mean IQ of 101.2 ($SD = 9.9$; $n = 24$), AVH− patients had a mean IQ of 101.3 ($SD = 8.9$; $n = 22$), and AVH+ patients had a mean IQ of 98.4 ($SD = 9.6$; $n = 23$). Welch's ANOVA did not show a significant group effect, $F(2,43.99) = 0.68$, $p = .512$. As expected, AVH− patients scored 0 on the PSYRATS-H subscale, while AVH+ patients showed elevated hallucination severity ($M = 24.9$, $SD = 7.3$, range = 11–39). Healthy controls did not complete the PSYRATS-H subscale.

Head motion was low and similar across groups. Mean framewise displacement was 0.16 mm ($SD = 0.07$) for controls, 0.18 mm ($SD = 0.09$) for AVH− patients, and 0.19 mm ($SD = 0.08$) for AVH+ patients. A small number of participants in the healthy-control and AVH− groups had more than 20% high-motion volumes, but the group means remained below common quality-control thresholds. Motion-clean analyses were treated as sensitivity checks.

Whole-Brain Group Differences: Sentences Versus Reversed Speech

Whole-brain comparisons examined differences between AVH− and AVH+ patients. Corrected analyses included age, IQ, and sex as covariates and used 10,000 permutations. No cluster survived maximum-cluster-size family-wise error correction for sentences > reversed speech, speech > reversed speech, or words > sentences.

Exploratory thresholded maps showed scattered frontal and parietal regions where AVH− activation exceeded AVH+ activation during sentence processing relative to reversed speech. Exploratory maps also showed temporal and frontal regions where AVH+ activation exceeded AVH− activation. These maps were retained as descriptive visualizations only and were not treated as corrected evidence of localized subgroup differences.

Surface Brain Maps

Surface maps projected the sentences > reversed speech contrast onto cortical regions. Descriptive AVH+ greater-than-AVH− effects were most evident in the left lateral temporal cortex, especially the left middle temporal gyrus (L-MTG) and left superior temporal sulcus (L-STS). These results suggest that left temporal speech-processing regions may show different activation patterns across AVH subgroups, but the surface maps should be interpreted as exploratory because corrected whole-brain inference found no significant clusters.

Region-of-Interest Activation

Region-of-interest (ROI) analyses focused on speech and language areas with the strongest observed activation effects. For the sentences > reversed speech contrast, AVH+ patients showed greater activation than AVH− patients in the L-MTG. Mean activation was positive in AVH+ patients ($M = 0.131$, $SD =$

0.184) but slightly negative in AVH− patients (M = -0.024, SD = 0.165). The exploratory pairwise Welch test was t = 3.01, p = .0044, $|d|$ = 0.89.

A similar pattern was observed in the L-STS. AVH+ patients showed higher activation (M = 0.056, SD = 0.134) than AVH− patients (M = -0.063, SD = 0.125), t = 3.11, p = .0033, $|d|$ = 0.92. This represented the largest raw ROI effect. The negative mean activation in AVH− patients suggests reduced differentiation between sentences and reversed speech in this left temporal speech-processing region.

However, neither effect survived the corrected three-group omnibus analysis. For sentences > reversed speech, L-STS showed $F(2,45.24) = 5.06$, p = .0104, q = .0995, and L-MTG showed $F(2,44.53) = 4.50$, p = .0166, q = .0995. Therefore, the pairwise effects should be interpreted as exploratory rather than as corrected subgroup findings.

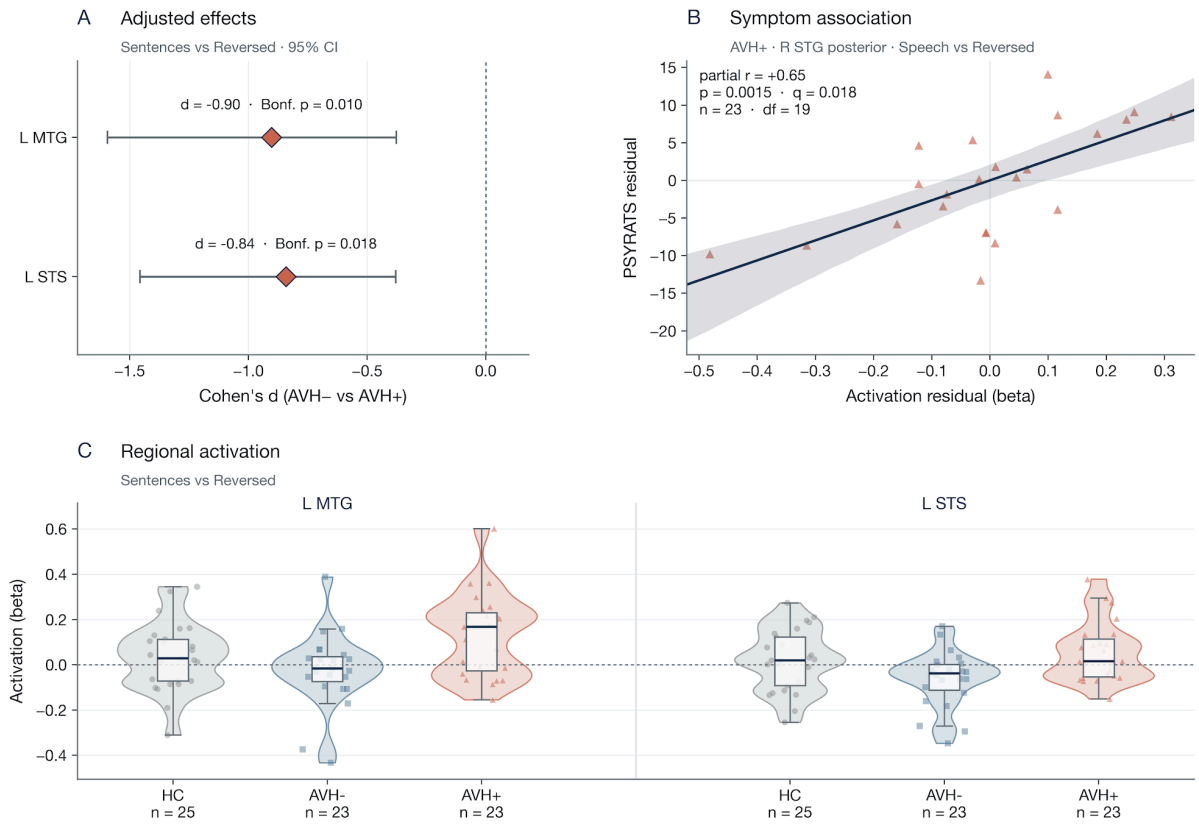


Figure 2: Summary of the principal replication results

For the words > sentences contrast, all three groups showed negative mean activation in the L-MTG, indicating greater activation for sentences than isolated words. AVH+ patients showed the most negative values (M = -0.118, SD = 0.125). The same general pattern appeared in the L-STS, where AVH+ patients showed more negative values than AVH− patients (M = -0.094, SD = 0.094 versus M = -0.008, SD = 0.107). These omnibus ROI effects also did not survive within-contrast FDR correction.

Detailed L-MTG Effect

One of the largest raw ROI effects was observed in the L-MTG for the sentences > reversed speech contrast. AVH+ patients showed substantially higher activation than AVH− patients in this region. Because the L-MTG contributes to speech and language comprehension, this result may indicate that

AVH+ patients recruit left temporal semantic-language regions differently during intelligible speech processing. However, the effect was identified post hoc and did not survive the corrected omnibus ROI family.

Post hoc covariate-adjusted sensitivity analyses showed similar patterns after adjustment for age, IQ, sex, and mean framewise displacement. With AVH− minus AVH+ as the modeled contrast, adjusted effects were negative: L-MTG, adjusted d = -0.90, raw p = .0052, two-test Bonferroni p = .0105; L-STS, adjusted d = -0.84, raw p = .0090, Bonferroni p = .0179. The full model used 69 complete cases, and the motion-clean model used 65. Because the two ROIs were selected after examination of the exploratory results, these analyses remain post hoc.

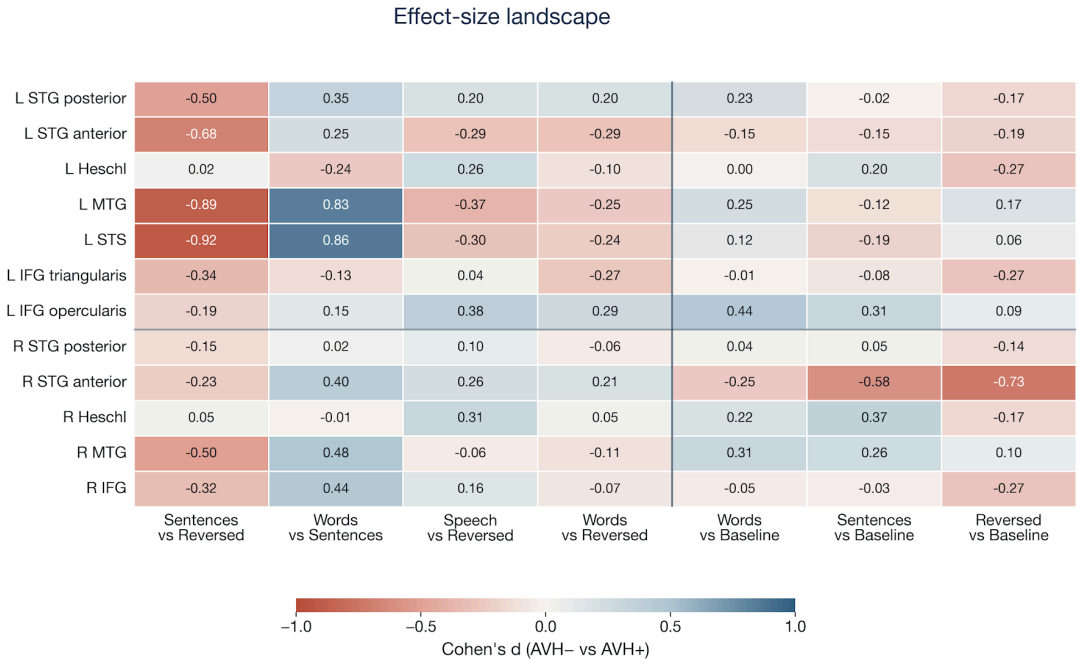


Figure 3: Exploratory effect-size overview across speech and langauge ROIs

PSYRATS Correlations

Clinical correlation analyses examined the relationship between PSYRATS-H hallucination severity and ROI activation during speech processing. The strongest association was observed in the right

posterior superior temporal gyrus (R-STG posterior) for speech > reversed speech. The raw correlation was $r = .586$, $p = .0033$, $q = .040$. After controlling for age and IQ, the partial correlation was $r = .647$, $df = 19$, $p = .0015$, $q = .018$. This exploratory pattern suggests that stronger right posterior temporal responses to intelligible speech may be associated with greater hallucination severity among patients who experience AVH.

Functional Connectivity

Exploratory functional connectivity analyses compared ROI-to-ROI connectivity between AVH− and AVH+ patients. Connectivity was estimated using Pearson correlations across BOLD time series from 12 speech-related ROIs, followed by Fisher *z* transformation. In the difference matrix, positive values indicated stronger connectivity in AVH− than AVH+ patients, while negative values indicated stronger connectivity in AVH+ than AVH− patients.

Two ROI pairs reached nominal significance at $p < .05$. The L-STS–right inferior frontal gyrus (R-IFG) connection was stronger in AVH− patients than AVH+ patients (difference = 0.11, $t = 2.26$, $p = .029$). The left posterior superior temporal gyrus (L-STG posterior)–L-MTG connection was stronger in AVH+ patients than AVH− patients (difference = -0.18, $t = -2.13$, $p = .039$). However, neither effect survived correction for multiple comparisons. These findings should therefore be treated as exploratory rather than conclusive evidence of altered functional connectivity. Task-evoked activity may also inflate task-connectivity estimates [13].

All-ROI Overview

Across 12 speech- and language-related ROIs, the clearest exploratory group differences appeared in left temporal regions. In the L-MTG, AVH+ patients showed positive sentence-related activation ($M = 0.131$), whereas AVH− patients showed little or no sentence-related activation. The raw effect size for this

difference was large ($|d| = 0.89$). The L-STS showed a similar pattern, with AVH+ patients showing positive activation ($M = 0.056$) and AVH− patients showing negative activation ($M = -0.063$), $|d| = 0.92$.

The left anterior superior temporal gyrus showed a moderate uncorrected group contrast, but the group distributions overlapped more strongly. Other left-hemisphere regions, including Heschl's gyrus, the opercular and triangular parts of the inferior frontal gyrus, and the posterior superior temporal gyrus, showed substantial overlap and values near zero. Right-hemisphere ROIs did not show clear categorical group differences between AVH+ and AVH− patients. No three-group ROI test survived within-contrast FDR correction.

Laterality and MVPA

Laterality analyses showed qualitative trends but did not identify significant AVH+ versus AVH− differences for the sentences > reversed speech contrast or for the other tested conditions. Healthy controls tended to show left-dominant responses across speech-related ROIs, whereas AVH− patients showed a more right-shifted temporal pattern with relatively preserved left inferior frontal involvement. AVH+ patients showed stronger right-sided involvement in posterior superior temporal regions during speech processing and weaker left inferior frontal dominance than healthy controls or AVH− patients. These patterns are consistent with possible right-hemisphere bias in auditory-temporal processing and reduced left-frontal specialization for sentence processing in AVH+ patients, but the absence of statistically significant laterality effects means that these findings should be treated as descriptive.

MVPA analyses also did not provide strong evidence for a clean AVH+ versus AVH− distinction. Whole-brain classification used 40 patients with available maps (20 per group). Accuracy ranged from 42.5% to 50.0%, and all permutation *p*-values were at least .347. This suggests that multivariate activation patterns were not sufficient to reliably separate patients with hallucinations from patients without hallucinations.

Discussion

This study examined neural responses to auditory perception in individuals with schizophrenia with and without auditory verbal hallucinations. The original study concluded that schizophrenia patients with and without AVH showed broadly similar auditory-language activation during speech perception. Both patient groups showed reduced activation in the left primary auditory cortex relative to healthy controls, suggesting a general auditory processing deficit associated with schizophrenia rather than a deficit specific to hallucinations [1].

Prior research suggests several possible neural routes by which hallucinations may arise, including altered interactions among auditory, language, memory, salience, and self-monitoring systems [2, 4]. The present replication focused on 12 speech- and language-related ROIs, including bilateral superior temporal gyrus regions, middle temporal gyrus, Heschl's gyrus, inferior frontal gyrus, and the left superior temporal sulcus. The left inferior frontal gyrus ROIs included pars triangularis and pars opercularis regions traditionally associated with speech production, while the posterior temporal ROIs sampled regions involved in speech perception and comprehension [5].

In our analysis, the clearest subgroup differences appeared in left temporal language ROIs. Hallucination severity in AVH+ patients was also positively associated with right posterior STG activation during intelligible speech, suggesting that individual differences in hallucination severity may be related to temporal speech-network response. Together, these patterns suggest that hallucinations may not reflect auditory stimuli being heard differently only at the earliest sensory stage. Instead, AVH may involve altered engagement of distributed speech networks, which could distort speech interpretation and increase the likelihood that internally generated cognitive material is misattributed as external speech.

Overall, the replication analysis was broadly consistent with the idea that auditory-language abnormalities in schizophrenia are not limited to AVH+ patients. At the same time, exploratory ROI and

surface-map analyses suggested possible subgroup differences in left temporal speech-processing regions, especially the L-MTG and L-STS. These results may reflect altered recruitment of temporal language networks during speech perception in patients who experience hallucinations.

However, these subgroup findings should be interpreted cautiously. The original study did not report significant corrected differences between AVH+ and AVH− patients, and the corrected replication analysis likewise found no significant whole-brain clusters FDR-significant ROI effects. Schizophrenia is associated with reduced auditory cortex activation overall, while AVH-related differences in temporal language activation, laterality, and connectivity remain possible but require further confirmation.

Limitations

Several limitations qualify these findings. First, the original experimental design used a passive block design with relatively coarse temporal resolution. Because BOLD responses were measured over blocks of words, sentences, and reversed speech, the analysis could not determine whether activation differed at the exact moments when AVH+ participants were experiencing hallucinations. The task also did not provide direct measures of word comprehension, attention, or effort, making it difficult to separate comprehension from engagement with the task.

Second, the dataset examined responses to speech stimuli rather than a broader range of auditory inputs. It is possible that activation patterns would differ for tones, environmental sounds, emotional voices, or self-generated speech. The AVH+ group also varied in hallucination frequency and severity, while the main grouping variable treated hallucination status as a categorical AVH+ versus AVH− distinction. A continuous approach to hallucination severity across patients may capture symptom variation more precisely.

Third, the replication analysis was cross-sectional and single-site, limiting generalizability to other scanners, samples, and clinical populations. Some secondary analyses also used reduced quality-controlled samples. A larger independent sample would be needed to establish whole-brain connectivity conclusions and to test whether ROI-level effects generalize reliably.

Finally, several follow-up analyses did not support a clean AVH+ versus AVH− distinction, suggesting AVH-related differences are subtle and partly dependent on analysis choices.

Despite these limitations, the broader patient-control finding remains important: schizophrenia patients showed reduced activation in the left primary auditory cortex during auditory perception, pointing to an early auditory information-processing deficit relating to some sort of cognitive-sensory dysfunction

Conclusion

Overall, AVH-related differences are reflected in speech-network engagement, especially in the left temporal regions, particualry L-MTG and L-STS. The broader pattern remains cautious as analyses did not show significant AVH+ versus AVH− differences despite qualitative trends, with the strongest clinical association being the positive relationship between PSYRATS-H hallucination severity and right posterior STG speech responses in AVH+ patients.

The observed evidence supports two main conclusions. First, reduced left primary auditory cortex activation appears to be a broader schizophrenia-related auditory processing deficit rather than a marker specific to auditory verbal hallucinations. Second, AVH may involve altered engagement of distributed speech and temporal-language networks, especially when hallucination severity is considered. These findings should be interpreted as promising but exploratory, and future work with larger samples, event-specific symptom measures, and preregistered correction procedures is needed to clarify whether AVH has a reliable neural signature during speech perception.

Data and Code Availability

The source dataset is publicly available from OpenNeuro under accession ds004302 (dataset DOI: 10.18112/openneuro.ds004302.v1.0.1). Soler-Vidal et al. (2022) [1] and code avalibility is located in repository JeremyManwaring/Schziophrenia_Speech_Analysis. (04fa579)

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