**Title:** The Efficacy of Neural Stimulation in Individuals With Schizophrenia

**NCT number:** NCT05746494

**Document date:** Nov. 19, 2024

# Trial Protocol

## 1. Study Overview

Paranoia is the unfounded and pervasive belief that people intend to hurt oneself (Freeman et al., 2007) and is a prominent symptom in schizophrenia spectrum disorders (SSD) (Green & Phillips, 2004), with over 90% of individuals with first-episode SSD showing heightened levels of paranoia (Moutoussis et al., 2007). Elevated levels of paranoia can predict the onset of psychosis in youth with clinical high risk (Cannon et al., 2008), and has been associated with impaired social functioning (Hajdúk et al., 2019; Pinkham, Harvey, et al., 2016), more suicidal ideation and behaviors (Freeman et al., 2011, 2019), and increased violence (Coid et al., 2013; Keers et al., 2014) in individuals with SSD. Given these negative consequences, efficient treatments for paranoia are desperately needed in SSD. Therefore, there has been growing interest in elucidating the neuropathological processes underpinning paranoia, which may pave the way for developing biologically based interventions, such as neurostimulation, among patients with significant levels of paranoia. A technique used in this project, transcranial direct current stimulation (tDCS), may hold significant promise for these purposes due to its few adverse effects, tolerable experience, relatively low cost, and portability. Additionally, this study's use of ecological momentary assessments (EMA) will facilitate a better understanding of how treatment effects can be observed in, and impacted by, real-world social situations, thereby laying the groundwork for elucidating the duration of real-world improvements and developing potential long-term treatment gains for individuals with SSD.

## 2. Background

### *2.1 Occurrence of Paranoia*

Paranoid ideation is the unfounded and pervasive belief that people intend to harm oneself (Freeman, 2007) and is a prominent symptom in schizophrenia spectrum disorders (SSD) (Freeman et al., 2002; Green & Phillips, 2004), with over 90% of individuals with first episode SSD showing heightened levels of paranoia (Moutoussis et al., 2007). In collaboration with the World Health Organization, an early prospective study followed 1379 individuals with psychotic risks in 10 countries and found that when symptomatic individuals made their initial contact with services, nearly 50% of all cases of psychosis were characterized by the presence of persecutory delusions, the second most common symptom being delusions of reference (Sartorius et al., 1986). Social-environmental risk factors for paranoid ideation include adverse childhood experiences (e.g., trauma, parental neglect) (Bentall et al., 2012; Shevlin et al., 2015; Sitko et al., 2014), deprived neighborhoods (Wickham et al., 2014), social stress (Kesting et al., 2013), lower social rank (Berry et al., 2018; Freeman et al., 2014; Saalfeld et al., 2018), and discrimination (Janssen et al., 2003). At the individual level, cognitive and psychological antecedents of paranoia include anxiety and depression (Ben-Zeev et al., 2009; Freeman et al., 2012), sleep disturbance (Freeman et al., 2009; Kasanova et al., 2020), anomalous internal experience (Freeman, 2007; Freeman & Garety, 2014), worrying thinking style (Freeman & Garety, 2014; Martinelli et al., 2013), negative cognitions about the self and others (e.g., low self-esteem, being critical of the self, expecting negative interpersonal interactions) (Bentall & Fernyhough, 2008; Freeman & Garety, 2014), as well as reasoning biases (e.g., "jumping to conclusions", belief updating) (Dudley et al., 2016; Krkovic et al., 2021; Pytlik et al., 2020). Although paranoia has historically been linked to SSD, it is now conceptualized as existing on a transdiagnostic

continuum (Combs et al., 2006). Emerging research has shown that paranoid thinking is not restricted to a single diagnosis; instead, it is present in a variety of clinical conditions, including major depressive disorder and bipolar disorder (Bentall et al., 2009), post-traumatic stress disorder (PTSD) (Alsawy et al., 2015; Freeman et al., 2013), and Alzheimer's disease (AD) (Kloszewska, 1998; Ropacki & Jeste, 2005). For example, in 2003, Cook and colleagues (Cook et al., 2003) proposed a distinction between paranoid and misidentification subtypes of AD, with a median prevalence of delusions in AD estimated to be 36% (Ropacki & Jeste, 2005). In AD, delusions typically involve paranoid elements, such as the belief that people are trying to hurt or steal (Kloszewska, 1998). This continuum also extends into the general population where the prevalence of paranoid ideation was estimated to be strikingly high (i.e., at least 10-15%) (Freeman, 2007). Similarly, in a large community sample in England (N = 7281), 18.6% adults endorsed paranoid thoughts, and 1.8% reported being the target of possible plans to inflict them with serious harm (Freeman et al., 2011). This pattern extends into adolescence with a recent study based on 801 adolescents in the United Kingdom finding that 7-32% of teenagers reported having paranoid thoughts on a weekly basis and that girls demonstrate considerably higher levels of paranoia than boys (Bird et al., 2019).

*2.2 Paranoia and Social Functioning*

Elevated levels of paranoid ideation can predict the onset of psychosis in youth with clinical high risk (Cannon et al., 2008) and have been linked to a number of negative outcomes including more suicidal ideation and behaviors (Freeman et al., 2011, 2019), increased violence (Coid et al., 2013; Keers et al., 2014), and impaired social functioning (Hajdúk et al., 2019; Pinkham, Harvey, et al., 2016) in individuals with SSD. Social functioning appears to be particularly impacted. In comparison to non-paranoid patients with schizophrenia, patients with active paranoid ideation demonstrated larger deficits maintaining social interactions and social acceptability in the real world (Pinkham, Harvey, et al., 2016). A recent study on children and adolescents (age = 11-17 years; N = 301) seeking mental health services revealed that paranoia exhibited the strongest unique association with peer problems and was linked to increased psychological problems over time (Bird et al., 2021). Further, Fett and colleagues (Fett et al., 2022) examined the daily situations associated with paranoid thoughts in clinical patients with psychotic disorders, first-degree relatives, and healthy controls based on the experience sampling method (ESM). They found that patients were frequently alone (i.e., 68.5% of the study time) and had higher levels of paranoia when alone compared to being with other people. In contrast, their first-degree relatives experienced more paranoia when spending time with others than when alone, and there was no significant relation between time alone vs. with others and paranoia in the healthy controls (Fett et al., 2022). Previous findings also indicate that difficulties in interpersonal relationships were strongly related to "the perception of being discussed by others behind their back" in both patients with SSD and a community sample as revealed in a study based on network analyses (Hajdúk et al., 2019). In the patient group, the "perceived prior experience of being treated unfairly" aspect of paranoia was uniquely associated with worse social functioning, while the "negative opinions about people" aspect of paranoia was uniquely linked to poorer interpersonal functioning in the general community (Hajdúk et al., 2019). Finally, in both clinical and general populations, paranoia appears to mediate the relationship between childhood adversity and social functioning (Palmier-Claus et al., 2016).

Research on subclinical paranoia has also provided similar evidence regarding the

negative impacts of paranoia on social life. A large national study (N = 7281) found that paranoia was linked to impaired social functioning, reduced perceived social support and social cohesion, and more workplace stress (Freeman et al., 2011). Behaviorally, individuals with subclinical paranoia maintained further physical distance from the researcher and spent more time reading the consent form (Combs & Penn, 2004) as compared to non-paranoid peers. Individuals with heightened sub-clinical paranoia also reported less social involvement, fewer social interactions, and more social competence difficulties (Combs et al., 2013) than their peers. Literature regarding the associations between paranoia and specific types of interpersonal relationships is relatively limited, with only 2 studies considered relevant here. One early study investigated the relation between personality disorder symptoms and marital functioning in 99 newly-married couples in the community using a daily-dairy method. Results indicated that increased paranoid personality disorder was negatively correlated with relationship sentiment (South, 2014). More recently, a study examining how paranoia impacts roommate relationships in 103 same-sex college roommate dyads revealed that students with higher paranoia reported lower roommate relationship satisfaction and poorer college adjustment, and that students who had roommates with higher levels of paranoia also reported lower satisfaction with their roommates (Springfield, Ackerman, et al., 2021).

*2.3 Paranoia and Social Cognition*

The observed social difficulties associated with paranoia may arise from a variety of deficits in social cognition: the mental processes of perceiving, interpreting, and reacting to the social environment (Green et al., 2015). Social cognitive deficits have been identified as a core feature of mental disorders with marked interpersonal dysfunction, including SSD (Green et al., 2015, 2019; Penn et al., 2008). Among all social cognitive domains, hostility/hostile interpretation biases and the propensity to mistrust unfamiliar faces appear to be at the center of paranoid ideation. When encoding ambiguous social information, individuals with elevated levels of paranoia displayed more negative interpretation bias as measured by the scrambled-sentences task (SST), such that they created more paranoid sentences compared to the non-paranoid individuals (Savulich et al., 2015, 2017; Trotta et al., 2021). Paranoid individuals with schizophrenia, in particular, demonstrated hostile attributional biases [measured by the Ambiguous Intentions Hostility Questionnaire (AIHQ)] and the tendency to view unfamiliar others as untrustworthy (measured by the Trustworthiness Task) (Pinkham, Harvey, et al., 2016). Comparatively, in the healthy population, individuals with subclinical paranoia showed greater hostile and blaming attributional biases in ambiguous situations (measured by AIHQ) compared to those with low paranoia (Combs et al., 2013), and higher levels of paranoia were negatively associated with trustworthiness ratings (measured by the Trustworthiness Task) (Klein et al., 2018). As noted above, perceived hostility is one core feature of paranoid ideation, thus the Hostility Scale of the Personality Inventory for DSM-5 (PID-5-HS) has also been used to as a proxy measure of paranoia, which revealed strong associations with AIHQ and paranoia (Buck et al., 2016).

*2.4 Neural Correlates of Paranoia: The Amygdala-Prefrontal Circuits*

Given the negative consequences of heightened paranoia, there has been increasing interest in identifying the neuropathological mechanisms underlying paranoid ideation. Identification of such a mechanism would offer promise for developing biologically based interventions, such as neurostimulation, among patients with significant levels of paranoia.

*2.4.1 The critical role of the Amygdala*

The etiology of paranoid ideation may have a neural basis rooted in networks involving the amygdala, an important hub of the limbic system that has been implicated in multiple processes related to paranoia (e.g., trust, fear conditioning) (Davis & Whalen, 2001; Koscik & Tranel, 2011). Numerous studies have supported the role of the amygdala in unconscious fear (Öhman et al., 2000, 2007), conditioned fear (Rogan et al., 1997; Wilensky et al., 2006), perceiving fearful or threatening facial expressions (Adams et al., 2003; Adolphs et al., 1995; Graham et al., 2007) and linguistic threats (Isenberg et al., 1999; Schmidt et al., 2010), as well as detecting saliency related to reward (Anderson & Phelps, 2001; Cunningham & Brosch, 2012; Mahler & Berridge, 2012; Zheng et al., 2017). In addition, the amygdala is important in differentiating between ingroup and outgroup members (Hart et al., 2000; McCutcheon et al., 2018), which some propose as the fundamental evolutionary function of paranoid ideation (Raihani & Bell, 2019). For example, a recent study recruited Black and White British participants and asked them to view Black and White race faces while undergoing MRI scanning (McCutcheon et al., 2018). They found that both groups exhibited increased activity in right amygdala in response to outgroup faces; however, greater right amygdala activation was observed in the Black group when viewing outgroup faces than in the White group, and lower neighborhood ingroup density was associated with greater right amygdala activation to white faces in the Black group (McCutcheon et al., 2018). The interpretations of these findings are consistent with prior literature suggesting that increased amygdala activation/responses may reflect perceived outgroup threats instead of racial bias or prejudice (Chekroud et al., 2014). Indeed, although paranoia is characterized by the conviction that others intend to cause harm, these assumptions often apply to a specific social group (e.g., neighbors, police) and involve misunderstanding of group boundaries and activities (Raihani & Bell, 2019).

In patients with SSD, neuroscientific findings are somewhat counterintuitive, in that reduced, rather than increased, amygdala activity has been found in paranoid individuals when completing social threat perception tasks (Goghari et al., 2010; Pinkham, Hopfinger, Pelphrey, et al., 2008) and when viewing pictures of negative emotions (Williams et al., 2004, 2007). To reconcile these findings with increased amygdala activity in non-clinical populations under fearful and threatening situations (LaBar et al., 1998; McCutcheon et al., 2018; Öhman, 2005; Phelps et al., 2001), research revealed that such task-related amygdala hypoactivity was actually associated with heightened resting cerebral blood flow (CBF) in schizophrenia patients with prominent paranoid ideation, compared with non-paranoid schizophrenia patients and healthy controls (Pinkham et al., 2015). Task-driven neural activity and resting-state/baseline neural functioning capture distinct features of brain function. In traditional functional magnetic resonance imaging (fMRI), signals are collected based on oxygen levels in the blood (i.e., BOLD signal), with the rationale that increased neuronal activity will lead to more oxygen use and subsequent blood flow from the surrounding regions to support metabolism (Heeger & Ress, 2002). Therefore, stimulus-based signals can reveal the regions associated with certain brain functions corresponding to the tasks, whereas resting state signals reflect spontaneous brain activity without performing any tasks (Smitha et al., 2017). As such, these findings imply that schizophrenia patients with active paranoid ideation may have elevated levels of amygdala activity at rest and without being exposed to any threatening stimuli, which may in turn produce a ceiling effect and limit the potential for increased task-evoked activation in amygdala imaging

studies when truly threatening stimuli are presented (Pinkham et al., 2015). Collectively, the literature indicates that heightened amygdala function at baseline may be posited as a crucial neural mechanism underlying paranoid ideation and that neuromodulation techniques aiming to inhibit/suppress amygdala function at rest may have important therapeutic implications in reducing paranoia.

*2.4.2 The Amygdala-prefrontal Circuits*

Intriguingly, the amygdala collaborates with the prefrontal cortex (PFC) in processing and modulating responses to potential threats (Gold et al., 2015; Lee et al., 2012), and importantly, PFC is hypothesized to receive and down-regulate threat signals detected by the amygdala (Gold et al., 2015; Lee et al., 2012; Wager et al., 2008), suggesting that effective modulation of amygdala by PFC may limit paranoid thinking. Supporting this mechanistic model, previous studies have found that PFC-amygdala coupling may undergird successful emotion regulation (Lee et al., 2012), and decreased activations in PFC have been reported in paranoid individuals with schizophrenia (Williams et al., 2004, 2007). Further, increased functional connectivity within limbic networks (i.e., between hippocampus and amygdala) and PFC has been associated with elevated levels of paranoia in patients with SSD (Walther et al., 2021), and hyperconnectivity between amygdala and PFC was also found in a paranoid patient group compared with a non-paranoid patient group in our previous work specifically examining the neural circuits implicated in paranoid ideation (Fan et al., 2021). Collectively, these findings indicate that the observed hyperconnectivity may be due to a hypervigilant amygdala that is constantly sending false alarms to PFC and that ineffective regulation of these signals by PFC may result in paranoid thinking. As such, these findings also point to the possibility of attenuating paranoid thinking by stimulating PFC, which may further regulate and inhibit amygdala function.

The VLPFC in particular represents an important region in modulating amygdala functioning (Monk et al., 2008) and inhibiting paranoid ideation, as suggested by previous work linking reduced activity in VLPFC and increased activation in amygdala to elevated levels of paranoia (Pinkham, Hopfinger, Ruparel, et al., 2008). When comparing brain activations in rating untrustworthy vs. trustworthy faces in paranoid individuals with schizophrenia, there was no significant difference in activations of the social cognitive network [including amygdala, fusiform gyrus, superior temporal sulcus, ventrolateral prefrontal cortex (VLPFC), medial PFC (MPFC)]; however, a greater activation of these brain regions for untrustworthy faces was observed in nonparanoid individuals with schizophrenia and healthy controls, and such an increased activation was correlated with better social functioning (Pinkham, Hopfinger, Ruparel, et al., 2008). Although the literature convergently highlights the major role of the VLPFC-amygdala circuit in developing and maintaining paranoid ideation, these studies are correlational and do not involve any experimental manipulation of VLPFC and/or amygdala function to reveal the hypothesized causal effects of these processes on paranoia. As detailed below, transcranial direct current stimulation (tDCS) offers a way to directly alter neural activation that can be used to test such mechanistic models.

*2.5 Neural Stimulation in Schizophrenia*

*2.5.1 Transcranial Direct Current Stimulation (tDCS)*

In recent years, there has been a growing interest in employing tDCS to modulate brain function noninvasively (Berryhill, 2014; Filmer et al., 2014). tDCS delivers low amplitude direct

current to the brain through electrodes placed on the scalp, which theoretically hyperpolarizes or depolarizes the membrane potentials and then alters the excitability of the cortex (i.e., to increase or decrease the likelihood of neuronal firing) (Brunoni et al., 2012; Priori et al., 2009). Further, the effects of tDCS are hypothesized to extend beyond the stimulation time through synaptic plasticity (Kronberg et al., 2017; Liebetanz et al., 2002), suggesting that long-term brain, and the corresponding behavioral, alterations might be expected as a result of repetitive tDCS. In general, tDCS has been found effective in improving the symptoms of patients with depression (mostly by targeting brainstem and the frontal cortex) (Fregni et al., 2006; Kalu et al., 2012), AD (mostly by targeting the temporal cortex) (Boggio et al., 2011, 2012; Cai et al., 2019), and stroke (Elsner et al., 2013; Hamilton et al., 2011). In healthy populations, tDCS has also demonstrated efficacy in improving cognitive function (e.g., working memory) (Berryhill & Jones, 2012; Hoy et al., 2013) and motor processing ability (Antal et al., 2011; Hunter et al., 2009). Additional benefits of tDCS include few adverse effects, high tolerability, relatively low cost, and high portability (Bikson et al., 2016, 2018).

Despite the promise of tDCS and the early successes noted above, tDCS has many unknowns regarding its mechanism and best practices for application, which can make the process of experimental design difficult. There is no universally accepted methodology for the optimal stimulation protocol, and the current understanding of the short- and long-term processes of tDCS is still somewhat limited (Nitsche et al., 2008). General considerations include positioning of electrodes, the intensity and duration of stimulation, “online” vs. “offline” stimulation (detailed below), single vs. multiple/repeated sessions, and individual variability. In stimulating human brains, low-intensity currents (i.e., a few mA) are often employed; when applying tDCS to individuals with SSD, a range of 1-3 mA has been used in previous research, with 2 mA being the most frequently used intensity parameter (Brunoni et al., 2014; Gupta et al., 2018). Each stimulation session typically lasts between 15 and 30 minutes, with 20 minutes being the most usual duration (Brunoni et al., 2014; Gupta et al., 2018). Other more complicated methodological considerations involve choosing between an “online” or “offline” study design. “Online” stimulation means that the main outcomes are assessed during the stimulation, while “offline” stimulation means that participants are asked to sit quietly during stimulation rather than engage in a specific task, assuming that aftereffects of tDCS will be observed in subsequent task performance (Brunoni et al., 2012). The “online” design is posited to train new cognitive or motor abilities, and thus may be most beneficial to rehabilitation (Priori et al., 2009). For example, the majority of studies treating post-stoke aphasia used the “online” approach as demonstrated in a review (de Aguiar et al., 2015), probably because online tDCS may maximize the benefits of speech therapy sessions by facilitating treatment effects, while offline tDCS is hypothesized to only activate the language system and prepare it for the tasks that will be used in subsequent interventions (de Aguiar et al., 2015). Finally, it is not surprising that repeated sessions may produce more prominent treatment effects compared to just one single session. A recent study that applied single-session tDCS to patients with schizophrenia found null results in reducing auditory hallucinations (Selvaraj et al., 2021); on the contrary, a meta-analysis revealed that patients with schizophrenia who received tDCS twice daily or more than 10 sessions in total had reduced auditory hallucinations, and that those who received at least 10 tDCS sessions also reported reduced negative symptoms (Kim et al., 2019). Similar suggestions come from another meta-analysis indicating that higher frequencies (e.g., 2 sessions each day) and total number of

stimulation sessions (≥10) are essential to obtain optimal treatment benefits in schizophrenia (Cheng et al., 2020). Notably, tDCS can be administered safely even when used twice daily (2mA, each for 20 min) for up to 4 weeks, as demonstrated by a previous study on schizophrenia patients (Lindenmayer et al., 2019).

*2.5.2 Effects of tDCS on Psychotic Symptoms in Schizophrenia*

Previous research demonstrates that tDCS holds promise in ameliorating some symptoms of schizophrenia, in that early case reports and recent studies using randomized control trials have found significant improvement in hallucinations and negative symptoms in patients with SSD by stimulating DLPFC (Bose et al., 2014, 2018; Brunelin et al., 2012; Chang et al., 2018, 2020; Gomes et al., 2015, 2018; Lindenmayer et al., 2019; Palm et al., 2016; Sreeraj et al., 2018; Valiengo et al., 2020). Furthermore, improvement in social functioning (Chang et al., 2020; Narita et al., 2017; Shiozawa et al., 2013) and clinical insight (Bose et al., 2014; Chang et al., 2018, 2019, 2021; Kao et al., 2020) has also been observed in individuals with SSD, suggesting that the therapeutic efficacy of tDCS to DLPFC may be multifold and could have generated functional improvement in real life. However, null effects (Fitzgerald et al., 2014; Selvaraj et al., 2021; Smith et al., 2020) and placebo effects (Fröhlich et al., 2016) have also been reported, suggesting that further investigation is needed. Additionally, the abovementioned studies mostly investigated hallucinations and positive symptoms in general, with no study focusing specifically on paranoia or examining whether the beneficial effects of tDCS can be observed for paranoia symptoms.

*2.5.3 VLPFC as the Stimulation Target*

As alluded, mounting research has highlighted the role of VLPFC in the pathophysiology of schizophrenia as well as paranoid ideation. For example, decreased activation in VLPFC was observed in individuals at ultra-high-risk for psychosis (Koike et al., 2011), and reduced VLPFC gray matter volume was associated with more negative symptoms in individuals with schizophrenia (Zierhut et al., 2013). A previous study observed decreased functional connectivity between the left inferior frontal gyrus (IFG) and VLPFC in patients with auditory verbal hallucinations compared to healthy controls, indicating that hallucinations may result from a disconnection between the speech production system (e.g., IFG) and the verbal monitoring system (e.g., VLPFC) (Clos et al., 2014). Altered structure and activation of VLPFC have also been linked to impaired cognitive function in psychosis, including impaired working memory (Goldstein et al., 2011; Zierhut et al., 2013), attention (Schneider et al., 2007), cognitive (Buchy et al., 2016; Orfei et al., 2013) and metacognitive insight (Spalletta et al., 2014), as well as motor (Kaladjian et al., 2007) and response inhibition (Kaladjian et al., 2011).

Furthermore, VLPFC is also involved in processing and responding to social information. Not surprisingly, VLPFC collaborates with amygdala to help facilitate successful emotion regulation (Green et al., 2015), a process that is compromised in psychosis (Modinos et al., 2010; Morris et al., 2012; Van Der Velde et al., 2015). In addition, increased response in VLPFC was observed when viewing angry faces compared with neutral faces in individuals with a history of suicidal behaviors (Olié. et al., 2015), emphasizing the role of VLPFC in social threat perception. VLPFC has also been linked to the "jumping to conclusions" bias (JTC; a cognitive distortion in which individuals make judgements before evaluating all pertinent information thoroughly) (Langdon et al., 2010) in schizophrenia. As demonstrated by a recent meta-analysis (Vucurovic et al., 2020), JTC was linked to increased effective connectivity between a task-positive network

and a task-negative network (with VLPFC as the region-of-interest to form the network) in both patients with delusions and healthy individuals (Andreou et al., 2018). Intriguingly, in the general population, attenuated VLPFC activity and connectivity were associated with higher social anhedonia (Hooker et al., 2014; Yin et al., 2015), poorer emotion management (Yin et al., 2015), and more paranoid ideation (Hooker et al., 2014), indicating that reduced VLPFC functioning may contribute to elevated levels of paranoia, which may in turn lead to more active social withdraw behaviors to prevent potential distress and harm. Collectively, these studies provide important evidence supporting VLPFC as a target for reducing paranoid ideation in schizophrenia, and indicate that empirical studies examining the efficacy of modulating VLPFC functioning on paranoia are warranted.

To begin to address this gap, our preliminary work tested the efficacy of single-session tDCS to VLPFC among undergraduate students with heightened subclinical paranoia. While preliminary, we found a significant reduction in paranoid ideation after active stimulation but not sham stimulation (Springfield, Isa, et al., 2021). Combined with previous work reporting that regions of VLPFC demonstrated increased functional connectivity with amygdala in paranoid patients compared with non-paranoid patients (Fan et al., 2021), our pilot tDCS study indicates that VLPFC may be a potentially important treatment target for clinical levels of paranoia in patients with SSD. As such, using tDCS to impact functioning of VLPFC not only offers perspective regarding the neurobiological mechanisms of paranoid ideation by providing evidence of a causal link between VLPFC activity and the experience of paranoia, but also holds important implications for treating clinical levels of paranoia in patients with SSD.

## 3. Project Outline, Goals, and Hypotheses

To address these issues, the current research aimed to investigate the effects of tDCS on paranoid ideation and social functioning in individuals with SSD. Individuals who met diagnostic criteria for schizophrenia or schizoaffective disorder, demonstrated clinically significant levels of recent paranoia, and had no contraindications for neural stimulation (e.g., presence or history of epilepsy or seizures) were recruited. Our proposed mechanistic model suggests that either decreasing activation of the amygdala or increasing activation of prefrontal regulatory regions (e.g., VLPFC) may reduce paranoia. However, given the amygdala's subcortical location, VLPFC was targeted in the present study as it is more feasibly modulated via tDCS. A double-blind, crossover design was used to administer both active and sham stimulation conditions to each participant to allow within-subject comparisons. The most widely adopted and commonly used parameters were utilized during active stimulation to ensure safety and efficacy (i.e., 2mA, 20min) (Brunoni et al., 2014; Gupta et al., 2018). To increase potential efficacy, 2 sessions of stimulation were administered with a time interval of ≥3 hours (Bose et al., 2014; Brunelin et al., 2012) for each condition. We utilized a higher frequency here compared with our pilot study (Springfield, Isa, et al., 2021) because the alleviation of clinical levels of paranoia may require multiple sessions of stimulation (Cheng et al., 2020; Selvaraj et al., 2021), which may increase efficacy as suggested by previous research (Cheng et al., 2020; Kim et al., 2019). Offline stimulations were employed because there are no tasks that can be used to train the neural networks related to paranoid ideation (i.e., online stimulation) and previous literature indicates that cognitive activity during tDCS may attenuate or even abolish the stimulation effects (Horvath et al., 2014). Finally, in addition to measuring improvements in paranoia and

social functioning in the lab setting, we also administered short daily surveys to participants multiple times per day for 7 days before and after the 1st stimulation session, as well as after the 2nd stimulation session. These ecological momentary assessments (EMA) allow for capturing baseline paranoia severity as well as any delayed effects of neural stimulation in the real world.

*3.1 Aims and Hypotheses*

The specific goals and hypotheses of this project are listed below:

***Goal 1:*** To examine whether applying tDCS to VLPFC will reduce paranoid symptoms in SSD.

*Hypothesis 1a:* Patients with SSD will show reduced paranoia symptoms after the active VLPFC stimulation condition relative to sham stimulation as measured by *in-lab assessments* (i.e., decreased self-report paranoia, less attributional bias and hostility, more trust to strangers).

*Hypothesis 1b:* Patients with SSD will show reduced paranoia symptoms after the active VLPFC stimulation condition relative to sham stimulation as measured by *EMA assessments* (i.e., reduced feelings of paranoia in daily life).

**Goal 2:** To examine whether applying tDCS to VLPFC will improve social functioning in SSD.

*Hypothesis 2a:* Patients with SSD will show better social performance after the active VLPFC stimulation condition relative to sham stimulation as measured by *in-lab assessments* (i.e., improved self-report of social functioning).

*Hypothesis 2b:* Patients with SSD will show better social functioning after the active VLPFC stimulation condition relative to sham stimulation as measured by *EMA assessments* (i.e., more interactions with others, more positive experience in, and expectations of, daily social interactions).

## 4. Participants

A total of 62 participants (age = 18-64) diagnosed with schizophrenia or schizoaffective disorder with current (in the past week) or recent (in the past month) paranoia were enrolled in the study. Twelve participants withdrew ($n = 4$), were lost to follow-up ($n = 7$), or were determined ineligible after the enrollment visit ($n = 1$), resulting in 50 participants completing the whole procedure (see **Table 1** in the main article). Non-completers did not differ significantly from the completed sample in age ($p = 0.813$), gender ($p = 0.693$), race ($p = 0.321$), ethnicity ($p = 0.970$), IQ ($p = 0.322$), years of education ($p = 0.538$), handedness ($p = 0.861$), or CPZ-equivalent ($p = 0.176$). However, a higher proportion of non-completers were not taking any medications for their mental disorders ($n_{Not\text{-}taking\ medication} = 8$, 66.66% of the subsample) compared to completers ($n_{Not\text{-}taking\ medication} = 3$, 6.00% of the subsample; $\chi^2$ (1) = 24.41, $p < 0.001$), suggesting a lower adherence to treatment and possibly more severe psychopathological symptoms in this population. Supporting this notion, more non-completers showed current paranoia ($n_{current\ paranoia} = 12$, 100.00% of the subsample) compared to completers ($n_{current\ paranoia} = 38$, 76.00% of the subsample; $\chi^2$ (1) = 3.57, $p = 0.059$) at a marginally significant level.

*4.1 Sample Size Estimation*

In our pilot study using an undergraduate sample, a medium effect size was revealed in the active stimulation group (Cohen's $d = 0.51$), thus it was used for sample size estimation using G*Power 3.1 (Faul et al., 2009) in the current study. For research goals 1 and 2, we planned to use repeated-measures ANOVAs to test the tDCS effects on VLPFC on paranoia and social functioning, and power analyses yielded a sample size of 46 in total ($f = 0.25$, power =

0.80, α = .05, correlation among repeated measures = 0.3) in order to detect a medium effect. Given the potentially high retention rate due to the repeated-stimulation characteristics of this study, we intentionally overrecruited. Recruitment continued until the end of January 2024, aligning with the anticipated completion of our data collection period. As a result, a total of 63 participants consented, 50 of whom were randomized and completed the study.

*4.2 Recruitment and Eligibility*

The current sample was recruited from a database of previous participants in our lab and via mental health agencies in the Dallas-Fort Worth region. Study fliers were placed in outpatient clinics, and interested individuals either contacted study personnel directly or asked to be contacted by the study team. This recruitment approach has been consistently used by our research team supervised by Dr. Amy Pinkham, and has been found to yield a substantial number of participants, respect patient privacy, and minimize the possibility of coercion.

Eligible participants were between the ages of 18 and 64 inclusively, had a diagnosis of schizophrenia or schizoaffective disorder, and demonstrated either current (in the past week) or recent (in the past month) paranoia. Participants were not asked to change any part of their current treatment regimen, and all medications were accepted. Participants were required to have been clinically stable for a minimum of 6 weeks with no dose changes >20% for a minimum of two weeks. Exclusion criteria included: 1) presence or history of a pervasive developmental disorder or mental retardation as defined by IQ < 70, 2) presence or history of neurological or medical disorders that contraindicate neural stimulation (e.g. presence or history of epilepsy, seizures, etc.), 3) demonstrating sensory limitations, including uncorrectable visual or hearing impairments that interfere with assessment, 4) history of electroconvulsive therapy, 5) lack of proficiency in English, and 6) substance use disorder not in remission in the past 6 months.

## 5. Procedure

As shown in **Figure 1** below, this was a double-blind, within-subjects, crossover design comparing the effects of active vs. sham tDCS. Participants completed 3 long visits (i.e., 1 enrollment visit, 2 stimulation visits). The 2 stimulation visits were at least a week apart (with an average of 14.58 days between the stimulation visits in the current study) to make sure that the tDCS effects from the first visit dissipated, and the first simulation visit was expected to occur 7 days after the enrollment visit (average days = 13.59 days in the current study).

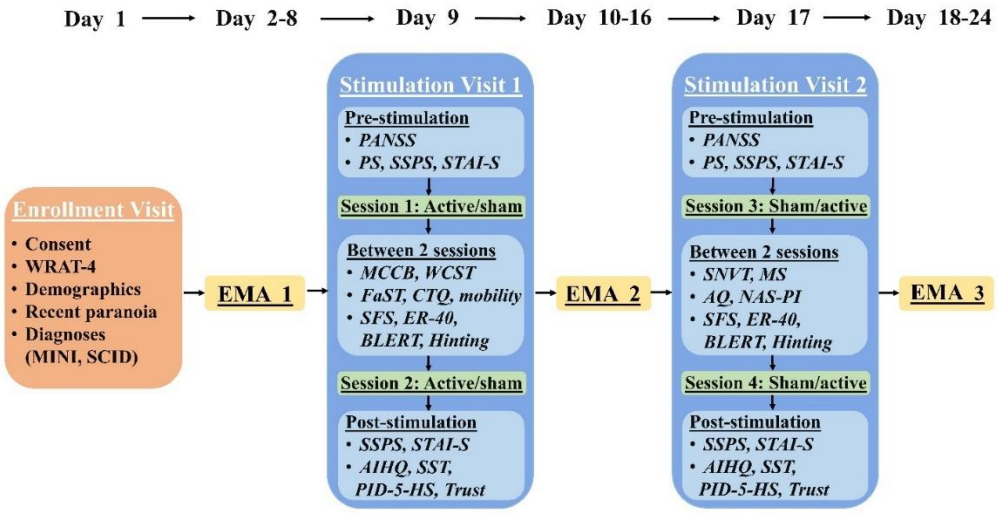


**Figure 1. Flowchart of the study procedure**

## 6. Stimulation Protocol

### *6.1 Stimulation Procedure*

After completing pre-stimulation assessments, participants started the first stimulation procedure of the visit via neuroConn's programable Direct Current stimulator. Electrodes were affixed to the participant's head to target the stimulation area as determined for each individual via the international 10-20 EEG placement system with Modified Combinatorial Nomenclature. For stimulating the right VLPFC, the anode was placed over F6 (Montreal Neurological Institute coordinates: 44, 32, -12 (Pinkham, Hopfinger, Ruparel, et al., 2008), and the contralateral homolog area (F5) was targeted for the stimulation of the left VLPFC (see **Figure 2** for 10-20 EEG placement). Electrodes were placed identically for the control condition. Following the neurostimulation procedure, participants were asked to remain seated in the chair and browse through magazines quietly for 30 minutes to allow stimulation effects to stabilize (Samani et al., 2019).

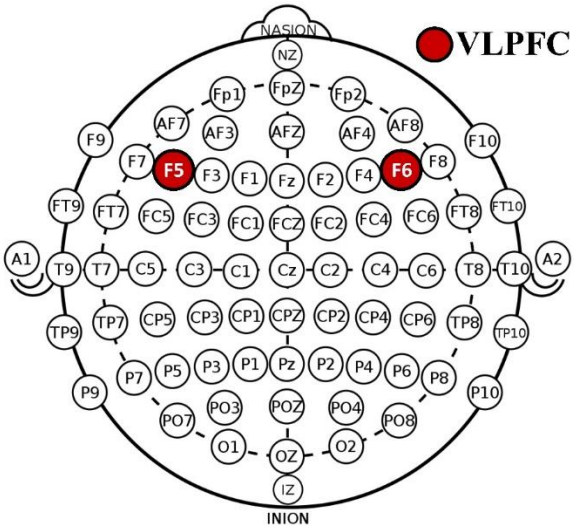


**Figure 2. 10-20 EEG placement**

*6.2 Counterbalance and Randomization*

The active stimulation and sham control conditions were counterbalanced, meaning that each participant had the same possibility of experiencing either the active or sham condition at the first visit, and the other condition at the second visit. In the current study, 26 participants received the active stimulation at their Visit 1, while the remaining 24 received it at their Visit 2. This counterbalance procedure was achieved via the "study mode" preprogrammed feature of the NeuroConn machine, which randomly assigned participants to either the active stimulation or sham group in a double-blinded way. Specifically, each participant received a 5-digit code, which was paired with one active stimulation code and one sham control code to ensure that each participant did not receive two stimulation or control conditions. After study completion, each participant's study code for that given visit was revealed and recorded. The project manager maintained the list containing the condition information corresponding to each visit during the study in case the blind conditions needed to be revealed in real time (e.g., if participants reported adverse effects during tDCS).

*6.3 Stimulation Parameters*

The total stimulation time of 40 minutes was divided into two twenty-minute sessions to allow repeated stimulation, which may increase efficacy (Cheng et al., 2020; Kim et al., 2019). The active stimulation included active current at 2mA, ramping up over 15 seconds, sustaining for 20 minutes, and finally ramping down for 15 seconds. The dimensions of each electrode were 5 × 7 centimeters in size (35 $cm^2$), which led to a current density of 0.5714 $mA/cm^2$. For the sham condition, electrodes were still attached for 20 minutes each session, but the actual stimulation time was limited to 45 seconds per session, with 15 seconds of gradual stimulation, leading up to 15 seconds of full stimulation, and finally 15 seconds of ramping down. This limited stimulation mimicked the ramp up phase of active stimulation, making it more difficult for participants to guess the condition. The machine also automatically performed impedance control checks throughout the neurostimulation to ensure that both the participant and the experimenter could not tell which condition was being administered. Following each session,

inquiries into the participants' perceptions of the suspected condition showed that their accuracy in identifying the correct condition was below chance levels (47%), supporting the efficacy of the blinding procedures.

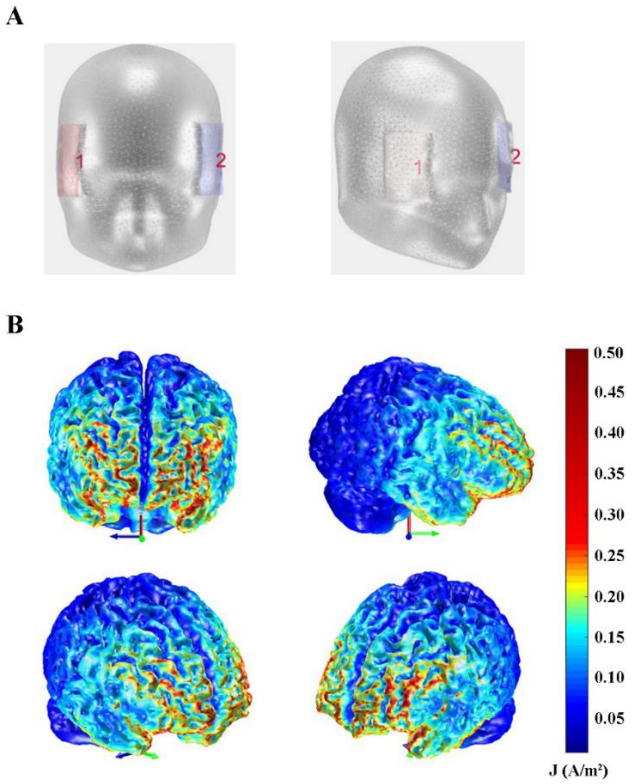


**Figure 3. Computational simulation of tDCS-induced current density. (A)** shows the montage location in the study (1 = right VLPFC, 2 = left VLPFC). **(B)** reveals the distribution of the current density estimated based on the stimulation location and parameters.

*6.4 Current Modeling*

To better characterize the current distribution during the stimulation, current modelling was conducted for the active condition. This computational simulation utilized a finite element model derived from MRI, implemented through the Comets toolbox (Jung et al., 2013) in Matlab (http://www.COMETStool.com). **Figure 3** illustrates a detailed three-dimensional (3D) numerical analysis of the current density generated by tDCS based on the montage applied in this study. It reveals that our targeted VLPFC is one of the regions receiving the most intense stimulation, along with the dorsal medial PFC and a portion of the DLPFC.

## 7. Assessments

*7.1 Enrollment Visit*

At the enrollment visit, consent was obtained from participants, and demographic information was also collected. Paranoia screening and diagnostic interviews were performed to determine eligibility. Current medications were reported by the patients, and they were asked to

remain on a stable dose of medication throughout their participation in the study unless specifically recommended by their physician. After that, participants completed daily EMA surveys for 7 days to assess baseline paranoia level and social functioning in daily life. They then attended the first stimulation visit. At both stimulation visits, the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1987) was administered by trained research personnel to assess the full range of symptoms in individuals with SSD. Daily EMA surveys between Stimulation Visit 1 and 2, as well as after Stimulation Visit 2, were also sent to participants to monitor any changes in paranoia and social functioning after tDCS in the participant's day-to-day life.

*7.2 Stimulation Visits: Pre-stimulation Assessments*

***Trait Paranoia.*** The Paranoia Scale (PS) (Fenigstein & Vanable, 1992) is a self-report measure to assess trait paranoia. This measure contains 20 items (e.g., "Someone has it in for me", "It is safer to trust no one") and is measured on a five-point Likert scale (1 = not at all applicable, 5 = extremely applicable). Scores range from 20-100, with higher scores indicating higher paranoia. The PS demonstrated good reliability and validity in previous work on patients with schizophrenia (Pinkham et al., 2012; Tiernan et al., 2014). In the current study, PS showed excellent internal consistency in both Stimulation Visit 1 (Cronbach's $\alpha = 0.941$) and Stimulation Visit 2 (Cronbach's $\alpha = 0.930$).

***State Paranoia.*** The State Social Paranoia Scale (SSPS) (Freeman et al., 2007) was used to examine paranoia levels immediately before and at the conclusion of stimulation within each visit. Participants indicated how much they agreed with each of 20 statements (e.g., "Someone was hostile towards me", "Someone was trying to isolate me") using a five-point Likert scale (1 = do not agree, 5 = totally agree). Additionally, the SSPS contains three subscales, measuring paranoid, neutral, and positive thinking. Scores range from 20-100, and higher scores represent higher state paranoid ideation. Acceptable reliability and validity have been demonstrated in previous research in patients with psychosis (Freeman et al., 2015; Veling et al., 2016). In the current study, the total scale (Visit 1: Cronbach's $\alpha_{pre} = 0.819$, Cronbach's $\alpha_{post} = 0.840$; Visit 2: Cronbach's $\alpha_{pre} = 0.798$, Cronbach's $\alpha_{post} = 0.880$) and the persecution subscale scale (Visit 1: Cronbach's $\alpha_{pre} = 0.924$, Cronbach's $\alpha_{post} = 0.926$; Visit 2: Cronbach's $\alpha_{pre} = 0.903$, Cronbach's $\alpha_{post} = 0.911$) showed outstanding internal consistency. The neutral thinking (Visit 1: Cronbach's $\alpha_{pre} = 0.750$, Cronbach's $\alpha_{post} = 0.457$; Visit 2: Cronbach's $\alpha_{pre} = 0.603$, Cronbach's $\alpha_{post} = 0.803$) and positive thinking (Visit 1: Cronbach's $\alpha_{pre} = 0.706$, Cronbach's $\alpha_{post} = 0.731$; Visit 2: Cronbach's $\alpha_{pre} = 0.611$, Cronbach's $\alpha_{post} = 0.853$) subscales showed mostly acceptable internal consistency.

***State Anxiety.*** Considering that anxiety has been consistently identified as a construct that overlaps with paranoia (Martin & Penn, 2001; Startup et al., 2007), we also measured state anxiety to examine potential changes after tDCS. The State subscale of State-Trait Anxiety Inventory (STAI-S) includes 20 items measuring current feelings (e.g., "I am tense", "I feel nervous") on a four-point Likert scale (1 = not at all, 4 = very much so; scores range 20-80) (Spielberger, 2010), with higher scores indicating higher anxiety at this moment. The STAI-S demonstrated excellent internal consistency in both visits (Visit 1: Cronbach's $\alpha_{pre} = 0.879$, Cronbach's $\alpha_{post} = 0.869$; Visit 2: Cronbach's $\alpha_{pre} = 0.847$, Cronbach's $\alpha_{post} = 0.908$).

*7.3 Assessments Between the Two Stimulation Sessions*

As noted, participants were asked to sit quietly for 30 minutes after each stimulation

session to enable the stimulation to work thoroughly throughout the neural networks. Next, they completed several assessments of basic cognitive function and social cognitive ability/bias that have been generally linked to psychotic symptoms and paranoia but that were not of primary importance and therefore not reported here. Relationships among paranoia, social decision-making, and aggression were also explored in this segment for secondary analyses. Since these analyses were not the main focus of the project, they are not included in the main text (details of all measures administered between stimulation sessions can be found in **Appendix A**). Participants were offered a 30-minute break before the second stimulation session of that visit. The only measure relevant to the main goal of this study is overall social functioning as detailed below:

***Birchwood Social Functioning Scale (SFS; Stimulation Visit 1 and 2).*** The SFS (Birchwood et al., 1990) measures 7 domains of social adjustment based on self-reports (range = 0-223, Cronbach's $\alpha_{visit\ 1} = 0.915$, Cronbach's $\alpha_{visit\ 2} = 0.897$), including social engagement/withdrawal (range = 0-15; Cronbach's $\alpha_{visit\ 1} = 0.422$, Cronbach's $\alpha_{visit\ 2} = 0.561$), interpersonal communication (range = 0-9; Cronbach's $\alpha_{visit\ 1} = 0.426$, Cronbach's $\alpha_{visit\ 2} = 0.269$), prosocial activities (range = 0-66; Cronbach's $\alpha_{visit\ 1} = 0.879$, Cronbach's $\alpha_{visit\ 2} = 0.815$), recreation activities (range = 0-45; Cronbach's $\alpha_{visit\ 1} = 0.684$, Cronbach's $\alpha_{visit\ 2} = 0.749$), independence-competence (range = 0-39; Cronbach's $\alpha_{visit\ 1} = 0.911$, Cronbach's $\alpha_{visit\ 2} = 0.934$), independence-performance (range = 0-39; Cronbach's $\alpha_{visit\ 1} = 0.915$, Cronbach's $\alpha_{visit\ 2} = 0.920$), and employment (single item; range = 0-10). A higher score on each subscale indicates better functioning in the respective social domain, while a higher overall score signifies better social functioning in general. The SFS was originally developed for schizophrenia patients and has been widely used to measure social functioning outcomes in this population (Birchwood et al., 1990; Burns & Patrick, 2007).

*6.7 Stimulation Visits: Post-stimulation Assessments*

***State Paranoia and State Anxiety.*** SSPS and STAI-S were administered as described in the pre-stimulation assessments.

***Ambiguous Intentions Hostility Questionnaire (AIHQ).*** The AIHQ is a widely used measure to assess hostile attributional style in ambiguous situations (Combs et al., 2007). It is selected as one of the primary outcome measures because hostile perception is a crucial component of paranoia (Combs et al., 2009; Zaytseva et al., 2013), and there is a robust, medium-to-large association between paranoia and hostile attributional bias measured by AIHQ (r fluctuates at around 0.3-0.5) (An et al., 2010; Combs et al., 2009; Klein et al., 2018; Langdon et al., 2013). Five ambiguous scenarios were presented to participants, and they gave reasons why each situation happened, indicated how much they thought that people did this to them on purpose, how angry they would be, how much they would blame others, and what they would do about it. Responses to the first and last questions were recorded verbatim for later coding, and responses to questions 2-4 were rated on Likert scales (1-6 for if the behavior was on purpose and 1-5 for how angry they were and how much they would blame others). These three Likert scale items were averaged to form the blaming attribution subscale of AIHQ, which showed

outstanding internal consistency in both visits (Cronbach's $\alpha_{visit\ 1} = 0.905$, Cronbach's $\alpha_{visit\ 2} = 0.901$). For the open-ended questions, hostility and aggression biases were rated by three independent, trained raters using a five-point Likert scale (1 = not hostile/aggressive at all, 5 = very hostile/aggressive). Inter-rater reliability was computed by ICC among all the raters, with ICC > .80 being acceptable (ICC = 0.892 in the present study). Thus, the AIHQ yielded three summary scores, the Blaming Score, the Hostility Bias Score, and the Aggression Bias Score.

***Scrambled-sentences task (SST):*** In the SST (Wenzlaff, 1993), participants reordered strings of scrambled words to create sentences of either paranoid or nonparanoid meanings by selecting 5 out of the 6 provided words. For example, participants could reorder the words from "Winner, am, born, I, loser, a" to "I am a born winner" (positive interpretation) or "I am a born loser" (negative interpretation). They were given 4 min to finish as many as possible. The total number of paranoid sentences was divided by the total number of completed sentences and then multiplied by 100 to generate a percentage bias score, with a greater percentage indicating a higher level of paranoid interpretation bias. Previous research has demonstrated moderate-to-strong correlations between SST scores and paranoia (r fluctuates at around 0.4-0.6) (Savulich et al., 2015, 2017).

***Hostility Scale of the Personality Inventory for DSM-5 (PID-5-HS):*** PID-5-HS (Krueger et al., 2012) contains 10 self-report items (e.g., "I snap at people when they do little things that irritate me") assessing pathological hostility on a 4-point scale (0 = very false or often false, 3 = very true or often true), with higher total scores indicating more hostility (range = 0-30). PID-5-HS showed a high correlation with AIHQ blame score (r = 0.47) in previous research (Buck et al., 2016). In the current study, the internal consistency of PID-5-HS was excellent (Cronbach's $\alpha_{visit\ 1} = 0.902$, Cronbach's $\alpha_{visit\ 2} = 0.913$).

***Trustworthiness Task.*** The trustworthiness task requires participants to indicate how much they trust each of 60 grayscale facial stimuli along a 7-point Likert scale (-3 = very untrustworthy, 3 = very trustworthy) (Adolphs et al., 1998). Total scores were calculated by averaging across responses and hence varied from -3 to +3, with higher values indicating a greater tendency to trust others. There is a small-to-medium association between trustworthiness scores and paranoid ideation (r fluctuates between -0.1 and -0.3) (Buck et al., 2016; Klein et al., 2018).

**8. Ecological Momentary Assessments**

EMA surveys were administered for a total of 21 days – 7 days prior to simulation visit 1, 7 days after stimulation visit 1, and 7 days after stimulation visit 2. Participants received 3 randomly timed surveys assessing current paranoid symptoms and social interactions each day. The surveys were sent in the morning, mid-day, and evening, with random initiation at time intervals $\geq 2$ hours. Participants were allowed to respond to the survey within 1 hour after the survey was sent. This protocol is in line with previous work and was demonstrated to yield ideal intra-day variation and compliance (Jones, Moore, Depp, et al., 2021; Jones, Moore, Pinkham, et al., 2021). Each survey queried the frequency of auditory hallucinations and paranoid ideation along a 7-point scale, as well as the emotions associated with and perceptions of social interactions since the last survey (e.g., did you interact with anyone since your last survey?). Affective states since the past alarm were rated on a 7-point scale using the following items: sad/depressed, excited, irritated, happy, belongingness, and burden to others. Expectations

regarding subsequent interpersonal situations were also rated on a 7-point scale (e.g., how much interest do you have in interacting with people for the rest of the day?). **Appendix B** contains the complete list of EMA questions (i.e., paranoia: Q1; social functioning: Q3, Q4, Q5, Q6, Q7, Q14, Q15). Adherence was monitored remotely via a secure platform, and participants who missed 3 consecutive surveys were contacted by research personnel to identify and resolve any issues. Compensation of the EMA periods was determined by the actual number of surveys completed to encourage adherence.

A total of 47 (out of 50) participants had full data across three EMA periods. One participant did not have a phone and chose not to participate in the EMA protocol. Two participants completed baseline EMA but lost their phones afterwards, and thus chose not to complete the two remaining EMA periods. EMA compliance in the current study was above 80% across the three periods (EMA1 = 82.74%, EMA2 = 83.38%, EMA3 = 80.04%).

**9. Statistical Analyses Plan**

For all tests, a conventional threshold of $\alpha = 0.05$ was used. While opting not to correct for multiple comparisons may permit alpha inflation, we did not want to risk missing any potential therapeutic effects in this treatment study by increasing the likelihood of Type II errors. Therefore, in line with a large body of existing literature, findings that passed the significance level of $\alpha = 0.05$ are presented.

**Hypothesis 1a:** To test the reduction in paranoia measured in the lab (active vs. sham stimulation), repeated-measures ANOVAs were performed for self-reported state paranoia measured both before and after the stimulation, while paired-samples t-tests were performed for paranoia-related outcomes measured only after stimulation (i.e., attributional bias and hostility, trust to strangers).

**Hypothesis 1b:** Multilevel modeling (MLM) with maximum likelihood robust estimation was used to test the differences in daily feelings of paranoia (i.e., Q1) across the three EMA periods (EMA-baseline vs. EMA-active vs. EMA-sham), with the condition of the EMA period being dummy-coded.

**Hypothesis 2a:** To test the improvement in social functioning measured in the lab (active vs. sham stimulation), paired-samples t-tests were performed for self-reported social functioning measured only after the stimulation.

**Hypothesis 2b:** MLM with maximum likelihood robust estimation was used to test the differences in daily social functioning (i.e., Q3, Q4, Q5, Q6, Q7, Q14, Q15) across the three EMA periods (EMA-baseline vs. EMA-active vs. EMA-sham), with the condition of the EMA period being dummy-coded.

Notably, one of the daily social functioning questions (i.e., Q3: Since the past alarm, who have you interacted with?) has a different score range. It is quantified as the frequency of social interactions, where a higher score indicates more interactions across a broader range of people categories (range = 0-5). In contrast, the remaining six items adhere to a score range of 1-7. Therefore, we performed MLM separately for Q3 (named as social interaction frequency) and for the average of items Q4, Q5, Q6, Q7, Q14, and Q15 (named as social interaction motivation and experience) for parsimony.

**10. Participant Payment**

Participants were compensated $20 for completing the enrollment visit, $100 for each of the two in-lab stimulation visits, and up to $16 for each of the 7-day EMA periods (three EMA periods in total), totaling a maximum of $268. Participants who enrolled in the study but were found ineligible during screening were still compensated $20. If participants stopped taking part in the study or were withdrawn by the research team, they received $5 for each 20 minutes completed during the lab visits. For example, if they completed 1 hour of the study, they were paid $15. Compensation for each EMA period was determined by the number of surveys completed, with $0.75 paid for each survey except for the final survey, which was worth $1 (to allow for an even amount of $16 per EMA period if all 21 surveys were completed). Parking on campus was provided for participants.

## 11. Risk and Benefits Assessments

### *11.1 Potential Risks*

It is possible that participants may feel tired, fatigued or anxious during the research procedure; however frequent breaks were built into the lab portion of the study to reduce this possibility. Additionally, participants were informed that they may take a break or terminate their participation at any time. The most common side effect of tDCS neurostimulation is skin redness just beneath the electrode. This effect usually wears off within one hour. Other potential risks associated with tDCS include:

1. Itching at the electrode location; typically lasting less than an hour;
2. A small percentage of participants may experience mild dizziness upon stimulation onset. After stimulation, this only lasts a few seconds and has no effect on balance;
3. Occasionally, temporary skin damage may occur beneath the electrode. This results in a week-long darkening of the skin. At the planned current, the size of these marks, should they occur, is only a few millimeters, posing a low risk;
4. After neurostimulation and the waiting period, participants may feel drowsy, but this effect dissipates within one hour.

Finally, whenever information is gathered, there is a potential risk of loss of confidentiality. This cannot be guaranteed, but every effort has been done to protect the privacy of participants' information. Participants were assigned a random number that was used to identify their data, and the file linking the number to the participants' email addresses and birthdates was stored separately from the data they provide. This file will be erased upon completion of the study. All data is stored in locked filing cabinets in secure offices at UTD, as well as on password-protected/encrypted computers at UTD, and only study personnel who have received extensive training in methods to protect confidentiality have access to the data.

### *11.2 Potential Benefits*

It is possible that neurostimulation may reduce paranoid ideation and/or improve social functioning for participants after active stimulation. However, the effects of tDCS are often temporary, and we do not anticipate that such a short-term stimulation will have a lasting effect. If this study is effective, subsequent research can investigate the ideal frequency and duration of neurostimulation to deliver direct, long-lasting benefits.

This project may lay the foundation for distributing large-scale, easy-to-deliver standardized treatments for patients. Additionally, this study's use of ecological momentary assessments (EMA) will facilitate a better understanding of how treatment effects can be

observed in, and impacted by, real-world social situations, thereby laying the groundwork for elucidating the duration of real-world improvements and developing potential long-term treatment gains for individuals with schizophrenia.

## APPENDIX A: SUPPLEMENTARY MEASURES

***MATRICS Consensus Cognitive Battery (MCCB; Stimulation Visit 1).*** The MCCB (Nuechterlein et al., 2008) was used to capture cognitive function, including processing speed (Trail Making Test: Part A, Animal Naming), working memory (Letter-Number Span), and verbal learning (Hopkin's Verbal Learning Test-Revised). Previous research has found impaired cognitive profiles in patients with schizophrenia (Kern et al., 2011).

***Wisconsin Card Sorting Test (WCST; Stimulation Visit 1).*** The WCST (Berry et al., 2015; Nelson, 1976) was used to assess executive function (e.g., planning, set changing capacity). In this task, 4 stimulus cards and 64 response cards were presented on the computer. Participants were asked to match each response card to one of the stimulus cards according to one of three possible criteria (i.e., color, shape, or number). Immediately following each sort, participants were informed if they were correct or incorrect. After 10 correct sorts, the sorting rule changed, and participants had to discover a new rule. A subsequent string of 10 correct responses triggered another rule change, and this continued until all 64 cards were sorted. The number of categories/criteria achieved, total errors, and perseverative errors (i.e., when the card is sorted according to a category concept that the participant was told was erroneous) were calculated. Previous research has found that more perseverative errors were made by schizophrenia patients with paranoid ideations compared to healthy controls (Berry et al., 2015).

***The Fast and Slow Thinking Questionnaire (FaST; Stimulation Visit 1).*** The FaST (Hardy et al., 2020) contains 10 items measuring fast and slow thinking biases when having paranoid thoughts on a 5-point scale (1 = not at all, 5 = totally; range = 10-50), with higher scores indicating higher proneness for fast or slow thinking. This measure was included because previous research has suggested the involvement of fast and slow thinking biases in paranoia in both clinical and general populations (Garety et al., 2021; Hardy et al., 2020).

***Childhood Trauma Questionnaire*** (***CTQ***; ***Stimulation Visit 1)***. Given that trauma has previously been associated with paranoia (Freeman et al., 2002; Freeman & Fowler, 2009), we used the CTQ Short Form (Bernstein et al., 2003) to assess childhood trauma and control for it. The CTQ Short Form contains 28 items (e.g., "People in my family said hurtful or insulting things to me", "I believed that I was physically abused") on a five-point scale (1 = never true, 5 = very often true). Scores range from 28-140, with higher scores indicating higher level of childhood trauma.

***Penn Emotion Recognition Task (ER-40; Stimulation Visit 1 and 2).*** The ER-40 was used to assess emotion recognition ability (Kohler et al., 2003). Forty color photographs of faces expressing one of 4 basic emotions (happiness, sadness, anger, fear) or a neutral expression were presented to the participants. After viewing each face, they were asked to choose the correct emotion. The dependent variable consists of the accuracy score ranging from 0 to 40, with a higher score indicating higher emotion recognition ability. Impaired ER-40 performance was observed in patients with schizophrenia (Butler et al., 2009), but inconclusive results were reported in studies on individuals with prominent paranoid ideation. Previous research found nonsignificant differences in ER-40 performance between paranoid and non-paranoid patients with schizophrenia (Pinkham, Harvey, et al., 2016); however, individuals with subclinical paranoia showed reduced accuracy in identifying negative emotions compared to individuals with low paranoia (Combs et al., 2013). Some studies also reported that paranoid patients are more likely to misinterpret neutral facial expressions as angry and threatening (Pinkham et al.,

2011; Tso et al., 2015).

***Bell Lysaker Emotion Recognition Test (BLERT; Stimulation Visit 1 and 2).*** The BLERT (Bell et al., 1997) measures the ability to recognize seven emotions: happiness, sadness, fear, disgust, surprise, anger, or no emotion. Twenty-one videos (each one lasts ~10 seconds) of a single male actor were presented, which include facial, verbal, paraverbal, and body movement information. Participants were asked to view these segments and identify the emotion shown. The total number of correct identifications was used to reflect performance (range = 0-21), with a higher score indicating better emotion recognition ability. The BLERT has good psychometric properties in both clinical and general populations (Pinkham, Penn, et al., 2016), and has demonstrated high sensitivity and specificity in differentiating individuals with varying degrees of social cognitive deficits (Hajdúk et al., 2018).

***Hinting task (Stimulation Visit 1 and 2).*** The Hinting task was used to assess the ability to infer the true intent of interpersonal conversations (Corcoran et al., 1995). Ten short passages were read aloud, each of which is a social interaction between two characters and ends with one of the characters providing a hint. Participants were asked to indicate what they think the character truly meant. Responses are scored in real-time, and if a correct response is not given after the first hint, a second hint is provided. Each item is thus scored as either 0 = incorrect response, 1= correct response after second hint, or 2 = correct response after first hint. Difficulty in mentalizing/Theory of Mind (ToM) as measured by the Hinting task has been linked to more auditory hallucinations in patients with psychosis and more paranoid-like experiences in non-psychotic first-degree relatives (Versmissen et al., 2008). Performance on the hinting task also moderated the relationship between paranoia and social functioning, as revealed by a study of 88 adult patients with SSD, which found a negative correlation between paranoia and social functioning in patients with hinting scores in the bottom 78$^{th}$ percentile, but such a correlation became nonsignificant for patients with hinting scores in the top 22$^{nd}$ percentile (Phalen et al., 2017).

***Residential mobility history (Stimulation Visit 1).*** Residential mobility is the frequency that an individual moves from one place to another (Oishi, 2010). Residential mobility has been found to be associated with a greater desire to expand one's social network (Oishi et al., 2013), but also with poorer emotional and behavioral outcomes (Jelleyman & Spencer, 2008) and lower well-being (Oishi & Schimmack, 2010). Therefore, to investigate the relationship between residential mobility and paranoia, we collected data on the history of residential mobility in this study. Participants were asked to indicate the number of times that they have moved from one city/town to another since birth, followed by a list of all cities/towns to which they have moved and their ages when they moved (Oishi, 2010).

***Social norm violation task (SNVT; Stimulation Visit 2).*** Recent studies investigating how paranoia affects social decision-making found that paranoia was associated with decreased cooperation and increased punishment towards partners in economic games (Raihani et al., 2021; Raihani & Bell, 2018). In this study, we measured social norm violation perception to test its role in paranoia and aggression. In this social norm violation task (Mu et al., 2015), participants rated the appropriateness of 48 norm-violating behaviors (e.g., “John is talking on his cell phone in the movie theater”) on a 7-point scale (1 = strongly inappropriate, 7 = strongly appropriate). All items were reverse-coded, and higher scores reflect being more critical towards the violation.

***The Moralism Scale (MS; Stimulation Visit 2).*** The MS (Janoff-Bulman et al., 2009)

was used to further measure the perception of prescriptive (i.e., did not do the good thing, for example, did not help others) and proscriptive (i.e., do the bad thing, for example, cheat or steal) moral violation in the current study. The MS contains 20 scenarios where the character is considering conducting a certain moral (violation) behavior or not (10 items for prescriptive morality and 10 for proscriptive morality). Participants indicated whether the character in each situation should or should not do the behavior on a 9-point scale (1 = feel very strongly he/she should not do, 9 = feel very strongly he/she should). Scores were calculated for prescriptive and proscriptive (will be reverse-coded) morality, respectively, with higher scores indicating a higher weight has been assigned to prescriptive or proscriptive morality.

***Aggression Questionnaire (AQ; Stimulation Visit 2).*** The AQ includes 29 items assessing aggressive behavior and tendencies across 4 dimensions: physical aggression, verbal aggression, anger, and hostility (Buss & Perry, 1992). It is rated on a 5-point scale ranging from 1 (extremely uncharacteristic of me) to 5 (extremely characteristic of me), with higher scores indicating higher levels of aggression. In previous research, a significant positive correlation was found between persecutory ideation and aggression reported via AQ in both individuals with SSD and the general community (van Dongen et al., 2011).

***Novaco Anger Scale and Provocation Inventory (NAS-PI; Stimulation Visit 2).*** The NAS-PI is another self-reported measure examining anger reactions in everyday life (Novaco, 2003). The first part of NAS-PI contains 48 items tapping in to 3 dimensions: cognitive, arousal, and behavioral. Participants rated each item along a 3-point scale (1 = never true, 3 = always true), with higher scores indicating higher levels of aggression tendency. The second part contains 25 items depicting provoking situations, and participants rated how angry each of them makes them feel on a 4-point scale (1 = not at all angry, 4 = very angry), with higher scores indicating higher anger disposition. Good psychometric properties have been demonstrated among individuals with psychotic disorders (Novaco, 2003), and previous research has found that NAS-PI was positively associated with the suspiciousness item of PANSS in individuals with psychotic disorders (Bucci et al., 2013)

## APPENDIX B: EMA QUESTIONS

**Q1. Since the past alarm, how much have you had thoughts that you really can't trust other people?**
(1 = not at all, 7 = very much)
**Q2. Since the past alarm, how much have you been bothered by voices?**
(1 = not at all, 7 = very much)
**Q3. Since the past alarm, who have you interacted with? Select all that apply.**
[haven't interacted with others; friends; spouse, partner, or other family; with roommates, co-workers, or others you know; with strangers; other people (please specify)]
**Q4. How much interest or motivation did you have interacting with others since the last alarm?**
(1 = not at all, 7 = very much)
**Q5. How much pleasure or enjoyment did you feel in these interactions?**
(1 = not at all, 7 = very much)
**Q6. How did you feel toward others in the interactions?**
(1 = on guard or threatened; 7 = trusting or warm)
**Q7. What do you think others were thinking about you?**
(1 = unlikeable or inferior; 7 = likeable or capable)
**Q8. Since the past alarm, how much have you felt sad or depressed?**
(1 = not at all, 7 = extremely)
**Q9. Since the past alarm, how much have you felt energized or excited?**
(1 = not at all, 7 = extremely)
**Q10. Since the past alarm, how much have you been feeling like you belong or fit with others in your life?**
(1 = not at all, 7 = extremely)
**Q11. Since the past alarm, how much have you felt irritated or upset?**
(1 = not at all, 7 = extremely)
**Q12. Since the past alarm, how much have you felt that you were a burden on others?**
(1 = not at all, 7 = extremely)
**Q13. Since the past alarm, how much have you felt happy?**
(1 = not at all, 7 = extremely)
**Q14. How much interest or motivation do you have in interacting with others later today?**
(1 = not at all, 7 = very much)
**Q15. How much do you want to avoid others later today?**
(1 = not at all, 7 = very much)

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