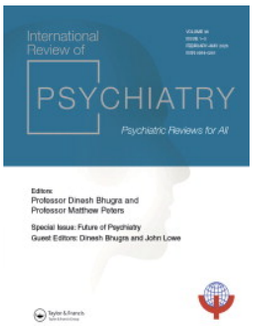

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RESEARCH ARTICLE



# Clinical and electrophysiological effects of cathodal high-definition transcranial direct current stimulation to the right superior temporal sulcus in psychosis spectrum disorders: a pilot proof of concept study

Rebekah L. Trotti[a,b], Nicolas Raymond[a], David A. Parker[c], Halide Bilge Turkozer[b,d], Prachi Patel[a], Brendan Stiltner[a], Daphne Ying[a], Willa Molho[a], Robert MG. Reinhart[e], Matcheri S. Keshavan[a,b] and Paulo Lizano[a,b,f]

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**ABSTRACT**

The right superior temporal sulcus (rSTS) plays a causal role in multisensory hallucinations, is associated with audiovisual integration (AVI), and displays abnormal activity in psychotic disorders. We sought to reduce psychosis symptoms including hallucinations while modulating AVI and rSTS activity with cathodal, high-definition transcranial direct current stimulation (HD-tDCS) of the rSTS. This double-blind, randomized, sham-controlled pilot study applied HD-tDCS to the rSTS using a ring montage (−1.5 mA cathode center and three .5 mA anode surrounds). Stimulation was administered ($N=6$ active, $N=6$ sham) for 5 days with 2 20-minute sessions per day. Assessments occurred at baseline, 5-day, and 1-month timepoints. Electroencephalography (EEG) was recorded during resting state and an AVI steady state (SSR) paradigm. Given the pilot nature of this study, analyses used an alpha threshold of .10. The active group demonstrated reduced clinician-assessed symptoms at 5-days and 1-month, attenuated AVI at 5-days, enhanced auditory SSR and attenuated alpha power over the rSTS at 1-month, and reduced self-reported symptoms at 5-days. Symptom changes correlated with alpha changes. Biological motion, functioning, mood, and cognition did not significantly change. Findings suggest therapeutic effects and successful target engagement. Results provide preliminary proof-of-concept evidence supporting the initiation of a larger trial in psychotic disorders.

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**KEYWORDS**
HD-tDCS; audiovisual integration; electroencephalography; psychosis; hallucinations

## Introduction

Auditory and visual hallucinations are common, prominent symptoms of psychosis spectrum disorders (PSD) like schizophrenia, and their presence predicts disability and disease outcomes (Clark et al., 2017). Existing treatments for hallucinations are often poorly tolerated, and up to 30% of patients are treatment resistant (Meltzer, 1997; Mørup et al., 2020). Despite decades of neuroscience research, few new or targeted treatments for hallucinations have been established. Transcranial electrical stimulation (tES) is informed by known neurological correlates of psychiatric illness and may provide a novel, well-tolerated treatment for hallucinations. Compared to other noninvasive neuromodulation approaches, tES is low-cost and portable, with potential for at-home use and clinical scalability. tES modulates neural activity by applying anodal or cathodal direct current to the cortex to affect the underlying neural membrane potential. Anodal and cathodal current are hypothesized to increase and decrease the resting potential, respectively, to change the rate of spontaneous neural firing in the targeted region (Yamada & Sumiyoshi, 2021).

Previous tES studies targeting hallucinations in schizophrenia have produced mixed results, and a recent meta-analysis by Guttesen et al. (2021), found no significant effect of tDCS on auditory hallucination severity (SMD ≈ −0.23, 95% CI: −0.49 to 0.02) compared with sham stimulation. However, most prior neuromodulation studies employed non-focal, two-electrode montages targeting the prefrontal cortex

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(Deniz et al., 2023; Li et al., 2025), with broad current fields and limited anatomical specificity. In contrast, the present study uses high-definition direct current stimulation (HD-tDCS), which increases spatial precision, allowing researchers to target specific cortical regions.

This study also uses a novel target for intervention. Though many areas have been implicated in hallucination production, lesion network mapping (Fox, 2018) suggests a causal role of the right superior temporal sulcus (rSTS) in hallucinations (Kim et al., 2021), suggesting it may be an ideal tES treatment target. This region is negatively connected with more than 90% of lesions causing hallucinations (Kim et al., 2021). Thus, when spontaneous activity decreases at lesion locations causing hallucinations, spontaneous activity in the rSTS increases. Thereby applying cathodal HD-tDCS to the rSTS may affect excitability of this region, modulating network dynamics to reduce the occurrence of hallucinations in people with PSD. We aimed to target the rSTS with cathodal HD-tDCS, examining both behavioral and neurophysiological changes to determine if this approach may reduce excitability of the rSTS to improve hallucinations. Targeting this biologically informed node with focal stimulation may address limitations of prior work and enhance engagement with specific causal circuitry. These considerations provide a framework for reconciling the present findings with earlier approaches and highlight the potential strengths of precise, network-guided neuromodulation.

The rSTS is active during multisensory hallucinations, receiving convergent somatosensory, auditory, and visual inputs. It is involved in audio-visual integration (AVI), which is impaired in people with PSD (Beauchamp et al., 2004; Gröhn et al., 2022; Jones & Powell, 1970; Mesulam, 1998; Tseng et al., 2020) and shares a strong association with hallucination severity (Stevenson et al., 2017). We reasoned that rSTS hyperexcitability may lead to hallucinations and increased global symptom burden by aberrant multisensory integration. AVI may be studied with electroencephalography (EEG) using steady-state stimuli, which are repetitive stimuli presented at a set frequency. Cortical neurons in the relevant sensory network entrain to the stimulus frequency, firing in a time-locked fashion at the stimulus' driving frequency, and eliciting a steady state response (SSR) (Picton et al., 2003; Vialatte et al., 2010). When auditory and visual steady-state stimuli are presented simultaneously, AVI may induce additional 'intermodulation' frequencies (Drijvers et al., 2021; Regan et al., 1995). We used this novel approach to measure AVI in this trial.

We conceptualized hallucinations in PSD as arising from aberrant multisensory binding within the rSTS, reflected in exaggerated intermodulation responses and altered intrinsic oscillatory activity. As a convergence zone for auditory and visual inputs, the rSTS is well positioned to support binding across modalities, and abnormal excitability in this region may promote excessive or inappropriate integration of sensory signals. This process can be indexed with intermodulation frequencies reflecting nonlinear integration across sensory streams. Based on this framework, we selected cathodal HD-tDCS as a targeted perturbation of rSTS circuit dynamics. While the effects of cathodal stimulation are likely not purely inhibitory, it is expected to bias local excitatory-inhibitory balance towards a lower-excitability state. We therefore paired stimulation with EEG measures of AVI and intrinsic activity to directly assess target engagement, rather than assuming a specific physiological effect underlying and clinical change.

Beyond its roles in AVI and hallucinations, the STS is involved in numerous other functions and behaviors including social cognition and perception, biological motion, theory of mind, speech and language (Deen et al., 2015; Hein & Knight, 2008) and general constructs like quality of life (Boyer et al., 2012). Due to these roles, rSTS modulation may also have a broader therapeutic effect on symptoms of PSDs. This trial will collect data on a range of psychiatric symptoms and behaviors to track these and other potential effects of neuromodulation.

To examine this hypothesis, this double-blind, sham-controlled pilot study examined if a 5-day course of twice daily, 20-minute cathodal HD-tDCS of the rSTS compared to sham stimulation may reduce clinical indicators of psychosis and hallucinations and enhance brain activity. We hypothesized that hallucinations will decrease and intermodulation frequency power will change from baseline to 5 day and 1-month follow-up in the active tDCS group but not sham. Given these hypotheses, we used a liberal alpha ($\alpha$ = .10) in this pilot proof of concept study. We also conducted exploratory analyses investigating if other psychosis symptoms, biological motion detection, global function, mood, and cognition change with stimulation.

## Methods

### *Participants*

This trial employed a randomized, double blind, parallel-arm design and took place at Beth Israel Deaconess Medical Center in Boston, MA. Inclusion criteria comprised diagnosis of schizophrenia,

schizoaffective disorder, or psychotic bipolar disorder by the Structured Clinical Interview for DSM-V (American Psychiatric Association, 2013), lifetime history of hallucinations, 18–80 years old, no changes to relevant antipsychotic medications for 1 month prior to participation, and a sufficient level of English for participation. Exclusion criteria included pregnancy or breastfeeding, IQ <60, any major neurologic illness, substance use disorder within 6 months of participation, positive urine drug screen, a history of moderate-to-severe visual impairment, significant suicidal ideation within 6 months of participation, serious medical illness or instability requiring hospitalization within the past year, metallic or electronic implants, recent or serious cardiac events, and skin sensitivity or claustrophobia precluding them from completing study protocols.

12 individuals with psychosis were enrolled and randomized to either sham ($n=6$) or active HD-tDCS ($n=6$; Figure 1). 12 participants completed a 5-day follow-up and 11 completed a 1-month follow-up. Participants and researchers involved in data collection and analysis were blind to study assignment.

All participants provided written informed consent prior to participation after obtaining a complete description of study procedures. The study was approved by the Institutional Review Board at Beth Israel Deaconess Medical Center (2019P0001016).

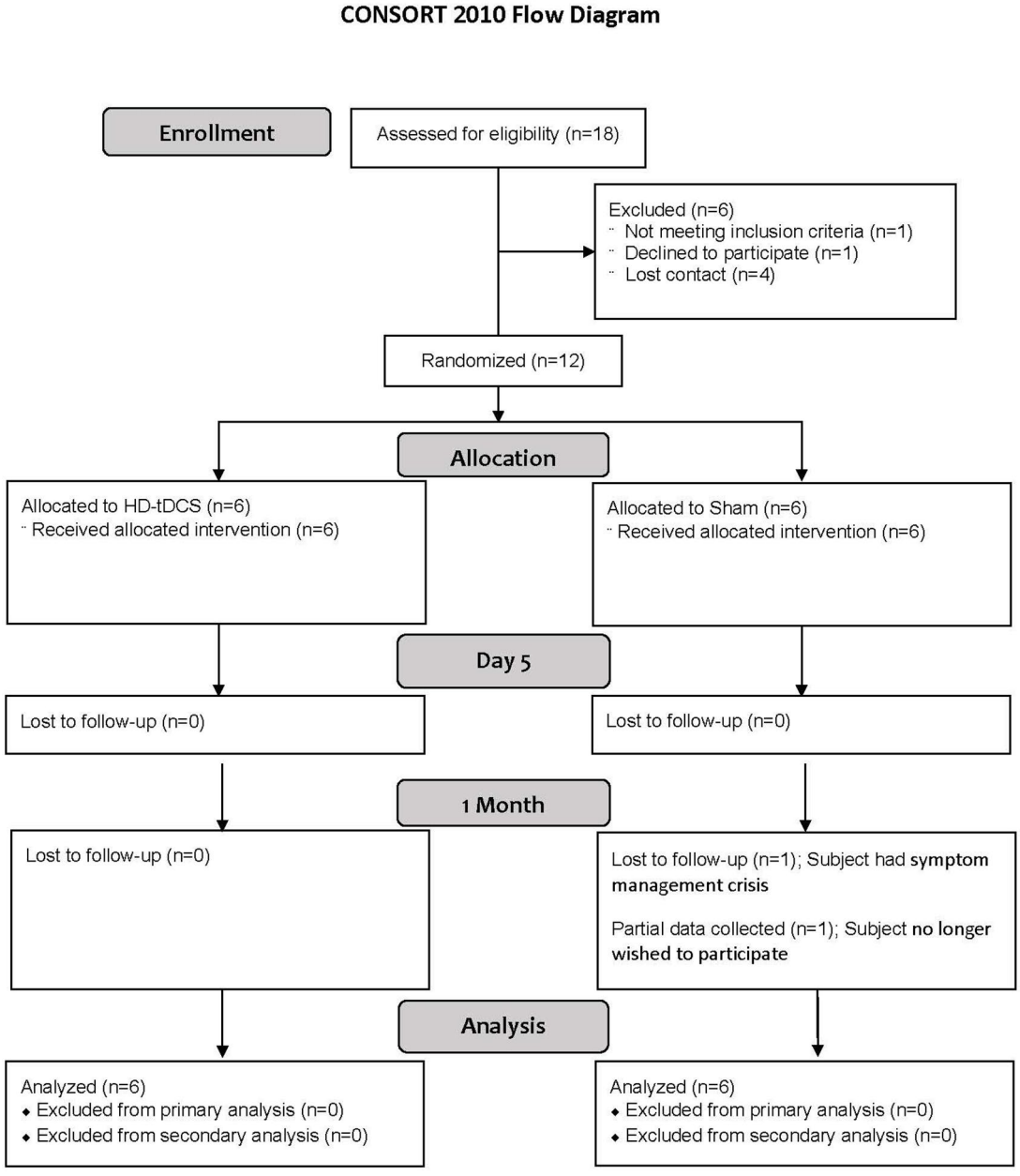


Figure 1. CONSORT Diagram for recruited participants in this trial.

### Procedures

HD-tDCS was administered using an MxN 9-channel HD-tES system (Soterix Medical) with sintered 12 mm Ag/AgCl electrodes in plastic holders, filled with conductive gel, and embedded in the Quik-Cap. Montage selection was guided by current flow modeling in HD-Explore (Soterix Medical) and ROAST (Huang et al., 2019), with the chosen montage balancing high focality and intensity at MNI coordinate x = 61, y = −20, z = −3. This montage includes one cathode (−1.5 mA) at T8, surrounded by 3 anodes (.50 mA each) at FT8, C6, and TP8 (Figure 2).

Participants received 2 consecutive 20-minute HD-tDCS sessions separated by a 5–10 minute break on 5 consecutive weekdays (Figure 2), as recommended by Kim et al.,2019 (Kim et al., 2019). The sham condition used the same montage, with only a 30 second ramp up, 30 second ramp down, and no stimulation for 20-minutes in-between.

Randomization used the 'blockrand' package in R Studio (Greg Snow, 2006) with block size set to 2. An unblinded researcher set the tES device to active or sham for each session out of view from the participant and blinded assessors. Topocaine 5 was applied 5 minutes before stimulation to minimize sensation differences between active and sham. After each session, participants completed a Sensation Questionnaire to gauge blinding adequacy and side effects (see Supplement).

### Primary outcomes

Participants completed the Positive and Negative Syndrome Scale for Schizophrenia (PANSS (Kay et al., 1987)) by clinician interview at all timepoints and subdomain scores were calculated (Positive, Negative, General).

### Active hallucinators

Participants experiencing active hallucinations completed the University of Miami Parkinson's Disease Hallucinations Questionnaire (UM-PDHQ; Papapetropoulos et al., 2008), which measures the severity of any sensory hallucination, and Auditory Hallucination Rating Scale (AHRS; Hoffman et al., 2003; Hoffman et al., 2005), which measures voice hearing hallucinations. 6 participants were experiencing active hallucinations (active $N=3$, sham $N=3$) and 4 were experiencing voice hearing (active $N=1$, sham $N=3$) at baseline. UM-PDHQ analyses used an average score from quantitative data across sensory modalities to simplify comparisons.

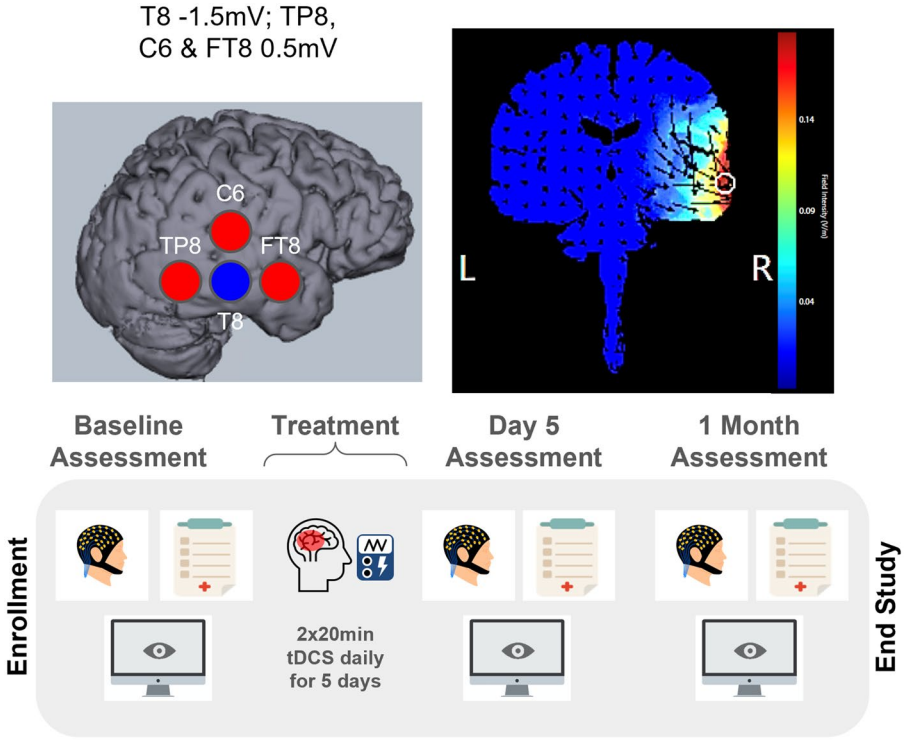


Figure 2. Study methods. The stimulation electrodes are shown at the top left with the cathode in blue and anodes in red. The resulting current flow model is shown at the top right, focused on the rSTS. The study timeline is shown on the bottom, depicting a baseline assessment, 5-day course of daily tDCS, 5-day follow-up, and 1-month follow-up.

### Secondary outcomes: EEG

12 participants completed EEGs at baseline and 5-day follow-up, and 10 completed EEGs at 1-month follow-up. 1 participant was excluded from all timepoints due to poor data quality and 1 was lost to follow-up. EEG employed a 128-channel HydroCel Geodesic Sensor Net (Magstim EGI) with participants in a sound- and light-shielded booth and impedances kept below 50 kΩ. Stimuli were presented using custom code (Neurobehavioral Systems).

#### AVI task

The AVI task contained auditory and visual steady state stimuli that were amplitude- and luminance-modulated at defined frequencies (ASSR: 40 and 80 hz, VSSR central visual field: 15 hz, VSSR peripherals: 12 hz; Supplement Figure S1). 20 trials were auditory-only (600 ms; half at 40 hz, half at 80 hz), 20 were visual-only (2000 ms), and 80 were audiovisual stimuli together. Participants were instructed to passively observe stimuli.

#### AVI variable extraction

Data were cleaned using the Harvard Automated Preprocessing Pipeline for Electroencephalography (HAPPE (Gabard-Durnam et al., 2018), parameters in Supplement), converted to time-frequency space in Matlab (EEGLab (Delorme & Makeig, 2004) newtimef), and averaged over trials. Power at fundamental frequencies and their harmonics (VSSR: 12, 15, 24, 30 hz; ASSR: 40 hz) were extracted from sensors over the relevant cortices (Supplement Figure S2) and intermodulation frequencies (40 hz ASSR ± VSSR frequencies (12/15 hz) and harmonics (24/30 hz): 55, 52, 70, 64, 25, 10, 28, and 16 hz) were extracted from sensors capturing rSTS activity (Figures S2, S3). To retain one composite measure for each stimulation type, we averaged over fundamental and harmonic frequencies for the VSSR and all intermodulation frequencies for AVI.

#### Resting state EEG

A 5-minute resting state (rsEEG) was collected as participants sat with their eyes loosely fixed on a white plus sign on a black screen. After preprocessing, data were transformed to time-frequency space in EEGLab (Delorme & Makeig, 2004) (see Supplement) and averaged into defined frequency bands consistent with Thomas et al.,2019 (Thomas et al., 2019): delta/theta (1–8 hz), alpha (8–12 hz), beta (13–30 hz), and gamma (30–55 hz). Power was averaged across sensors for an overall power metric and at stimulation sensors over the rSTS (Figure S2) for a ROI power metric.

As this study hypothesized changes in auditory-visual integration, the composite AVI measure was considered a hypothesis-driven outcome while VSSR, ASSR, and rsEEG measures were considered exploratory.

### Secondary outcomes: Behavioral and clinical

A biological motion task (Vonck et al., 2015) was administered where participants indicated which direction a figure was walking, with 3 increasing levels of nuisance dots (noise), to increase difficulty (easy, medium, and hard; 20 trials each). The percentage of correct responses at each level was calculated for analysis (see Supplement).

The Global Assessment of Function (GAF; Axis 5 on the DSM-IV (American Psychiatric Association, 2000)), Montgomery-Asberg Depression Rating Scale (MADRS (Montgomery & Asberg, 1979)), and Young Mania Rating Scale (YMRS (Young et al., 1978)) were completed by clinician assessment. Cognitive assessment used the Brief Assessment of Cognition (BAC App (Atkins et al., 2017)), administered by iPad (VeraSci, WCG Clinical). Composite and subdomain scores (see Supplement) were z-scored and adjusted based on normative scores (Keefe et al., 2004).

Finally, participants completed the Symptom Checklist-90 Revised (SCL-90R (Derogatis, 2017)) by self-report and subscores were calculated (Somatization, OCD, Interpersonal, Depression, Anxiety, Phobic, Paranoid, Psychoticism).

### Data analysis

#### Primary and secondary endpoints

Data were compared between baseline, 5-day, and 1-month timepoints. Primary endpoints included PANSS, UM-PDHQ, and AHRS scores. Secondary endpoints included EEG measurements from the AVI task (ASSR, VSSR, AVI) and resting state (4 frequency bands, ROI), biological motion accuracy, and GAF, MADRS, YMRS, BAC, and SCL-90R scores.

#### Statistics

Intent-to-treat analyses were conducted using custom code in R Studio. Multiple imputation was performed for missing datapoints using the 'Mice' package (Van Buuren & Groothuis-Oudshoorn, 2006) (0% baseline, 4.75% 5-day, 14.04% 1-month; see Supplement). Nonparametric ANOVAs with a group (active, sham) X time (baseline, 5-days, 1-month) design were conducted using the 'nparLD' package (Noguchi et al., 2012) to examine interaction effects and contrasts

employed nonparametric Wilcoxon Signed-Rank tests (within-subjects) and Mann-Whitney U tests (between-subjects). We also calculated Number Needed to Treat (NNT) using the metrics described by Mendes et al. (2017). We tested associations between changes in EEG and symptom variables by conducting Spearman correlations on change scores. We created change scores (follow-up minus baseline) for variables that significantly differed between timepoints. Holm-Bonferroni corrections were applied to multiple comparisons and rank biserial effect sizes were calculated. Since there were very few participants with active hallucinations (3 per group at baseline and 1-month, 2 per group at 5-days), only descriptive statistics for the UM-PDHQ and AHRS were calculated.

### Power analyses

Our prior trial of lesion-network guided tDCS demonstrated within-subjects effect sizes of .87 for clinical outcomes and .97–2.35 for EEG outcomes. Based on these effect sizes, we determined an alpha threshold of .10 would offer adequate power for this study (73% power for clinical and 63–99% power for EEG outcomes). Given that the intent of a pilot trial is signal detection, this threshold also has the advantage of minimizing false negatives. Following analysis, we used GPower (Kang, 2021) to complete post-hoc power analyses to estimate achieved power and a priori analyses to estimate appropriate sample sizes for a future confirmatory efficacy trial, reported in the (Supplement Table S12).

## Results

### Demographics

Individuals were diagnosed with schizophrenia ($N = 2$), schizoaffective disorder ($N = 5$), and bipolar I disorder with psychosis ($N = 5$). Sex distribution was equal between the sham and active groups (50% female). Age did not differ between sham (M = 37.5, SD = 14.84; range = 22–61) and active (M = 36.67, SD = 8.26; $F(1) = .01$, $p = .90$; range = 28–50). Groups also did not significantly differ on outcome variables at baseline (all $p > .10$).

### Primary endpoints

While few results survived corrections for multiple comparisons, statistical tests revealed several significant effects that suggest feasibility and mechanistic engagement.

#### PANSS

There were significant group X time interactions on the PANSS Total ($F(1,1.47) = 3.40$, $p = .048$) and General scores ($F(1,1.53) = 2.76$, $p = .08$). In the active group, PANSS Total significantly decreased from baseline to 5-days ($p = .03$, $p_{adj} = .09$; NNT = 2, 95% CI [1.02, 52.19]) and 1-month ($p = .05$, $p_{adj} = .11$; NNT = 2, 95% CI [1.02, 52.19]; Figure 3). General scores also decreased between baseline and 1-month ($p = .08$, $p_{adj} = .26$) in the active group. Though there was not a significant interaction on PANSS Positive, the active group also had decreased Positive scores at 5-days ($p = .09$, $p_{adj} = .27$) while sham did not. There were no significant interactions or within-group changes in the Negative subscale (full statistics and effect sizes in Table 1).

#### Active hallucinations

One participant per group stopped experiencing hallucinations at 5-days and one participant in the active group started experiencing low-intensity hallucinations at 1-month. The average UM-PDHQ score decreased for two active participants between baseline and 5-day and remained stable at 1-month. Sham participants remained approximately the same across time (Figure 4).

For the AHRS, the active participant experienced a 1-point increase in voice hearing frequency, but a 1-point decrease in how real they seem and loudness at the 5-day timepoint, for a total decrease of 1 point. At 1-month, all scores returned to baseline, but voice extensiveness increased by 2 points. In the sham group, AHRS Total slightly increased for 2 out of 3 participants at each timepoint (Figure 4).

### Secondary endpoints

#### Audiovisual EEG

There was a significant group X time interaction on the VSSR ($F(1,2) = 3.71$, $p = .02$), but neither group's scores significantly changed at either timepoint (all $p > .10$). Though there was not a significant group X time interaction on ASSR or AVI measures, ASSR significantly increased in the active group from 5-days to 1-month ($p = .06$, $p_{adj} = .18$) and AVI significantly decreased from baseline to 5-days ($p = .06$, $p_{adj} = .18$; Figure 5). There were no significant within-group effects at other timepoints or in the sham group (all $p > .10$; Table 2).

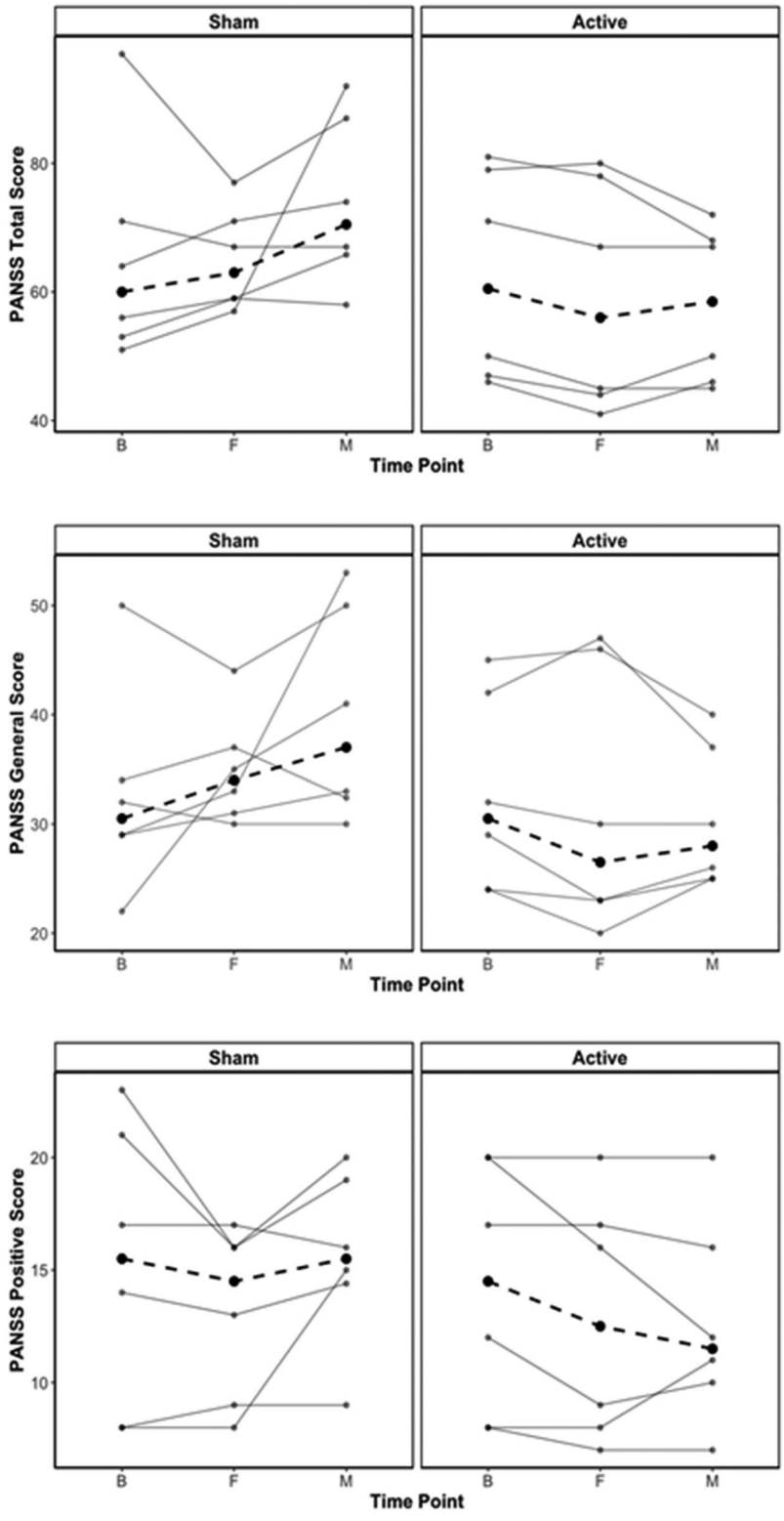


Figure 3. PANSS Total, Positive, and general scores for participants in the sham and active groups at baseline, 5-days, and 1-month (B/F/M). Solid lines represent individual participant data and bold dotted lines represent the group average.

### *Resting EEG*

There were no significant group X time interactions on resting EEG variables. However, in the active group, alpha power over the rSTS significantly decreased between baseline and 1-month ($p$=.06, $p_{adj=}$.18), but not at 5-days ($p$ = 1.0; Figure 5). All other measures and frequencies did not significantly change, and there were no effects in sham (all $p$ > .10; Table 3).

### Off-target effects

There were no significant group X time interactions on MADRS Total, BAC Composite, GAF, or biological motion (all $p > .10$), however there was a significant interaction on the YMRS Total (F(1,1.75) = 3.15, $p = .049$). In sham, YMRS significantly decreased from Baseline to 1-month (YMRS: $p = .06$, $p_{adj} = .18$), though there was no significant change in the active group ($p > .10$). The BAC Composite significantly decreased in the sham group from Baseline to 1-month as well ($p = .04$, $p_{adj} = .11$). There were no significant within-group changes in the active group or in the GAF, MADRS, or BAC subscales (all $p > .10$; Supplement Tables 3-4).

Table 1. Positive and negative syndrome scale results.

| | Comparison | Statistic | p | p adjusted | Rank Biserial r |
|---|---|---|---|---|---|
| Active HD-tDCS | | | | | |
| Total | B vs F | 20 | **0.03**** | **0.09*** | 0.90 |
| | B vs M | 14 | **0.05*** | 0.11 | 0.87 |
| | F vs M | 7 | 0.29 | 0.29 | 0.40 |
| Positive | B vs F | 6 | **0.09*** | 0.27 | 1.00 |
| | B vs M | 11 | 0.21 | 0.42 | 0.47 |
| | F vs M | 5.5 | 0.50 | 0.50 | 0.10 |
| Negative | B vs F | 6.5 | 0.36 | 0.71 | 0.30 |
| | B vs M | 8.5 | 0.13 | 0.40 | 0.70 |
| | F vs M | 3 | 0.61 | 0.71 | 0.00 |
| General | B vs F | 14.5 | 0.23 | 0.46 | 0.38 |
| | B vs M | 17.5 | **0.09*** | 0.26 | 0.67 |
| | F vs M | 9 | 0.39 | 0.46 | 0.20 |
| Sham HD-tDCS | | | | | |
| Total | B vs F | 8 | 0.74 | 1.00 | −0.24 |
| | B vs M | 5.5 | 0.88 | 1.00 | −0.48 |
| | F vs M | 1 | 0.97 | 1.00 | −0.87 |
| Positive | B vs F | 8.5 | 0.13 | 0.40 | 0.70 |
| | B vs M | 11.5 | 0.46 | 0.92 | 0.10 |
| | F vs M | 1 | 0.97 | 0.97 | −0.87 |
| Negative | B vs F | 11 | 0.50 | 1.00 | 0.05 |
| | B vs M | 6 | 0.71 | 1.00 | −0.20 |
| | F vs M | 5 | 0.79 | 1.00 | −0.33 |
| General | B vs F | 6.5 | 0.83 | 1.00 | −0.38 |
| | B vs M | 3 | 0.91 | 1.00 | −0.60 |
| | F vs M | 2 | 0.95 | 1.00 | −0.73 |

*Note.* Statistics were performed with one-tailed Wilcoxon Signed-Rank Tests.
*$p < .10$.
**$p < .05$.

### Self-reported symptoms

There was a significant group X time interaction on the SCL-90 Total (F(1,1.77) = 2.55, $p = .08$). In the active group, SCL90-R Total significantly decreased at 5-days ($p = .06$, $p_{adj} = .18$), explained by reductions in Somatization, OCD, and Anxiety (Figure 6, Table S5). The active group's SCL90-R Total returned to baseline at 1-month ($p = .01$, $p_{adj} = .11$), explained by significant increases in Somatization and Interpersonal scores (Figure 6, Table S5). In sham, SCL90-R Total significantly increased from baseline to 5-days ($p = .06$, $p_{adj} = .18$) and Somatization significantly decreased from baseline to 1-month ($p = .09$, $p_{adj} = .19$) and 5-days to 1-month ($p = .01$, $p_{adj} = .11$; all statistics in Supplement Table S5).

### Correlations

Change scores were calculated by subtracting Baseline values from both 5-day and 1-month data for measures that significantly changed in the active group (clinical: PANSS Total, Positive, General, SCL90-R; EEG: alpha ROI rsEEG, ASSR, AVI), resulting in 6 total correlations. 1-month change in alpha ROI rsEEG was significantly and positively correlated with PANSS Total ($p = .07$, $p_{adj} = .35$, r = .77) and PANSS General changes ($p = .05$, $p_{adj} = .30$, r = .81; Figure 5).

## Discussion

This pilot HD-tDCS trial examined if cathodal rSTS stimulation could reduce psychosis symptoms, including hallucinations, and improve audiovisual integration. Though this was a small proof-of-concept trial

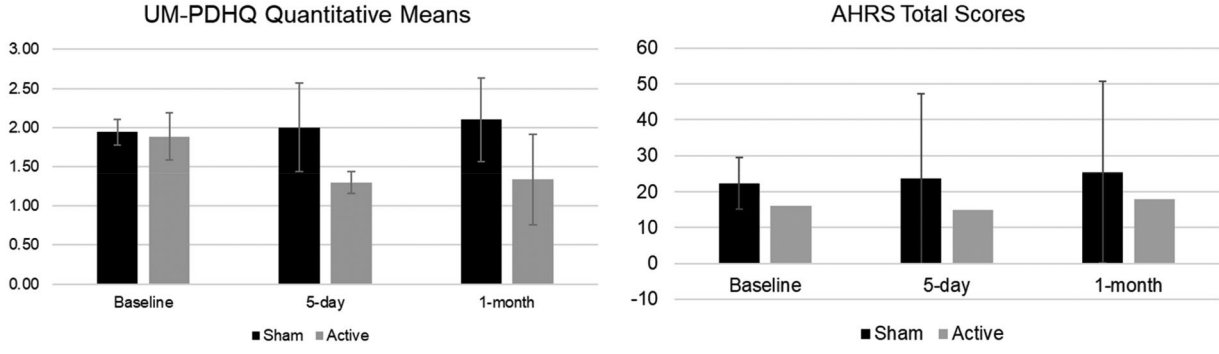


Figure 4. Active hallucination descriptive statistics. The average score for active tDCS participants ($N = 3$) decreased at 5 day and 1 month but remained stable for sham participants ($N = 3$). For the AHRS, the active participant's score decreased by 1 while the sham average score slightly increased ($N = 3$).

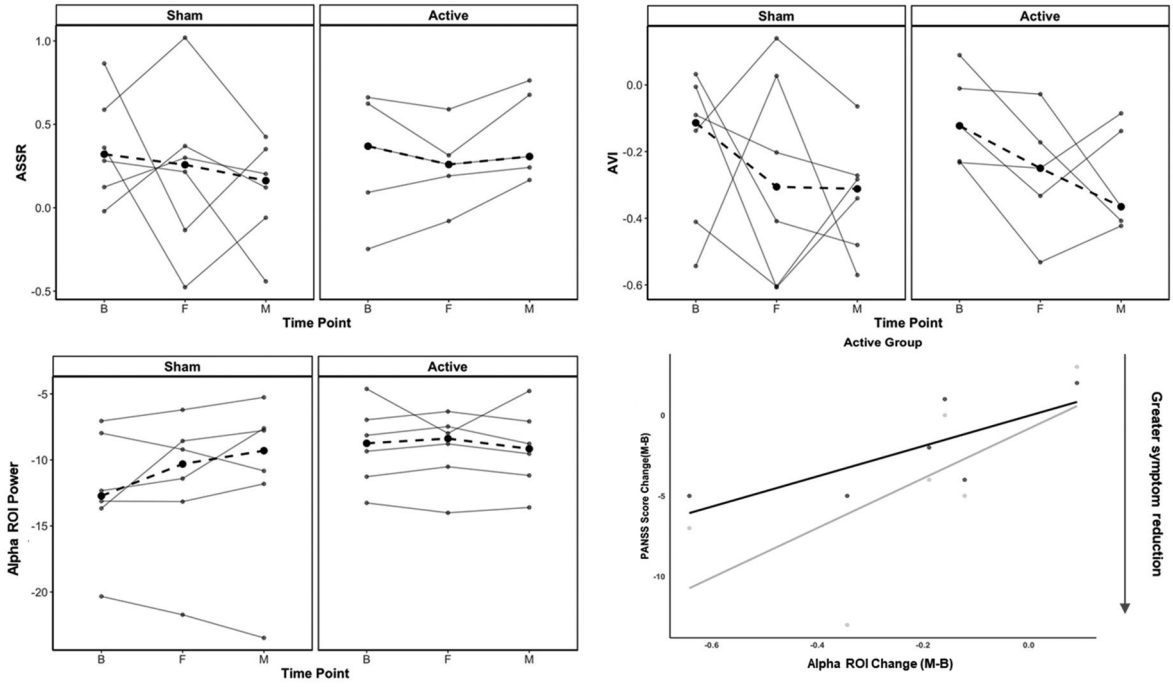


**Figure 5.** rsEEG, ASSR, and AVI scores and correlations at baseline, 5-day, and 1-month (B/F/M). There were significant effects over time in the active group, but not in the sham group. Solid lines represent individual participant data and bold dotted lines represent the group average. Alpha ROI change correlated with changes in both PANSS Total and General.

**Table 2.** Audiovisual EEG results.

| | Comparison | Statistic | *p* | *p* adjusted | Rank Biserial r |
|---|---|---|---|---|---|
| Active HD-tDCS | | | | | |
| ASSR | B vs F | 9 | 0.79 | 0.79 | 0.20 |
| | B vs M | 2 | 0.18 | 0.36 | −0.73 |
| | F vs M | 0 | **0.06*** | 0.18 | −1.00 |
| AVI | B vs F | 15 | **0.06*** | 0.18 | 1.00 |
| | B vs M | 13 | 0.18 | 0.36 | 0.73 |
| | F vs M | 9 | 0.79 | 0.79 | 0.20 |
| VSSR | B vs F | 2 | 0.18 | 0.36 | −0.73 |
| | B vs M | 1 | 0.11 | 0.32 | −0.87 |
| | F vs M | 6 | 0.79 | 0.79 | −0.20 |
| Sham HD-tDCS | | | | | |
| ASSR | B vs F | 12 | 0.83 | 1.00 | 0.14 |
| | B vs M | 18 | 0.14 | 0.43 | 0.71 |
| | F vs M | 14 | 0.53 | 1.00 | 0.33 |
| AVI | B vs F | 13 | 0.67 | 1.00 | 0.24 |
| | B vs M | 16 | 0.29 | 0.88 | 0.52 |
| | F vs M | 12 | 0.83 | 1.00 | 0.14 |
| VSSR | B vs F | 11 | 1.00 | 1.00 | 0.05 |
| | B vs M | 18 | 0.14 | 0.43 | 0.71 |
| | F vs M | 13 | 0.67 | 1.00 | 0.24 |

*Note.* Statistics were performed with one-tailed Wilcoxon Signed-Rank Tests. ASSR = Auditory Steady-State Response, AVI = Audiovisual Integration, VSSR = Visual Steady-State Response.
*$p < .10$.

and few p-values survived corrections, results generally supported hypotheses and merit further study. In the active group, symptoms improved by clinician and self-report along with a qualitative decrease in hallucination measures, indicating preliminary clinical efficacy. EEG measures showed modulation of auditory processing, audiovisual integration, and alpha activity over the stimulated cortex, suggesting successful engagement of the rSTS. Further, PANSS and alpha changes significantly correlated, suggesting a mechanistic link between neurophysiology and symptoms. Overall, this trial provided preliminary evidence that focal, cathodal modulation of the rSTS can alter both neurophysiological signatures of audiovisual integration and clinical symptoms in psychosis. We interpret these effects within a circuit-level framework in which the rSTS functions as a multisensory integration node whose excitability shapes the binding of auditory and visual inputs. Aberrant integration, reflected in dysregulated intermodulation responses, may contribute to hallucination-related perceptual distortions.

Our primary hypothesis was that cathodal rSTS stimulation would reduce hallucinations given the causal role of the rSTS in hallucination production, which was partially supported by results. PANSS Positive decreased at 5-days and trend-level results suggested reduced UM-PDHQ and AHRS-measured hallucinations that were preserved at 1-month. Though hallucination analyses were underpowered, they are supported by our prior case study, which showed a substantial drop in AHRS with rSTS stimulation (Raymond et al., 2025) and changes in ASSR, suggesting a specific impact of stimulation on sensory processing. In future trials, we will continue to examine the effects of stimulation on hallucinations, given their prevalence in psychotic disorders.

**Table 3.** Resting state EEG results.

| | Comparison | Statistic | *p* | *p* adjusted | Rank Biserial *r* |
|---|---|---|---|---|---|
| Active HD-tDCS | | | | | |
| Delta/Theta | B vs F | 10 | 1.00 | 1.00 | −0.05 |
| | B vs M | 3 | 0.14 | 0.43 | −0.71 |
| | F vs M | 7 | 0.53 | 1.00 | −0.33 |
| Delta/Theta ROI | B vs F | 14 | 0.53 | 1.00 | 0.33 |
| | B vs M | 7 | 0.53 | 1.00 | −0.33 |
| | F vs M | 5 | 0.29 | 0.88 | −0.52 |
| Alpha | B vs F | 8 | 0.67 | 0.88 | −0.24 |
| | B vs M | 15 | 0.40 | 0.88 | 0.43 |
| | F vs M | 16 | 0.29 | 0.88 | 0.52 |
| Alpha ROI | B vs F | 10 | 1.00 | 1.00 | −0.05 |
| | B vs M | 20 | **0.06*** | 0.18 | 0.90 |
| | F vs M | 14 | 0.53 | 1.00 | 0.33 |
| Beta | B vs F | 14 | 0.53 | 0.88 | 0.33 |
| | B vs M | 15 | 0.40 | 0.88 | 0.43 |
| | F vs M | 16 | 0.29 | 0.88 | 0.52 |
| Beta ROI | B vs F | 6 | 0.40 | 0.88 | −0.43 |
| | B vs M | 13 | 0.67 | 0.88 | 0.24 |
| | F vs M | 16 | 0.29 | 0.88 | 0.52 |
| Gamma | B vs F | 11 | 1.00 | 1.00 | 0.05 |
| | B vs M | 14 | 0.53 | 1.00 | 0.33 |
| | F vs M | 16 | 0.29 | 0.88 | 0.52 |
| Gamma ROI | B vs F | 6 | 0.40 | 1.00 | −0.43 |
| | B vs M | 9 | 0.83 | 1.00 | −0.14 |
| | F vs M | 15 | 0.40 | 1.00 | 0.43 |
| Sham HD-tDCS | | | | | |
| Delta/Theta | B vs F | 12 | 0.83 | 1.00 | 0.14 |
| | B vs M | 11 | 1.00 | 1.00 | 0.05 |
| | F vs M | 11 | 1.00 | 1.00 | 0.05 |
| Delta/Theta ROI | B vs F | 11 | 1.00 | 1.00 | 0.05 |
| | B vs M | 11 | 1.00 | 1.00 | 0.05 |
| | F vs M | 12 | 0.83 | 1.00 | 0.14 |
| Alpha | B vs F | 4 | 0.21 | 0.63 | −0.62 |
| | B vs M | 6 | 0.40 | 0.80 | −0.43 |
| | F vs M | 9 | 0.83 | 0.83 | −0.14 |
| Alpha ROI | B vs F | 10 | 1.00 | 1.00 | −0.05 |
| | B vs M | 7 | 0.53 | 1.00 | −0.33 |
| | F vs M | 9 | 0.83 | 1.00 | −0.14 |
| Beta | B vs F | 12 | 0.83 | 1.00 | 0.14 |
| | B vs M | 11 | 1.00 | 1.00 | 0.05 |
| | F vs M | 12 | 0.83 | 1.00 | 0.14 |
| Beta ROI | B vs F | 11 | 1.00 | 1.00 | 0.05 |
| | B vs M | 14 | 0.53 | 1.00 | 0.33 |
| | F vs M | 18 | 0.14 | 0.43 | 0.71 |
| Gamma | B vs F | 13 | 0.67 | 1.00 | 0.24 |
| | B vs M | 14 | 0.53 | 1.00 | 0.33 |
| | F vs M | 13 | 0.67 | 1.00 | 0.24 |
| Gamma ROI | B vs F | 12 | 0.83 | 1.00 | 0.14 |
| | B vs M | 11 | 1.00 | 1.00 | 0.05 |
| | F vs M | 16 | 0.29 | 0.88 | 0.52 |

*Note.* Statistics were performed with one-tailed Wilcoxon Signed-Rank Tests. ROI = Region of Interest over the rSTS.
*$p < .10$.

The potential therapeutic effect of stimulation was best reflected in the PANSS Total and General, and supported by the SCL90-R, indicating that both clinicians and patients can detect clinical improvement following HD-tDCS. PANSS Total scores decreased with a large effect at 5-days that was preserved at 1-month, indicating a short-term effect of stimulation that plateaus over time. This change at 5-days was the only test to survive multiple comparisons in the active group, fit a more conservative alpha cutoff of .05, and had a Number Needed to Treat of 2, suggesting that 5-day PANSS Total effects are the most robust indicator of intervention response. PANSS General also decreased at 1-month, suggesting broad effects on symptoms other than hallucinations. Importantly, there was also a significant group X time interaction on PANSS Total and General, indicating that the changes observed in the active group significantly outweighed the changes over time in sham. Furthermore, SCL90-R results supported these effects, with a significant group X time interaction where only the active group's Total scores decreased at 5-days, accounted for by reduced somatization, OCD, and anxiety.

As these observed clinical changes are quite broad, they could be explained by the STS's role in networks underlying numerous functions like social skills, face processing, speech processing, and theory of mind (Hein & Knight, 2008). Other noninvasive brain stimulation studies targeting the STS have similarly modulated face recognition (Gobbo et al., 2024), relationship memory (Marrero et al., 2020) and biological motion (Grossman et al., 2005). We speculate that downstream effects of these basic processes, like social functioning and complex cognition, may explain broad effects of HD-tDCS on psychiatric symptoms. Subsequent studies with increased statistical power and additional measures will further examine these possibilities.

Physiologically, we used audiovisual and resting state EEG to examine engagement of the rSTS, finding reduced AVI power at 5-days and rsEEG alpha over the rSTS at 1-month that correlated with PANSS changes. Though the directionality of neurophysiological AVI deficits in psychosis is unclear (Grohn et al., 2024), some studies suggest that neural hyperactivity during multisensory facilitation may characterize psychosis spectrum disorders (Stone et al., 2011). Given this prior work, it is possible that reduced AVI power could indicate neurophysiological improvement, where HD-tDCS moved neural activity closer to healthy levels. Further, the decrease in alpha rsEEG and correlation with PANSS changes supports the hypothesis of rSTS engagement and association with behavioral changes. However, like the reduction of AVI frequencies, it is not clear if this reduction represents a normalization, as a healthy comparison group is needed to clarify this possibility.

Though results are preliminary, enhanced ASSR, reduced AVI, reduced rSTS alpha power, and a correlation with behavior suggest that stimulation has an effect on its targeted cortex, consistent with our hypothesis that HD-tDCS will modulate rSTS activity to induce clinical improvement. Although the effects

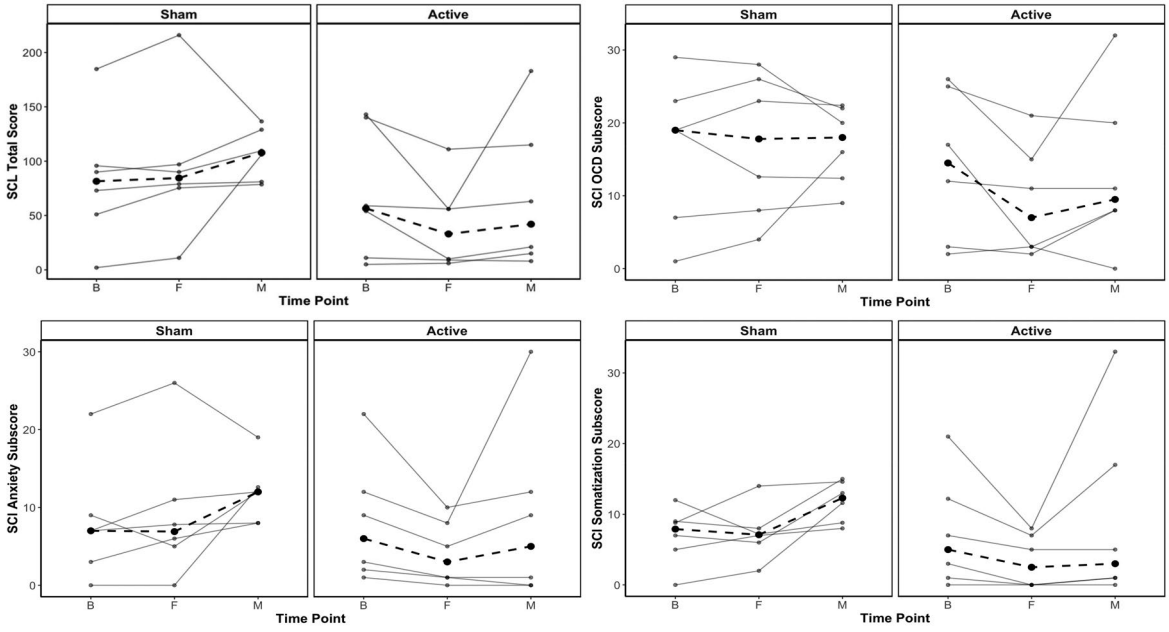


**Figure 6.** Changes in the SCL-90 between baseline, 5-day, and 1-month timepoints (B/F/M). Solid lines represent individual participant data and bold dotted lines represent the group average.

of cathodal stimulation are likely complex and cannot be reduced to a purely inhibitory mechanism (Lefaucheur & Wendling, 2019), these converging findings suggest that the intervention engaged rSTS circuit dynamics in a directionally meaningful way. Future studies will further assess these neurophysiological changes, including source analysis, behaviorally-relevant sensory tasks, and causal statistics, to test potential mechanisms underlying effects. Other studies directly comparing different neuromodulation methods (eg. rTMS, HD-tDCS, or tACS) targeting the rSTS may help clarify how distinct perturbations interact with circuit architecture to produce convergent or dissociable effects on multisensory integration and hallucinations.

More broadly, these results highlight the importance of combining targeted neuromodulation with intermediate neurophysiological measures. Rather than assuming that stimulation produces a specific change in excitability, this approach directly tests whether the intended circuit has been engaged and if changes in the circuit relate to behavioral outcomes. This framework may be particularly important for early-phase studies, where establishing target engagement is a critical step toward developing more precise interventions.

Regarding off-target effects, no within-group changes were seen in mood, functioning, or cognitive measures for the active group. Effects of sham on YMRS and BAC could be explained by practice effects, low power, or regression to the mean. Additionally of-note, the active group also did not demonstrate full stability on biological motion (see Supplement), suggesting that active stimulation may have slightly modified this function given the proximity of the rSTS to the motion processing region, V5/MT. In future studies, we will consider personalizing stimulation to individual neuroanatomy to more precisely target the intended region.

Strengths of our study include its novelty and a mechanistic approach, but the small sample size is a significant limitation. Additionally, interindividual variability, the wide age range, and diagnostic heterogeneity could impact results, as some prior studies have observed that individual brain features, age, and genetics impact tDCS response (Li et al., 2015). Furthermore, concurrent medications for mood and psychotic symptoms may interact with tDCS-induced neural changes. Therefore, effects should be interpreted with caution until larger trials are conducted. Still, results of this pilot study are promising, suggesting that cathodal rSTS stimulation is potentially efficacious for psychosis symptoms and successfully engages rSTS neurophysiology. Also notably, no significant adverse effects were reported (Supplement Figure S3), indicating that this novel therapy is feasible and safe. Larger clinical trials are needed to better evaluate suitability of the approach for clinical practice and should consider samples enriched with hallucinator participants, personalized neuroanatomical targeting, and pre-specified EEG biomarkers.

## Author contributions

CRediT: **Rebekah L. Trotti**: Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing; **Nicolas Raymond**: Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing – review & editing; **David A. Parker**: Conceptualization, Formal analysis, Methodology, Resources, Software, Validation, Visualization, Writing – review & editing; **Halide Bilge Turkozer**: Resources, Software, Writing – review & editing; **Prachi Patel**: Data curation, Investigation, Validation, Writing – review & editing; **Brendan Stiltner**: Investigation, Validation, Writing – review & editing; **Daphne Ying**: Investigation, Validation, Writing – review & editing; **Willa Molho**: Investigation, Validation, Writing – review & editing; **Robert MG. Reinhart**: Funding acquisition, Supervision, Writing – review & editing; **Matcheri S. Keshavan**: Conceptualization, Funding acquisition, Supervision, Writing – review & editing; **Paulo Lizano**: Conceptualization, Funding acquisition, Methodology, Project administration, Resources, Supervision, Writing – review & editing.

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