"The Effects of Transcranial Direct Current Stimulation (tDCS) on Auditory False Perceptions in Healthy Participants"

Tine Tronrud

MAPSYK360

MASTEROPPGAVE I ATFERD OG NEVROVITENSKAP

UNIVERSITETET I BERGEN

INSTITUTT FOR BIOLOGISK OG MEDISINSK PSYKOLOGI

VÅR 2022

Antall ord: 17083

Veileder: Marco Hirnstein, Department of Biological and Medical Psychology

Abstract

Auditory verbal hallucinations (AVH) have been associated with hyperactivity in the left temporoparietal cortex (TPC) and hypoactivity of the left dorsolateral prefrontal cortex (DLPFC). As a consequence, it was suggested that transcranial Direct Current Stimulation (tDCS), with the supposedly excitatory anode and the supposedly inhibitory cathode placed over the left DLPFC and left TPC, respectively, could alleviate AVH. However, despite promising findings, the results of this treatment are inconsistent. The aim of this study was to test if reversing the electrode montage in healthy participants would lead to an inverted effect, that is increases in auditory false perceptions (H_1). This would verify the neurocognitive theoretical foundation of the tDCS treatment of AVH. Moreover, we tested whether more false perceptions could be triggered when participants expected certain words (top-down effect) and when these words were embedded in human noise (bottom-up effect). Seventy-six participants were tested twice, once with real and once with sham tDCS. Thirty-nine participants got the electrode placement used in treatments of AVH in clinical patients, whereas thirty-seven got the reversed electrode set-up. The effect of tDCS was measured with a signal detection task (SDT) in each session. We failed to find any effect of electrode montage and the expected top-down/bottom-up interaction. Possible explanations for the lack of findings, including that tDCS is not targeting their intended neuronal networks, are presented.

Key words: transcranial Direct Current Simulation, tDCS, auditory verbal hallucinations, signal detection task, SDT, temporoparietal cortex, TPC, dorsolateral prefrontal cortex, DLPFC, auditory false perceptions, source monitoring, executive functions, inhibitory control

Sammendrag

Auditive verbale hallusinasjoner (AVH) har vært assosiert med hyperaktivitet i venstre temporoparietal cortex (TPC) og hypoaktivitet i venstre dorsolateral prefrontal cortex (DLPFC). Basert på disse funnene har det blitt foreslått at transkraniell direkte strømstimulering (tDCS), med den antatt eksitatoriske anoden og den antatte hemmende katoden plassert henholdsvis over venstre DLPFC og venstre TPC, kan lindre AVH. Til tross for lovende funn er imidlertid resultatene av denne behandlingen inkonsekvente. Målet med dette studie var å teste om en reversering av denne elektrode plasseringen hos friske deltakere ville føre til en omvendt effekt, det vil si økninger i auditive falske oppfatninger (H_1). Dette vil kunne bekrefte det teoretiske grunnlaget for tDCS-behandlingen av AVH. I tillegg testet vi om flere auditive falske oppfatninger kunne utløses når deltakerne forventet visse ord (top-down-effekt) og når disse ordene var innebygd i menneskelig støy (bottom-up-effekt). Syttiseks deltakere ble testet to ganger, en gang med ekte og en gang med falsk tDCS. Trettini deltakere fikk elektrodeplasseringen som er brukt i behandlinger av AVH hos kliniske pasienter, mens trettisyv fikk omvendt elektrode plassering. Effekten av tDCS ble målt med en signaldeteksjonsoppgave (SDT) under hver økt. Vi klarte ikke å finne noen effekt av elektrodeplassering eller den forventete top-down/bottom-up interaksjonen. Mulige forklaringer på manglende funn, som at tDCS muligens ikke retter seg mot tiltenkte nevrale nettverk, presenteres.

Nøkkelord: transkraniell direkte strømstimulering, tDCS, auditive verbale hallusinasjoner, AVH, signaldeteksjonsoppgave, SDT, temporoparietal cortex, TPC, dorsolateral prefrontal cortex, DLPFC, auditive falske oppfatninger, eksekutive funksjoner

Acknowledgements

The topic of this thesis was driven by my passion for the neuroscientific field of brain stimulation and the opportunity to investigate the neuronal mechanism that are involved in auditory verbal hallucinations. First and foremost, I would like to express my gratitude to my supervisor Marco for giving me the patience and confidence to complete this thesis. His guidance and counsel gave me both an increased understanding of the tDCS method and topic at hand. Thank you for your constructive feedback, excellent encouragement, and trust during this entire project. I would also like to extend my gratitude to Julien, who together with Marco made this project possible. Further, I would like to express an appreciation to my fellow brain enthusiasts Emika, Tina and Siri whose motivational words and positive spirits made the completion of this thesis possible. A special thanks to my partner Simen for being my everyday support system, and to my brother Kristoffer for dragging me out when breaks were desperately needed. Lastly, to my mum for being an incredible source of inspiration, always.

CONTENTS

1. Introduction

Hearing a voice in the absence of a speaker is perhaps one of the most fascinating and disconcerting experiences there is. These occurrences are often described by a broad range of individuals ranging from psychiatric/neurological patients to healthy persons (Jardri et al., 2013). For some, these voices come across as pleasant and enthralling, entertaining and even amusing. These individuals can find themselves being captivated and engaged in endless conversations and fascinating ideas (Jardri et al., 2013). However, just as easily can these voices become brutal, humiliating, criticizing, and disconcerting. Patients with schizophrenia often describe them as negative internal narrators that meddle in different aspects of their lives (Jardri et al., 2013). In some cases, the voices come in the form of commands that can occasionally elicit violent acts towards others or themselves (Bjorkly & Grondahl, 2016; Fujita et al., 2015). The fact that these voices remain imperceptible to others therefore creates several fundamental issues for both the individual experiencing them and society. Understanding the neuronal mechanisms that gives rises to these experiences are thus of critical importance. Much research has focused on brain imaging and brain stimulations methods in patients with schizophrenia to investigate the specific brain regions that contribute to the hallucinatory experiences of hearing voices (Brunelin et al., 2012; Kompus et al., 2011). However, this study wants to take it a step further by attempting to alter the specific neuronal mechanism in healthy participant instead, with the aim to provide some complementary empirical evidence for the specific mechanism involved.

1.1 Schizophrenia

Schizophrenia is a neuropsychiatric disorder that affects millions of individuals worldwide (nearly 1% of the world's population; McGrath et al., 2008). The disorder usually

presents itself in adolescence and young adults, and is one of the top 25 causes of disability in the world (Whiteford et al., 2013). Individuals with schizophrenia often display different dysfunction in their clinical presentation, course, and prognosis, with the disorder beginning suddenly for some and gradually for others (Tandon et al., 2008). At the same time, they tend to show an immense variation in their symptoms with some experiencing stable symptoms, and others a more varied manifestation (Tandon et al., 2008). The disorder is therefore considered to be very heterogenous making it difficult to define (Joyce & Roiser, 2007). As a result, researchers tend to focus on specific symptoms rather than the entire disorder itself (Hugdahl & Sommer, 2018). One typically differentiates between negative and positive symptoms. Whereas negative symptoms are characterized by an absence or a deficit in a normal mental function (e.g., lack of joy, reduced motivation, withdrawal and little apathy or indifference to oneself and others), positive symptoms are characterized as an adjustment to or a distorted form of a normal mental function (e.g., hallucinations, delusions, or catatonic behaviour; Tandon et al., 2008). These positive symptoms often refer to a perception that does not correspond to reality where, for instance, patients may hear a voice or sound, experience a smell, taste, or touch, see lights or objects that do not exist (Kolb et al., 2019, pp. 592-594). These symptoms will therefore often result in long-term psychiatric and humanistic problems for the patients that generally leads to a reduced quality of life (Chong et al., 2016; Millier et al., 2014).

Patients with schizophrenia usually require a substantial amount of healthcare that can impose a significant economic burden for both the patient, their families, and society as a whole (Fasseeh et al., 2017). In Norway, mental health disorders are estimated to cost around 31.7 billion NOK yearly in health care expenses alone (Kinge et al., 2017). Out of these, there are approximately 10,000 patients that are being treated for schizophrenia at any given time (Norsk helseinformatikk, 2021, 19 April). Schizophrenia patients are often a

marginalized part of the work environment (Biggs et al., 2010; Bricout & Bentley, 2000; Thornicroft et al., 2009) with approximately 94% being unemployed (Melle et al., 2000). The patients' psychotic symptoms or alternations in behaviour can often lead to more frequent relapses, readmissions, and increases in both anxiety and depression (Gould et al., 2001). These can all be challenging factors when it comes to functioning in an ordinary work environment. In Norway, 10% of those who receive disability benefits are diagnosed with schizophrenia (Norsk helseinformatikk, 2021, 19 April). A Norwegian study estimated that the total annual direct (mental health services), and indirect (productivity and social security) cost of 8399 individuals with schizophrenia would be around 7.9 billion NOK (Evensen et al., 2016). This is substantially larger than other highly costly diseases to treat, such as cancer and cardiovascular disease (Johannessen, 2002).

The International Classification of Diseases, Tenth Revision (ICD- 10) contains certain criteria and indicators that are needed for an individual to become diagnosed with schizophrenia. The patient must display at least two out of the following five symptoms of delusions, hallucinations, disorganized speech, disorganized/catatonic behaviour, or a negative symptom. These symptoms must also last for at least one month, where one of the symptoms must be either a delusion, hallucination, or disorganized speech (World Health Organization, 2019). Most diagnoses of schizophrenia are usually made during an active or acute phase where the patients display one or more positive symptoms (World Health Organization, 2019). At this stage, the medical attention will consist of a prompt treatment that aims to reduce or diminish the severity of these schizophrenia manifestations (Yang et al., 2021). The treatment often consists of a group of medications called antipsychotics or neuroleptics that is tailored to the patient on a trial-and-error basis (Marquardt et al., 2021). These medications are usually grouped into first (typical) or second (atypical) generation drugs (Marquardt et al., 2021). Most antipsychotics target dopamine receptors to a larger

(typical) or smaller (atypical) degree. However, atypical antipsychotics are more often preferred today due to less movement-related, and adverse side-effects (Geddes et al., 2000).

Even though some atypical antipsychotics are considered as a better alternative, they have their own limitations that can produce other severe metabolic adverse effects, such as obesity, diabetes, hyperglycaemia, dyslipidaemia and gynecomastia (Stępnicki et al., 2018). Another major problem with antipsychotic treatment is that there seems to be a vast difference in how patients with schizophrenia respond to them (Yang et al., 2021). For instance, many seem to be resistant to any sort of antipsychotic treatment (Yang et al., 2021). This means that their symptoms do not improve despite continuous medication (Kane et al., 2019; Kane & Correll, 2016). Auditory verbal hallucinations (AVH) are a common psychotic symptom in patients with schizophrenia (Andreasen & Flaum, 1991; Hugdahl et al., 2008; Pondé et al., 2017) and studies have shown that approximately 25-30% of these patients are resistant to any conventional antipsychotic medication (Demjaha et al., 2017; Shergill et al., 1998).

1.2 Auditory Verbal Hallucinations (AVH)

Hallucinations can occur in many different sensory modalities that can again be associated with many different types of disorders (Jardri et al., 2013, pp. 140-141). However, they do not seem to be distinct to these disorders. For instance, AVH can occur across many different types of psychiatric disorders such as in bipolar disorder, post-traumatic stress disorder, depression, delirium, borderline personality disorder, and in various types of dementia (Bohlken et al., 2017). In addition, AVH are not only related to psychiatric illnesses or illness in general, but are also found amongst the general population (5-15%; Vercammen & Aleman, 2010). It is therefore difficult to state that AVH is a characteristic symptom of schizophrenia, but perhaps one of the most common symptoms with approximately 60-80% of patients reporting them (Andreasen & Flaum, 1991; Hugdahl et al., 2008; Pondé et al.,

2017). AVH are experienced in similar ways as actual spoken sentences that can contain different frequencies of words, sentences, dialogues, and number of voices (from one to several; Daalman, Boks, et al., 2011). This often makes it difficult for patients to distinguish between what is real and what is their own imagination (Laroi, 2012).

It can be difficult to discriminate AVH in patients with schizophrenia and other psychiatric disorders (e.g., between borderline personality disorder and schizophrenia; Jardri et al., 2013, p. 146; Yee et al., 2005), although, some differences were reported. For instance, AVH in mood disorders are often more mood congruent, with depressive episodes eliciting more disparaging or nihilistic AVH content (Jardri et al., 2013, p. 146). Likewise, healthy-voice hearers often experience a higher level of control over their voices compared to AVH patients with schizophrenia (Jardri et al., 2013, pp. 146-147; Laroi, 2012). Healthy-voice hearers also tend to show a higher capability in correctly identifying if the voices are originating from their inner thoughts or an external source (Daalman, Boks, et al., 2011). In turn, schizophrenia patients tend to misattribute their inner speech as coming from an external source (Daalman, Boks, et al., 2011). Moreover, AVH experienced by schizophrenia patients tend to be more negative and insulting, as opposed to those experienced by healthy-voice hearers (Daalman, Boks, et al., 2011). The negative content of these voices can occur in the form criticism, comments, or commands (McCarthy-Jones et al., 2014) that are again associated with an increased risk of both violence towards others (Bjorkly & Grondahl, 2016) and self-harm (Fujita et al., 2015). In fact, suicide seems to be one of the largest contributor to the shorter life expectancy in schizophrenia patient with AVH (Sher & Kahn, 2019). Despite these differences, there are also some similarities between healthy-voice hearers and patients with schizophrenia in terms of the number of voices experienced (from one to several), the frequency of these voices, the volume and the degree of personification (i.e., determining if the voice is coming from a real/unreal or familiar/unfamiliar person; Daalman,

Boks, et al., 2011). In addition, functional magnetic resonance imaging (fMRI) studies show similar activations patterns in clinical and non-clinical AVHs (Diederen et al., 2012).

1.3 Neuronal Mechanisms of AVH

The neuronal basis of AVH has been related to several brain regions, and efforts to explain the underlying brain mechanism remain relatively elusive. However, the most robust and replicated findings in neuroimaging studies show spontaneous activation in the left hemispheric peri-Sylvian region (Jardri et al., 2011; Kompus et al., 2011; Kühn & Gallinat, 2012). The peri-Sylvian region consists of areas around the lateral sulcus (Sylvian fissure) that contains the classical speech perception areas: the primary auditory cortex (Heschl's gyrus) and Wernicke's area (planum temporale). Collectively, these areas are referred to here as the temporoparietal cortex (TPC; Hoffman et al., 2013; Moseley et al., 2014). fMRI studies on healthy individuals have shown that a variety of auditory stimuli can activate the TPC area, suggesting that it plays a role in auditory processing and speech comprehension (Allen et al., 2007). This activation is also stronger in the left hemisphere when it comes to speech sounds, as opposed to non-speech sounds (Binder et al., 2000). AVH patients, however, will often display aberrant activity in the TPC area. For instance, there seems to be an increased activity in the TPC area when healthy individuals listened to external voices, whereas patients with schizophrenia showed a similar activation during AVH in this area (Kompus et al., 2011). This suggests that the neuronal basis of AVH (i.e., hearing voices in the absence of any external stimuli) seems to be like the activations healthy individuals have when perceiving an actual, external speech sound. Moreover, healthy individuals showed decreased activity in the TPC area when generating internal voices (Allen et al., 2007; Simons et al., 2010), whereas schizophrenia patients tended to show increased activity (i.e., hyperactivity) in the TPC region

during AVH (Kompus et al., 2011). Thus, compared to healthy individuals, schizophrenia patients tend to elicit a hyperactivity in the TPC region during AVH.

A second brain area that shows aberrant neuronal activity during AVH in patients with schizophrenia is the left dorsolateral prefrontal cortex (DLPFC). The DLPFC is often associated with several executive function processes. Executive functions allow one to control certain processes including but not limited to: directing or shifting attention, maintaining, and manipulating information, working memory, inhibit unimportant and distracting information, execute goal-directed behaviour and control information (Bowie & Harvey, 2006). Compared to healthy controls, there is decreased activity in this region in patients with schizophrenia, especially in the left hemisphere (Hugdahl, 2015; Lawrie et al., 2002). This hypoactivity has also been shown in schizophrenia patients during AVH (Brunelin et al., 2012; Hugdahl, 2015; Koops et al., 2015). Collectively, these studies suggest that hyperactivity in the left TPC leads to the production of AVH, whereas hypoactivity of the left DLPFC leads to a diminished executive control over the AVH.

1.4 Deficits in Source Monitoring and Inhibitory Control

A common human ability is to determine whether one is hearing a voice or imagining a voice (e.g., thought), and identifying the source a voice is coming from. This ability is an important everyday process that helps individuals differentiate between internally generated information and externally driven information (Mondino et al., 2014). This act is often referred to as source monitoring and research suggests that this process is aided by the mechanism of corollary discharge (Nawani et al., 2014). In healthy individual, for instance, the left TPC area will become activated during external auditory verbal stimuli. However, during inner speech corollary discharge is generated to dampen the activity in the TPC region. This way, the mechanism of corollary discharge signals that the information is internally driven rather than

coming from an external source (Parlikar et al., 2019). Functional neuroimaging studies support this notion by showing that there is an activity increase in the TPC region when healthy participants listen to external voices, as opposed to an activity decrease when they generate internal voices (Allen et al., 2007; Simons et al., 2010).

Patients with schizophrenia, on the other hand, showed increased activation in the TPC during AVH (Lennox et al., 2000; Shergill et al., 1998). Theories have therefore emerged suggesting that AVH occur due to a dysfunction in source monitoring where the corollary discharge fails to dampen the activity in the TPC region. This will then result in AVH patients having difficulties distinguishing where the voice is coming from. Support for this notion comes from studies demonstrating that AVH patients with schizophrenia tend to state that their imagined words and sentences are coming from an external source, even when they were generated from their own thoughts (Bohlken et al., 2017; Hugdahl, 2015). Studies have also demonstrated that healthy individuals and non-AVH patients with schizophrenia are less likely to mistake internal events for an external source, when compared to AVH patients with schizophrenia (Brunelin et al., 2006; Stephane et al., 2010). However, these deficits in source monitoring have been reported amongst healthy individuals as well. Individuals without a diagnosis often report different levels of AVH proneness. Those who reported positive beliefs about their unusual perceptual experiences are reported as having a higher predisposition to AVH as opposed to those with a negative belief about these experiences (Larøi et al., 2004; Morrison et al., 2000). The high proneness individuals demonstrated rather different meta cognitive-beliefs and thought control strategies, as opposed to those with a low proneness to AVH (Morrison et al., 2000). Further, studies reported a deficit in source monitoring amongst those that have a high proneness for AVH, when compared to those with a low proneness (Garrison et al., 2017). Taken together, the above findings indicate that deficits in source monitoring is specific to AVH.

It has been suggested that the decreased activity in the left DLPFC outlined above (Hugdahl, 2015; Lawrie et al., 2002) might indicate an insufficient capability of exerting top-down control over the perceptual areas in the absence of any external stimulus (Hugdahl, 2009). In healthy individuals, for instance, the left DLPFC is suggested to function as a top-down mechanism that attributes internal voices as coming form their own thought. In other words, the DLPFC dampens the responsiveness in the TPC area during inner speech, and thus "alerts" the auditory sensory regions that the incoming speech is self-generated (Hugdahl, 2009). Theories have suggested that this insufficient top-down mechanism in schizophrenia patients manifests as a lack of inhibitory control (Waters et al., 2012). Inhibitory control allows one to disregard irrelevant information and also to set aside previously activated cognitive content (Waters et al., 2006). This enables healthy individuals to distinguish internal thoughts from voices that stem from external sources (Hugdahl, 2009). The relationship between inhibitory control and AVH has been linked to executive resources in the prefrontal cortex (Badcock & Hugdahl, 2012). Specifically, poor inhibitory control has been associated with AVH alone, without other positive or negative symptoms (Waters et al., 2012; Waters et al., 2006; Waters et al., 2003). Several studies have also shown that this poor inhibitory control mechanism relates to the prevalence and frequency of AVH (Michie et al., 2005; Soriano et al., 2009). In addition, these deficits are found in AVH patients with schizophrenia (Michie et al., 2005; Soriano et al., 2009), healthy voice-hearers, and those with a high predisposition for AVHs (Paulik et al., 2007). It was therefore suggested that reductions in inhibitory control contributes to the tendency of misattributing internal events to an external source (Daalman, van Zandvoort, et al., 2011).

However, if both schizophrenia patients and healthy voice hearers exhibit deficits in inhibitory control, why do their AVH experiences differ with respect to level of control and perceptual nature? As mentioned before, non-clinical voice hearers often exert a higher level

of control with less negative and/or intrusive voices as compared to clinical AVH patients (Daalman, Boks, et al., 2011; McCarthy-Jones et al., 2014). Research suggests that the differences can be explained due to a more subjective sense of control or the source of the intrusion itself (Daalman, Boks, et al., 2011). For instance, the intrusion is more related to an emotional dysregulation in healthy, hallucination-prone individuals, whilst it is more closely related to impaired memory in patients with schizophrenia (Badcock & Hugdahl, 2012; Daalman, van Zandvoort, et al., 2011; de Leede-Smith & Barkus, 2013; Paulik et al., 2007). Studies often showed similar brain activations in clinical and non-clinical AVH groups (Diederen et al., 2012). As mentioned before, AVH in both clinical and non-clinical groups is suggested to arise due to a hyperactivity of the left TPC region (i.e., bottom-up process) and a hypoactivity of the left DLPFC (i.e., top-down processes; Waters et al., 2012). However, in general, perception is not seen as a passive process where individuals only take in information from the external environment. Rather, perception is considered to be reconstructive were certain cognitive factors, such as prior knowledge, experiences, goals, and/or emotions, influence the experience of the stimuli (Kveraga et al., 2007). Differences in expectations, beliefs, insight, and hypervigilance between clinical and non-clinical AVH individuals might therefore contribute to the subjective and generally different experiences of these AVH.

1.5 Signal Detection Theory and the Neurocognitive Theoretical Model of AVH

Several studies employed signal detection tasks (SDT) with auditory stimuli to examine the underlying behavioural and neuronal mechanisms of AVH. Signal detection theory in general deals with the investigation and prediction into the ability to distinguish patterns, signals or stimuli from noise in uncertain environments (Bentall & Slade, 1985; Brookwell et al., 2013). In one of these SDTs, participants are asked to listen to recordings of white noise, in which sometimes a barely audible word is embedded in the noise (Barkus et al., 2011; Moseley et al.,

2014). Participants are then asked to indicate if they heard a word in the noise or not. The test has four possible outcomes: If the word is present, one could either identify it (hit) or not (miss). If there is no word present but only noise, one could either correctly state so (correct rejection) or perceive a word that is not there (false alarm). The latter response is used to establish the frequency of auditory false perceptions (Moseley et al., 2014). Paradigms based on signal detection theory allow for differentiation between the ability to discriminate the presence versus absence of a signal (i.e., "perceptual sensitivity") and the tendency to guess whether a signal is present or not (i.e., "response bias"; Bentall & Slade, 1985; Brookwell et al., 2013). In the context of auditory false perceptions, many studies reported that individuals with AVH are not necessarily worse at discriminating whether a word is present or not, but they exhibit a stronger liberal response bias. That is, they are more likely to indicate that they heard a word even when no word was presented (Alganami et al., 2017; Barkus et al., 2011; Bentall & Slade, 1985; Brookwell et al., 2013; Laloyaux et al., 2019).

However, there are other studies that report no such relation between AVH and response bias (Chhabra et al., 2016; Daalman et al., 2012; Hoskin et al., 2014). Moreover, some studies show that AVH patients have poorer perceptual sensitivity as compared to non-hallucinating patients (Chhabra et al., 2016; Li et al., 2002). A recent study used the SDT on a large sample of healthy participants and reported both a positive correlation between false alarm rate and the tendency to experience AVH, as well as a negative correlation between the response bias and the tendency to experience AVH (Moseley et al., 2021). Laloyaux et al. (2022) suggested that these inconsistent results might be due to the fact that many studies examine either top-down or bottom-up effects, while the neurocognitive theoretical model of AVH postulates that AVH arise from an interaction of top-down and bottom-up effects (Waters et al., 2012).

The neurocognitive theoretical model of AVH suggests that at bottom-up level, the hyperactivity of the auditory network can be triggered by external conditions, such as a noisy

environment (Waters et al., 2012). Most studies that employed SDT paradigms used white noise stimuli (i.e., non-human sound), that consists of a mixture of sound waves ranging between 1 Hz to 20 kHz (Laloyaux et al., 2019; Laloyaux et al., 2022). According to Laloyaux et al. (2022), there was no specific theory or rationale why non-human sound stimuli were used. Since humans tend to be more sensitive to frequencies ranging from 2-4 kHz, with the best sensitivity around 3 kHz (Laloyaux et al., 2022; Quam et al., 2017), Laloyaux et al. (2022) suggested that stimuli comprising those frequencies would be better suited for SDT paradigms.

Waters et al. (2012) neurocognitive theoretical model of AVH also emphasises that at top-down level, the hyperactivity in the TPC can be triggered by various types of cognitive and emotional factors (e.g., low cognitive control, response bias, high expectations, and increased hypervigilance; Laloyaux et al., 2022; Waters et al., 2012). Different top-down factors were identified to modulate the response bias in the auditory SDT (Laloyaux et al., 2022). For instance, when a sentence is presented, whose last word is embedded in noise, semantic expectations for that last word can be high (e.g., "the sky is… blue") or low (e.g., "the best would be…marriage"). High expectation words correlate with both better performances on the task, as well as a significantly higher response bias in individuals prone to AVH (Alganami et al., 2017; Daalman et al., 2012; Hoskin et al., 2014; Laloyaux et al., 2022; Vercammen & Aleman, 2010).

Due to the relevance of both top-down and bottom-up processes for AVH (Waters et al., 2012), Laloyaux et al. (2022) created a novel SDT paradigm that included two different types of noises (non-human versus human frequency sounds = bottom-up process) and two levels of expectation (high versus low = top-down process). They compared individuals with high proneness to AVH to those with low proneness to AVH and found a stronger liberal response bias in those more prone to AVH when both the masking noise mimicked human speech and when there was a high-level expectation of the targeted words (Laloyaux et al., 2022). At the

same time, the study reported no significant difference between the two groups when there was a low expectation of words or if these words were masked by a noise at a non-human frequency. Based on these results, Laloyaux et al. (2022) concluded that speech-like frequencies could trigger the hyperactivation in auditory networks, whilst high semantic expectation increases the likelihood of detecting these signals.

Considering its potential assets, this current study wanted to employ a SDT that incorporates the suggestions made by Laloyaux et al. (2022) study. Much research has focused on brain imaging methods to provide a further insight into the physiology of AVH (Allen et al., 2007; Hugdahl, 2015; Jardri et al., 2011; Kompus et al., 2011; Kühn & Gallinat, 2012; Lawrie et al., 2002). However, this study wanted to use a brain stimulation method instead to provide some complementary empirical evidence on the neuronal mechanisms involved in AVH. Neuroimaging studies are correlational in nature and do not allow drawing conclusions about the causality between certain brain activations and behaviour (Purves & Brannon, 2013). One could thus argue that neuroimaging methods do not provide a complete account for the different brain mechanism involved. On the other hand, brain stimulation techniques can look at how specific brain regions contribute causally to specific cognitive processes by altering brain functions (Woods et al., 2015).

1.6 Transcranial Direct Current Stimulation (tDCS)

tDCS is a non-invasive brain stimulation method that induces an electrical current via electrodes to modulate activity in the brain (Nitsche et al., 2008). Whereas other brain stimulation methods, such as transcranial magnetic stimulation, trigger an action potential in specific cortical areas, tDCS rather modulates its threshold by increasing (depolarizing) or decreasing (hyperpolarizing) neural membrane potentials (Nitsche et al., 2008). tDCS is applied directly on the scalp and provides a constant electric current with low amplitude. The

current is meant to influence or disrupt the neuronal processes in the specific targeted brain areas (Nitsche et al., 2008). One electrode is positively charged (anode), which increases cortical voltages, whilst the other is negatively charged (cathode; Nitsche & Paulus, 2000; Nitsche et al., 2008; Woods et al., 2015). At first, it was believed that the anode and cathode have an excitatory and inhibitory effect on the cortex, respectively, but it has become abundantly clear in the meantime that this is an oversimplification, as the anode has been shown to both excite and inhibit the neuronal processes in various studies (Boggio et al., 2009; Civile et al., 2021; Elmer et al., 2009; Fregni et al., 2005; Nitsche et al., 2012; Ohn et al., 2008; Plewnia et al., 2013). In any case, the tDCS current generally goes from the anode to cathode and continues back to the anode to complete a circuit. The aim of this method is to induce changes in brain activity that last longer than the stimulation period itself (Nitsche et al., 2008). This goal seems to be determined by the strength of the current, the duration of stimulation, and both the size and placements of the electrodes (Nitsche et al., 2008; Woods et al., 2015).

The effect of the stimulation is usually seen by comparing it to a sham or control state, where the power is turned off or not delivered. Compared to other neurostimulation methods, tDCS is considered to be a more tolerable, easier to use, and a relatively cheaper method (Nieuwdorp et al., 2015). The most common side effect reported are usually itching and/or tingling sensation directly underneath the electrodes (Koops et al., 2015). Some studies have referred to headaches, skin irritations, nausea, and/or fatigue. However, these often occur in stimulation procedures that last longer than 20 minutes, or when the current is relatively high (i.e., > 2 mA; Antal et al., 2017).

1.7 tDCS in AVH Patients

tDCS has been suggested as an effective alternative treatment option for AVH patients with schizophrenia, who do not seem to benefit from antidepressant medications or other therapies (Mondino, Jardri, et al., 2016). As previously mentioned, a persistent finding in the neuronal basis for AVH is hyperactivity in the left TPC (Allen et al., 2007; Kompus et al., 2011) and a hypoactivity in the left DLPFC (Brunelin et al., 2012; Hugdahl, 2015; Koops et al., 2015; see Figure 1 A). As a result, several studies aimed to simultaneously administer tDCS to both the left TPC and the left DLPFC (Brunelin et al., 2012; Koops et al., 2015; Mondino et al., 2014; Nawani et al., 2014; Nieuwdorp et al., 2015; Shiozawa et al., 2013). More specifically, the supposedly excitatory electrode (anode) was placed over the left DLPFC (i.e., to increase the reduced activity) and the supposedly inhibitory electrode (cathode) over the left TPC (i.e., to reduce the increased activity). Our study refers to this placement of electrodes as the "treatment montage" (see Figure 1 B).

The first study using the treatment montage (Brunelin et al., 2012) administered tDCS two times per day (20 min each session) for five consecutive days to thirty patients with schizophrenia. Current strength was 2 mA. Half of the participants received real stimulation, whereas the other half got no stimulation (i.e., sham condition). The study showed a decrease in AVH severity by 31% for the participants that received real tDCS, with the effect lasting for up to a three-month period after the stimulation process (Brunelin et al., 2012). Other placebo-controlled studies (Bose et al., 2018; Brunelin et al., 2012; Mondino et al., 2014), as well as case studies (Nawani et al., 2014; Rakesh et al., 2013; Shiozawa et al., 2013) demonstrated a similar reduction of AVH by using the same tDCS electrode montage (i.e., treatment montage). Collectively, these studies strengthen the theory that hyperactivity in TPC and hypoactivity in DLPFC contributes to AVH, and that the tDCS method has the potential to modulate this aberrant activity.

However, other studies failed to find significant treatment effects (Fitzgerald et al., 2014; Koops et al., 2018). For instance, Fitzgerald et al. (2014) conducted a study where tDCS was applied concurrently to the prefrontal cortex and temporoparietal junction with the same current strength (2 mA) as in Brunelin et al. (2012) study. However, the study showed no reduction in AVH severity. One reason for this might be that this study used a less frequent and intensive application of tDCS, as well as a different electrode montage set-up than that of Brunelin et al. (2012). On the other hand, Koops et al. (2018) used the same tDCS procedure as Brunelin et al. (2012) but still failed to find the same significant effect on AVH. When summarized, both systematic reviews and meta-analysis of tDCS tend to show only a small effect size on AVH (Guttesen et al., 2021; Kim et al., 2019; Pondé et al., 2017).

As previously mentioned, schizophrenia is considered to be a very heterogenous disorder with immense variations in clinical presentation, course, and prognosis (Tandon et al., 2008). Differences in symptoms, cognitive deficits, and medication parameters between these participants may therefore in part explain some of the inconsistent results in the above tDCS studies. Clinical studies also tend to have a relatively large participation criteria list, such as needing to have at least three AVHs per week, no recent admissions, and stable antipsychotic medication. This can often lead to a smaller number of participants being recruited and thus more ambiguous results (Nieuwdorp et al., 2015).

1.8 tDCS in Healthy Participants

There is an increasing number of studies that used tDCS in healthy participants, rather than clinical samples, to investigate the neuronal mechanisms that are involved in AVH (Mondino, Poulet, et al., 2016; Moseley et al., 2014). Even though it might seem counterintuitive to use healthy individuals to explain the neuronal mechanisms that are involved in AVH in schizophrenia patients, it does have some clear advantages. For instance,

it is easier to recruit a larger number of participants and studies can avoid having to take into account differences in symptoms, cognitive impairment, medication, or other difficulties that one can encounter when testing schizophrenia patients (Mondino, Poulet, et al., 2016; Moseley et al., 2014). Healthy participants can also contribute to more controlled conditions by investigating how different placements of electrodes might affect the frequency of auditory false perceptions. For instance, many studies used the tDCS treatment montage to increase the activity in the DLPFC and decrease the activity in the TPC in AVH patients with schizophrenia (see Figure 1 B; Brunelin et al., 2012). However, trying to reverse this model in a clinical sample, that is using tDCS to increase the already hyperactive TPC and decreases the already hypoactive DLPFC (see Figure 1 C), would be considered as unethical. Healthy participants can therefore give studies the opportunity for more controlled conditions when it comes to the placements of tDCS electrodes.

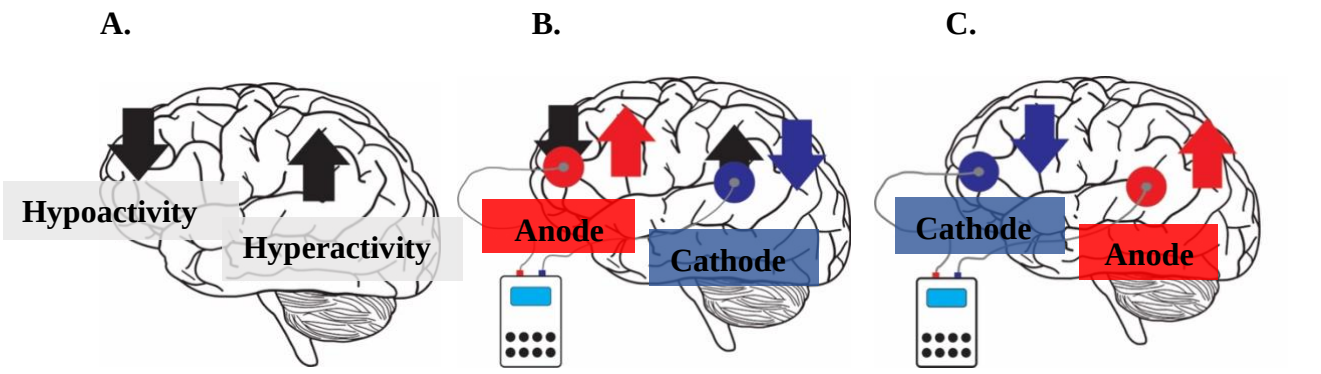
In general, few studies have focused on the role that tDCS has on auditory false perceptions. One study found that anodal stimulation over the left TPC increased auditory false perceptions in healthy individuals, compared to cathodal stimulation (Moseley et al., 2014). Moreover, Mondino, Poulet, et al. (2016) showed that anodal tDCS over the left TPC increased the likelihood to misattribute internally generated speech to external speech (i.e., deficits in reality monitoring). The study also showed that this stimulation procedure did not lead to any significant changes in the tendency to misattribute self-generated thoughts to self-generated speech (i.e., internal source monitoring), nor did cathodal stimulation over the left DLPFC lead to any modulations in neither internal nor reality monitoring abilities. Thus, these findings indicates that the TPC alone plays an important role in reality monitoring. However, literature suggests that the left DLPFC also plays an important role in AVH. Moseley et al. (2014) used anodal, cathodal, or sham tDCS on the left TPC on all their participants. Mondino, Poulet, et al. (2016), on the other hand, gave half of their participants

anodal and sham stimulation to the left TPC, whereas the other half got cathodal and sham stimulation to the left DLPFC. Neither of these studies therefore directly investigated the hypertemporal/hypofrontal model of AVH by exploring the role of the left TPC and left DLPFC simultaneously.

As previously mentioned, there seems to be a decrease in AVH severity when anodal stimulation is delivered to the left DLPFC and cathodal stimulation is delivered to the left TPC in AVH patients with schizophrenia (Brunelin et al., 2012; see Figure 1 B). As a consequence, studies have tried to reverse this model in healthy participants in order to mimic the hypertemporal/hypofrontal activity that is observed in AVH patients (Kusztrits et al., 2021). This current study refers to this electrode placement as the "AVH montage" (see Figure 1 C).

Figure 1.

The Neuronal Basis of AVH(A), The tDCS Treatment Montage(B) and The AVH Montage(C)



Neuronal Basis of AVH
Hyperactivity TPC
Hypoactivity DLPFC

Treatment Montage
Excitatory anode DLPFC
Inhibitory cathode TPC

AVH Montage
Excitatory anode TPC
Inhibitory cathode DLPFC

For instance, Kusztrits et al. (2021) tested participants with a source monitoring paradigm with the supposedly inhibitory cathode over the left DLPFC and the supposedly excitatory anode over the left TPC. The rationale was that this AVH montage would lead to similar behavioural deficits in source monitoring abilities in healthy participants as those observed in schizophrenia patients during AVH (Kusztrits et al., 2021). The study comprised two experiments. In the first, participants completed a source monitoring task during tDCS (online). In the second, the same task was completed after tDCS (offline). The results showed that tDCS had no effect on reality monitoring in both the online and offline experiment. Internal source monitoring, however, was improved in the online experiment compared to sham. These findings are in contrast with Mondino, Poulet, et al. (2016) study that showed internal source monitoring remained unaffected when stimulating healthy participants with anodal tDCS on the TPC region. However, different tDCS parameters in these studies, such as electrode montages as well as the set-up of the experimental task might have affected these results. For instance, the studies used different source monitoring paradigms.

1.9 Aims and Objectives

The literature suggests that AVH arise from simultaneous overactivation of the left TPC and reduced activity of the left DLPFC. Clinical studies showed some promising treatment benefits in reducing AVH with anodal and cathodal tDCS of the left DLPFC and left TPC, respectively (i.e., treatment montage; Bose et al., 2018; Brunelin et al., 2012; Mondino et al., 2014; Nawani et al., 2014; Rakesh et al., 2013; Shiozawa et al., 2013). However, other studies show no such treatment effects (Fitzgerald et al., 2014; Koops et al., 2016). The reason for these inconsistencies may lie in the large individual differences in schizophrenia patients' symptomatology and medication parameters (Fitzgerald et al., 2014, p. 278; Kolb et al., 2019; Nieuwdorp et al., 2015). An increasing number of studies therefore attempted to

uncover the underlying neuronal mechanisms of AVH by using tDCS in healthy participants instead. This can control for confounding variables that inevitably arise when testing schizophrenia patients, but, of course, limits the generalizability to schizophrenia.

tDCS studies on healthy individuals mainly focused on delivering separate stimulation procedures (i.e., anodal stimulation over the left TPC and/or cathodal stimulation over the left DLPFC) to induce auditory false perceptions (Mondino, Poulet, et al., 2016; Moseley et al., 2014). None of these, however, investigated the role of the left TPC and left DLPFC simultaneously. A recent study examined the effects of the AVH montage on source monitoring, but did not find support for the notion that healthy participants show similar behaviour as clinical patients with AVHs (Kusztrits et al., 2021). Unfortunately, the AVH montage only showed small effects on the measured brain areas (Kusztrits et al., 2021). However, the reason for this might not be due to the tDCS method itself, but rather the implementation of the experimental tasks. As mentioned above, Kusztrits et al. (2021) used tDCS AVH montage to test the effects of AVH on source monitoring. The present study, however, employed a SDT to test false auditory perceptions more directly.

The present study investigated the effects of tDCS on an SDT paradigm that was specifically chosen to incorporate both top-down and bottom-up processes as suggested by the neurocognitive theoretical model of AVH (Waters et al., 2012). To this end, the study employed a SDT paradigm developed by Laloyaux et al. (2022) that includes human sound and non-human sound stimuli (bottom-up) as well as high versus low levels of expectations (top-down). Additionally, we investigated the effects of electrode montage by comparing the montage used in treatment with a montage where anode and cathode are flipped (i.e., the AVH montage; see Figure 1 B and C). The overall goal was to test if brief tDCS administered to healthy individuals will temporarily alter the probability of experiencing auditory false perceptions in the AVH montage. More specifically, I hypothesised that simultaneous anodal

stimulation of the left TPC and cathodal stimulation of the left DLPFC will mimic the hypertemporal/hypofrontal pattern observed in patients with schizophrenia and lead to an increased rate of auditory false perceptions in healthy participants, as compared to anodal stimulation of the left DLPFC and cathodal stimulation on left TPC (H_1). Second, this effect should be particularly pronounced when participants expect a certain word (top-down effect of high expectation) and when the word is embedded in noise that resembles human speech (bottom-up effect; H_2).

Examining bottom-up, top-down, and montage effects simultaneously, requires a sample size that is beyond the scope of a single master's thesis. Thus, our study includes data that was collected by me, plus data collected in a previous master thesis, and a bachelor thesis. The bachelor thesis was partly supervised by me.

2. Method

2.1 Research Design

The study employed a double blind, randomized controlled experimental design. All participants attended two stimulation sessions (real versus sham tDCS) where the electrode montages (i.e., the placement of electrodes) and the stimulation mode (i.e., real or sham) was an independent group variable. Standard SDT measures (i.e., hit rate, false alarm rate, perceptual sensitivity A' and response bias β) served as dependent variables.

2.2 Participants

The random sample of participants in this study was a combined effort between a previous master thesis (n= 24), a bachelor thesis (n= 23) and my own recruited sample (n=30). I decided to exclude one participant from the combined dataset due to an unusually

long period between experimental sessions (30 days). No other participants were excluded from their original data sets. In total, 76 naïve participants (33 males; 43%, 43 females; 57%) aged 19-34 (*M*= 23.88, *SD*= 3.45) took part in this experiment. Participants were recruited via social media, word of mouth, and flyers that were distributed in several faculties in the University of Bergen and Haukeland University Hospital. Participants were selected according to the safety screening criteria for tDCS that was sent to them digitally prior to the first visit. None of the participants had partaken in any tDCS experiment before the first visit. Participants were only told that the study was investigating the effects of tDCS on auditory perception, thus remaining naïve to the specific aims of the study. All methods in this study were performed according to the relevant guidelines and regulations approved by the *Regional Committees for Medical and Health Research Ethics (2017/1732/REK vest)* and the University of Bergen. Participants were compensated with 250 NOK after their last session.

2.3 Materials and Stimuli

2.3.1 Experimental Documentation.

The study used a tDCS safety screening document that contained some well-established safety guidelines that would ensure that each participant met the medical requirements of the study and could safely participate (Bikson et al., 2016; Matsumoto & Ugawa, 2017). Participants in this study self-reported no neurological or psychiatric disorders (personal or first degree), no previous neurosurgical treatments (including eye surgery), no metal surgically implanted into the body, no chronic skin disorder, no dyslexia, or ADHD diagnosis, no pregnancy, or use of medication that could affect the task or pose risk for tDCS safety. In addition, all participants self-reported being fluent in both spoken and written Norwegian language and not consuming any drugs and alcohol at least 24 hours prior to the

tDCS sessions. None of the participants had taken part in any other TMS/tDCS on the same day as the experiment was conducted.

The study also used a consent form in accordance with the Norwegian law on research ethics and medical research that contained a detailed description of the test materials and the protocol relating to tDCS method, as well as the participants right to withdraw from the study at any time.

2.3.2 Questionnaires.

A questionnaire was used to collect basic demographic information such as gender, degree of education, nicotine uses, neurological disorders and participants' handedness. Out of all 76 participants 61 (80%) were students at the time of participation. The remaining 15 participants (20%) had already graduated. 16 participants (21%) reported snuffing and/or smoking habits, whereas 60 participants (79%) reported no current nicotine habits.

Handedness was assessed with the Edinburgh Handedness Inventory where participants indicated their right- or left-hand preferences during certain activities (e.g., write, draw, sweeping, open a box etc). The answers given provided a laterality score between -100 (exclusively left-handed) to +100 (exclusively right-handed; Oldfield, 1971). Using a cut-off score of 0, seventy-one participants were right-handed (93%), while five participants were left-handed (7%). This is comparable to the general population, where roughly 10% are left-handers (Lansky et al., 1988). Previous studies demonstrated that hemispheric language specialization is affected by handedness, with roughly 70% of left-handers and 95% of right-handers having left-hemispheric language specialization (Rasmussen & Milner, 1977). Since our study specifically targeted the left hemisphere because of its specialization for language processing, participants with atypical, right-hemispheric language specialization could pose a problem. However, given that statistically only a very small proportion of participants are

likely to show atypical language lateralization (ca. three to four right-handers and two to three left-handers), it was deemed unnecessarily expensive to verify language lateralization with neuroimaging and both left- and right-handers were included.

The study also used the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) questionnaire where participants answered "yes" or "no" to several questions (30 items) related to their thoughts, feelings, experiences, and preferences. This questionnaire was designed to measure psychotic characteristics or schizotypal tendencies in healthy individuals. The items include questions describing hallucinations, abnormal or magical thinking (e.g., "Have you felt feel like your head, arms and legs were not your own?" and "Can some people make you aware of them just by thinking about you?"). Studies have shown that patients with schizophrenia have relatively high scores on the O-life questionnaire (i.e., respond yes on 12 items or more), whereas those with a low proneness to hallucinations healthy have relatively lower scores (i.e., respond yes on 6 items or less; Thomas et al., 2019). Participants in our study mostly showed low, or perhaps, low to medium schizotypal tendencies (M= 7, SD= 5.21).

A tDCS Adverse Effects Questionnaire (Brunoni et al., 2011) was used to measure side effects (i.e., itchiness, headache, redness) of the real/sham stimulations that will be addressed in the results section.

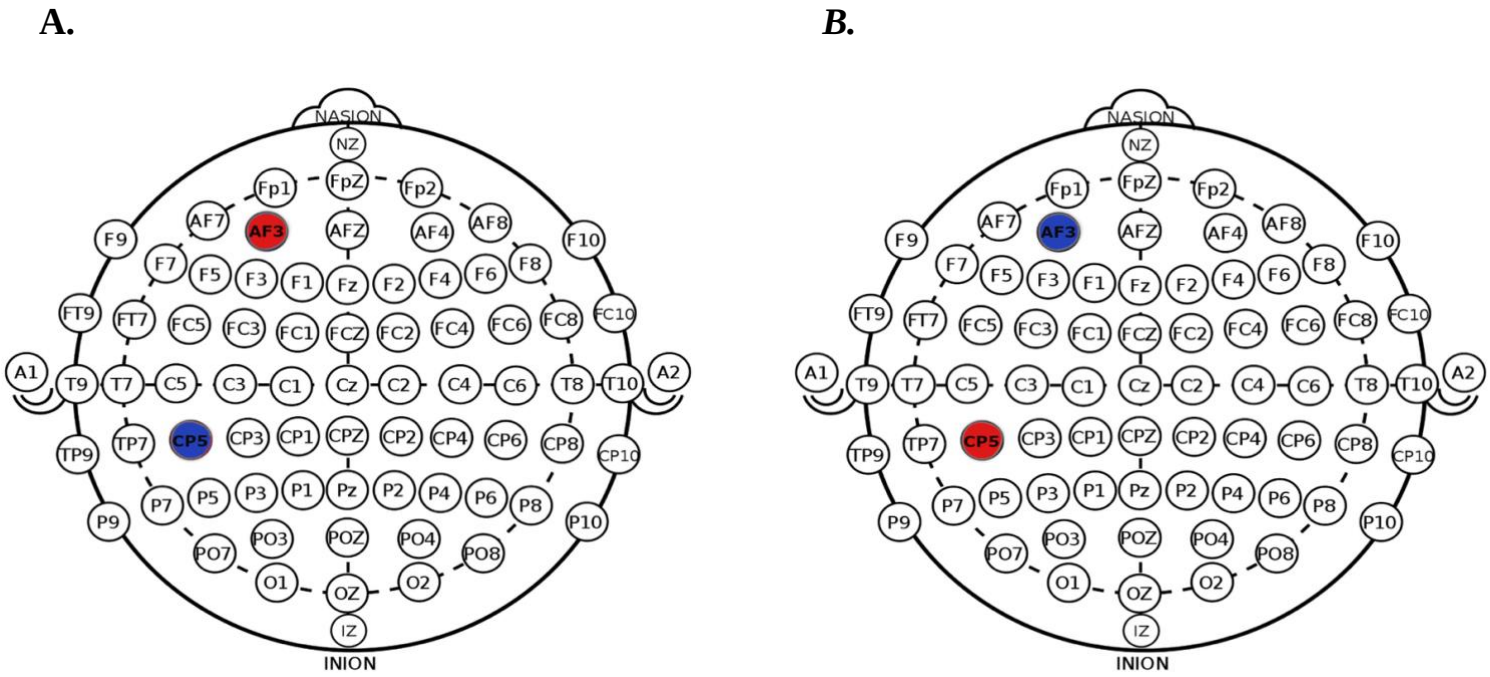
2.3.3 tDCS.

Stimulation was elicited by a battery driven constant current stimulator (NeuroConn DC-stimulator) and delivered through a pair of rubber electrodes (7 x 5 cm= 35 cm^2) in saline-soaked sponges (6 x 6 cm=36 cm^2) to generate good electrical contact with both the skin and scalp of the participants. The electrodes were also applied with gel to reduce any discomfort, such as itchiness, burning and tingling sensations. To ensure that the electrodes

were placed in the correct locations on the skull and to adjust for individual differences in head size across participants, an internationally agreed coordinated system was used (i.e., electroencephalogram; EEG 10-20 system; Yeom et al., 2014). One electrode was placed over the AF3 region (i.e., the left DLPFC) and the other electrode were placed over the CP5 region (i.e., the left TPC; see Figure 2). The electrodes were held in place by an adjustable rubber strap.

Figure 2.

The tDCS Electrode Placements Used in this Study: The Treatment Montage (A) and The AVH Montage (B)



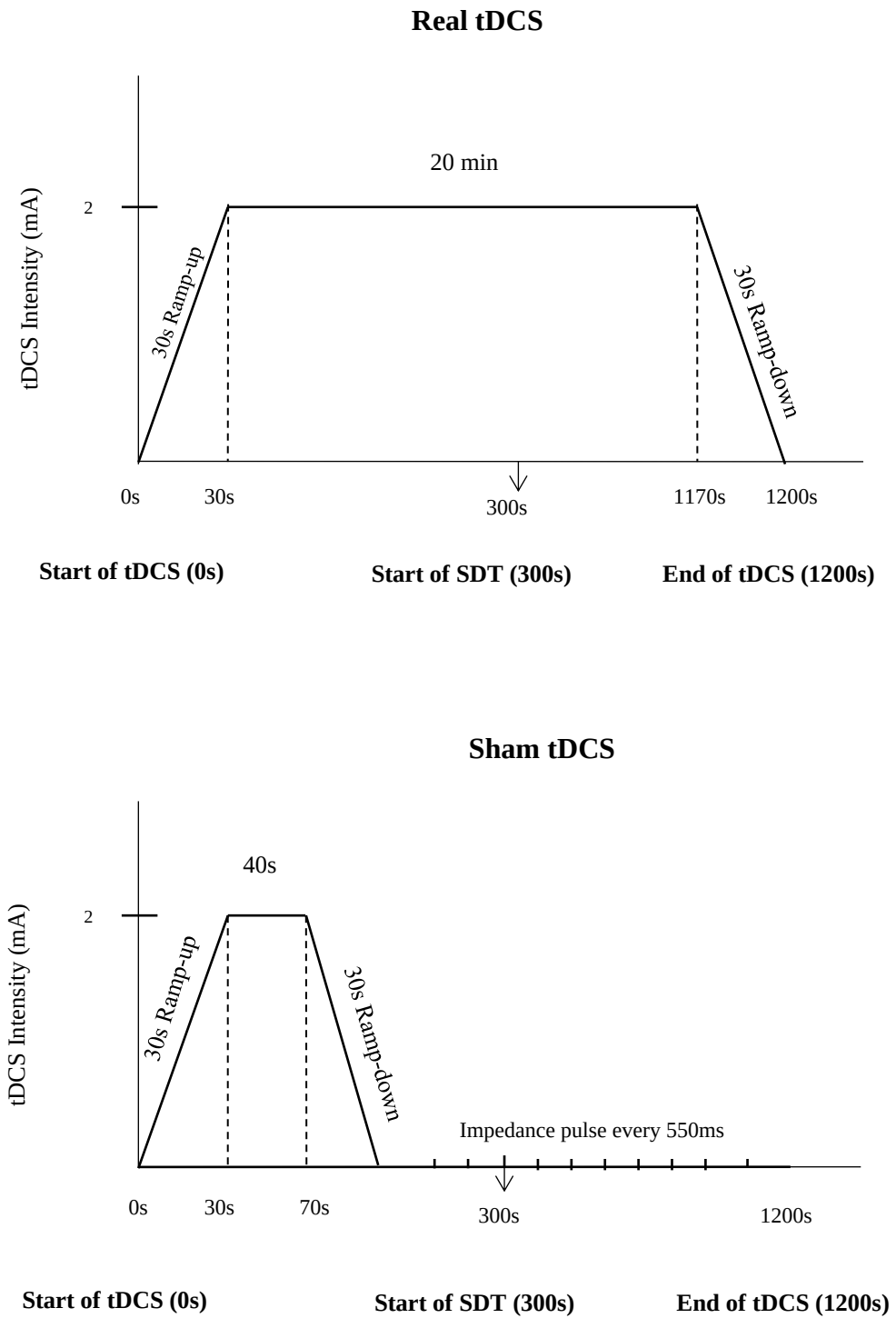
Note. Figure 2 shows the tDCS electrode placements targeted in this study. The targeted locations were based on the international EEG 10/20 system: the AF3 is the left DLPFC and the CP5 is the left TPC. The figure is based on an image taken from Kusztrits et al. (2021): https://doi.org/10.1371/journal.pone.0257010.g001

Our study used two different montage setups based on the supposedly hyper-, hypoactivation pattern in AVH (see Figure 2): the first montage (i.e., the treatment montage) involves anodal stimulation on the left DLPFC and cathodal stimulation on the left TPC (see Figure 2 A). The second montage (i.e., AVH montage) is the reversed setup, with the cathodal stimulation over the left DLPFC and anodal stimulation over the left TPC (see Figure 2 B). Thirty-nine participants (51 %) completed the experiment with the treatment montage and thirty-seven participants (49 %) with the AVH montage.

Regardless of montage, all participants received real stimulation in one session, and sham stimulation in the other. Forty-one participants (54 %) got real stimulation in their first session, and thirty-five (46 %) got sham in their first session. Both the experimenter and the participants were naïve as to which session the real or sham stimulations were delivered (double-blind). To make sure the experimenter was blinded to whether the participants received real or sham stimulation, the study used input codes to start the tDCS before each session. The real stimulation was administered for 20 minutes/1200 seconds with a constant current of 2 mA. Before reaching 2 mA, the current was ramped up for 30 seconds, and at the end of the stimulation the current was ramped down for 30 seconds (see Figure 3). During sham stimulation, the current was ramped up for 30 seconds and a 2mA current strength was delivered for approximately 40 seconds before it was ramped down within 30 seconds. After the ramp down, a small impedance control pulse was delivered every 550 milliseconds until the end of the stimulation (see Figure 3). In the sham condition, the 30 seconds ramping up would create a similar itching and tingling sensations that participants experienced during the real stimulation (Mondino et al., 2014). This way, sham feels like real tDCS. However, the 20 minutes continuous impedance testing in the sham condition is insufficient to alter membrane polarity, as opposed to the real tDCS (Nitsche et al., 2008).

Figure 3.

Illustration of the Real and Sham tDCS Procedures.



2.3.4 Signal Detection Task (SDT).

As described above, our study chose a SDT developed by Laloyaux et al. (2022) that is based on a paradigm by Hoskin et al. (2014). This SDT paradigm requires participants to listen to 70 recorded sentences, where the last word is either missing or embedded in one of two noises: non-human noise (white noise) and human noise (white noise in the same frequencies as human speech). 30 sentences in the SDT had the end-word masked by a noise, whereas 40 sentences ended with noise only. Participant were required to indicate whether a word was presented by responding either "yes" or "no" on a marked keyboard (c = yes, v = no). After the response, participants were asked to indicate on a scale from 1-4, how certain they are about their response (1 = uncertain, 2 = some uncertainty, 3 = some certainty, 4 = certain).

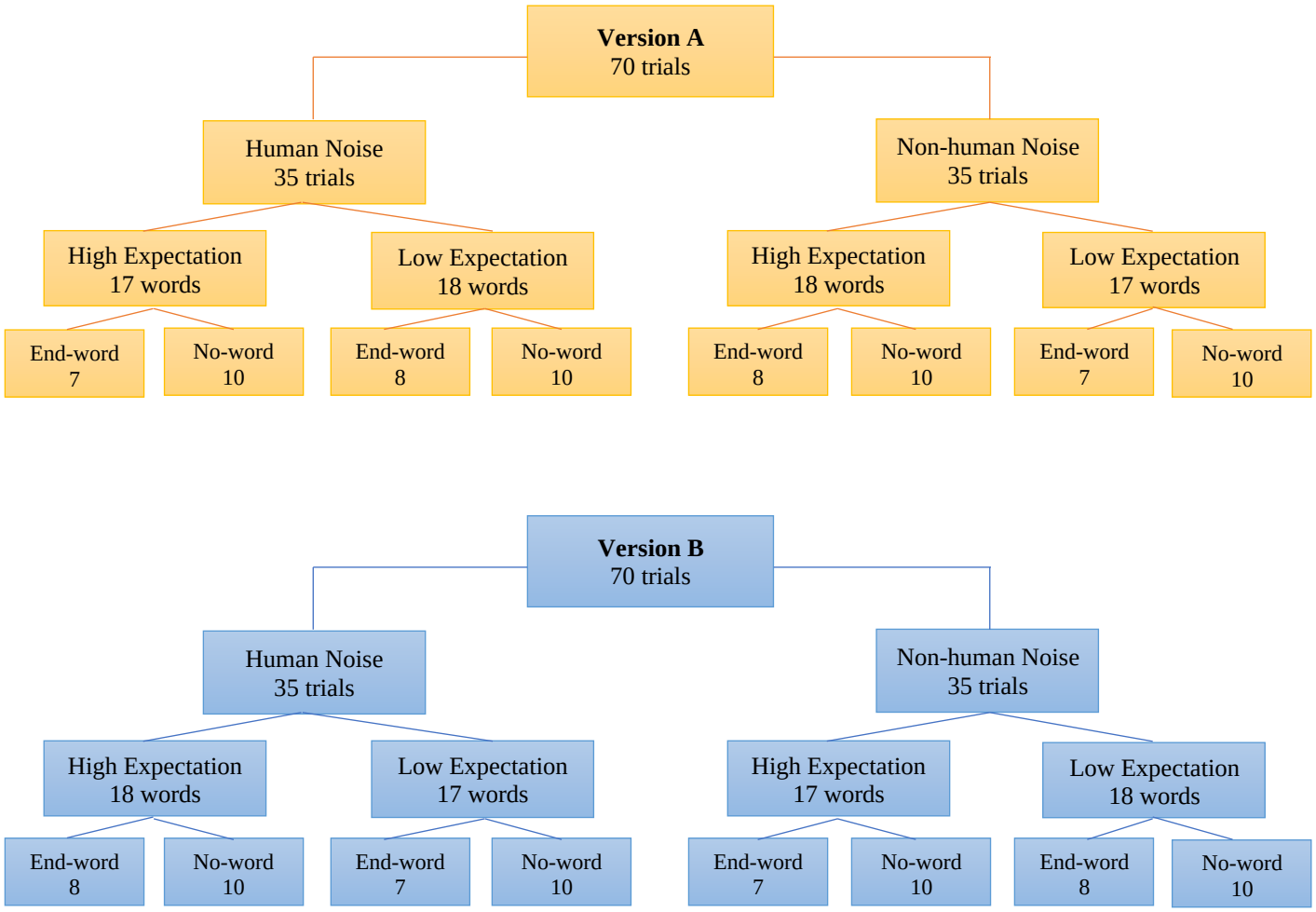
All sentences were recorded in Norwegian with a commonly used dialect from Bergen. Half of the sentences were spoken by a female voice (35), the other by male voice (35). The high or low expectation sentences were created in a separate pilot study by Laloyaux et al. (2022) where 25 participants were asked to fill in the missing word of sentences. High expectation sentences had at least 85% of participants reporting the same missing word, whereas low expectations sentences had a maximum of 35% (10-35%). our study used 35 sentences that created hight levels of expectation (e.g., sentence: "the apple fell from the...", end-word: "tree") and 35 sentences with a low expectation (e.g., sentence: "the best would be...", end-word: "marriage") of the end-word. There were between 4 and 9 words (including the end-word) in the sentences with high level of expectation (number of words *M* = 5.84, *SD* = 1.48), and between 3 and 8 words (including the end-word) in the sentences that created little or no expectation (number of words *M* = 4.81, *SD* = 1.09).

All sentences and end-words were easily comprehensible, emotionally neutral, and impersonalized. All participants partook in two different versions of the SDT (version A and

version B) that both contained 70 items total. Participants completed either version A or B in their first session and the remaining version in their second session to minimize practice effects. Distribution of the two versions was counterbalanced, such that thirty-five participants (46 %) were given version A during real stimulation, whereas forty-one participants (54 %) were given version B during real stimulation. Both the A and B version had equal number of sentences with human/non-human noise, female/male voice, and words with high/low expectations (see Figure 4). Each version of the SDT roughly takes about 10-15 minutes to complete and the order of the sentences were pseudorandomized.

Figure 4.

The Distribution of Sentences and End-words in the SDTs



Note. Figure 4 shows the distribution of sentences and end-words in both version A and B of the SDT (i.e., 70 sentences categories into type of noise, degree of expectation, and whether the end-word is present).

The following measures where derived from the SDT: *"hit rate", "false alarm rate", "perceptual sensitivity* A'*",* and *"response bias* β*".* The definitions and calculation steps for these measures were taken from Stanislaw and Todorov (1999) which include:

(1) Hit rate (i.e., probability of responding "yes" to trials where a word was presented) was calculated by dividing the number of hits by the total number of trials, in which a word was presented.

(2) False alarm rate (i.e., probability of responding "yes" to trials where no word was presented) was calculated by dividing the number of false alarms by the total number of trials in which no word was presented.

(3) Response bias (β) (i.e., the general tendency to respond either "yes" or "no") was calculated using a mathematical formula taken from Stanislaw and Todorov (1999):

$$\beta = e^{\left\{\frac{[\Phi^{-1}(FA)]^2 - [\Phi^{-1}(Hit)]^2}{2}\right\}^2}$$

The Φ ("phi") function converts z scores into probabilities. Thus, calculations of β requires the constant e to be multiplied by the square difference between the square false alarm rate (FA) rate and the square hit rate (Hit) divided by two. β = 1 indicates no bias, β < 1 indicates a bias towards responding "yes", and β > 1 indicates a bias towards saying "no" (Moseley et al., 2014). This means that if participants have a β value less than 1 they are more inclined to respond "yes" (liberal bias). On the other hand, having a β value that is higher than 1 indicates a preference to respond "no", that is, a more conservative response bias and being less likely to believe that an end-word is present.

(4) A' (i.e., the ability to hear an end-word against the white noise) typically ranges from .5 (i.e., the signal cannot be distinguished from noise) to 1 (i.e., perfect performance).

Thus, a higher A' values indicate a better sensitivity (Stanislaw & Todorov, 1999). A' was calculated as follows:

$$If\ Hit \geq FA\ then\ A' = 0.5 + \frac{(\mathrm{Hit} - \mathrm{FA})(1 + \mathrm{Hit} - \mathrm{FA})}{4\mathrm{Hit}(1 - \mathrm{FA})}$$

$$If\ Hit < FA\ then\ A' = 0.5 - \frac{(\mathrm{FA} - \mathrm{Hit})(1 + \mathrm{FA} - \mathrm{Hit})}{4\mathrm{Hit}(1 - \mathrm{Hit})}$$

Typically, d' is used as an indicator of perceptual sensitivity. However, since it could not be verified that both signal and noise were normally distributed and had the same standard deviation in this yes/no response task, A' should be calculated instead (Laloyaux et al., 2022).

2.4 Procedure

Prior to the study, three separate randomization lists were created for each experimental study (one for our study, one for the previous master thesis, and one for the bachelor thesis). In all studies, the different conditions (i.e., electrode montage, SDT version and stimulation conditions) were counterbalanced to control for any nuisance variables. More specific, each study set a target for the number of participants that would be recruited. Participants were then counterbalanced between the tDCS montage (treatment montage and AVH montage) and the order in which they would receive version A or B of the SDT and real or sham stimulation (session 1 and 2; see Figure 5).

The randomization list used in our study contained the counterbalancing procedure for 30 participants. As previously mentioned, the list used input codes instead of real/sham labels. These codes were assigned by a person that was not involved in the actual data collection. The randomization list was then given to the experimenter (me) with the

instructions as to what electrode montages, input codes and SDT version should be used for each participant in each session, with participant 1 starting at the top (see Figure 5).

Figure 5.

Example of the Randomization Lists that was *used to Counterbalance Various Experimental Factors.*

Nr.	LDLPFC	ID code	Session 1 Input code	SDT 1	Session 2 Input code	SDT 2
1	Anode		18739	A	23613	B
2	Cathode		47452	A	28303	B
3	Anode		48014	B	54320	A
4	Cathode		23373	B	24005	A
5	Anode		43909	B	14542	A

Note. Figure 5 shows an example of the randomization list that was used in our study. For example, participant 1 would get anodal stimulation on the left DLPFC in both sessions where input code 18739 and SDT A will be used in their first session.

All participants took part in two sessions that lasted approximately 1 hour each (i.e., two hours in total). The study aimed for a 7-day gap between the two sessions, but due to illness, covid restrictions and other unforeseen events some participants completed the trial sooner or later than this. The number of days between the two sessions therefore varied between 4 to 18 days (M = 7.68, SD = 2.62). Although, fifty-six participants (74 %) had a gap of 6-8 days. Prior to both sessions, participants were asked to complete a tDCS safety checklist as described above (Bikson et al., 2016; Matsumoto & Ugawa, 2017). Prior to the

first session, all participants signed a written consent form. After giving consent, participants were asked to fill in a general information form with basic demographic and hand dominance questions. Subsequently, participants were given the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) questionnaire.

Afterwards, the tDCS electrodes were attached to the participants' head. Before each session, a short stimulation test was conducted to check that the impedance level displayed on the stimulator was within adequate levels (>14 k ohms). If impedance was too high, adequate procedural checks (Thair et al., 2017) on cables, electrodes, and so on were carried out until the short stimulation test ran smoothly. The short stimulation test also gave participants an opportunity to experience the sensations of the tDCS before the actual experiment. During this test, participants were told that the current would be ramped up for 30 seconds and a 2mA current strength would be delivered for approximately 10-15 seconds before it was ramped down again for 30 seconds. Before the stimulation test, participants were informed that they could experience tingling and/or itching sensations.

After the short stimulation test, participants were moved to a soundproof room and seated in front of a stationary computer. Once seated, the experimenter used an input code to activate either the real or sham tDCS. To ensure that both target areas had been stimulated long enough to exert behavioural effects, stimulation was administered for approximately 5 minutes (300 seconds) before the start of the SDT in both the real and sham condition. During these 5 minutes, a semi-open HiFi headphone (Beyerdynamic DT 880 Premium 32-Ohm) were placed on the participants head and adjusted to cover both ears. Participants were then given some general information about the SDT and asked to complete a short practice trial that consisted of 3 sentences and end-words. After the practice trial, participants were asked if they understood the experimental task and given an option to undergo an additional practice trial, if necessary.

Once the tDCS current had been delivered for 300 seconds, participants commenced the SDT that lasted for approximately 10-15 minutes. Participants remained alone in soundproof room, except for the experimenter occasionally entering to check the impedance level. However, participants were told to ignore the experimenter during the task. All participants completed the SDT before the stimulation ended. This means that the tDCS outlasted the signal detection task in all sessions. Participants were asked to remain seated until the stimulation was complete during which the headphones were removed. After real/sham tDCS finished, electrodes were removed, participants' skin was inspected for redness, and participants were asked to complete the adverse side effects questionnaire. After the second session, both the participant and the experimenter were required to answer in which session (1 or 2) they believed real stimulation had been administered. Subsequently, participants received compensation (250 NOK), were thanked and debriefed about the specific aims of the study.

2.5 Data Analysis

All statistical analyses were conducted using IBM SPSS Statistics version 27. A significant level of $p < .05$ was applied to all subsequent analyses and there were no violations in sphericity (ε) assumptions (Mauchly´s test of sphericity = $p > .05$). The variances of the different dependent factors were roughly equal and there was no need for sphericity corrections (Greenhouse-Geisser; $\varepsilon < 0.75$, or Huynh-Feldt correction; $\varepsilon > 0.75$).

The study used a 2 x 2 x 2 x 2 mixed ANCOVA for the SDT with stimulation condition (real/sham tDCS), degree of expectations (high/low), and noise type (human/non-human) as within-subject variables. The tDCS montage (treatment/AVH) was used as a between-subject variable. As mentioned, there were some differences in the days between the two sessions, thus the study included "days gap" as a covariate on all the SDT dependent

measures (i.e., hit rate, false alarm rate, perceptual sensitivity, and response bias). The study also ran an additional, exploratory 2 x 2 x 2 within-subject ANCOVA *without* the tDCS montage grouping (but keeping stimulation condition, degree of expectations, and noise type as within-subject variables), to maximize statistical power for a possible three-way interaction (H_2).

Effect sizes were provided as partial eta squared values with the standard convention that: $\eta^2 = 0.01$ indicates a small effect; $\eta^2 = 0.06$ indicates a medium effect; $\eta^2 = 0.14$ indicates a large effect (Maxwell et al., 2017). If the ANCOVA showed any main effects, estimated marginal means and standard errors were reported for all variables. Further, if the ANCOVA revealed any interaction effects, post hoc paired samples t-tests were employed with Bonferroni adjustments, and means, standard errors and t-values were reported for these interactions. The study also ran one sample t-tests for any significant main or interaction effects on β values to estimate whether the bias was significant lower or higher than 1, indicating whether participants had a bias towards saying "no" or "yes", respectively.

A G*power analysis (Faul et al., 2009; Faul et al., 2007) was employed to determine how much power a sample of 76 participants would have in finding a significant 2 x 2 x 2 interaction. The G*Power analysis settings were as follows: power = 0.80, $\alpha = 0.05$, number of groups = 1, correction among repeated measures = 0.5, non-sphericity correction = 1. G*Power does not directly support interactions in repeated measures designs, but the interaction can be coded indirectly by using the following formula for the number of measurements (2-1) * (2-1) * (2-1) + 1 = 2. Finally, this study used the "from SPSS" effect size specification based on the notion that G*Power assumes that the effect size (Cohen's f) and the correlation between repeated-measures factors are separate. However, this correlation is already factored into the effects size in the "η^2" from SPSS that is based on the sum of

squares (Lakens, 2013). The G*power analysis revealed an effect size of $f = 0.33$, implying that our study could detect a medium effect with 0.80 power.

Before conducting the statistical analysis, the study checked for outliers in each dependent variable using a well-established outlier labelling rule created by Hoaglin and Iglewicz (1987). The study detected 5 potential outliers: 1 participant below the lower quartile (25%) and 4 participants above the upper quartile (75%). However, these participants were not classified as consistent outlier across all the four dependent measures. Moreover, all significant and non-significant effects remain the same, regardless of whether those five participants were included or not. I therefore decided to run the statistical analyses including all 76 participants to keep statistical power maximum.

To test whether blinding was successful a binomial nonparametric analysis with the test proportion 0.5 was run using the proportion of correct guesses from the participants and the experimenter. Finally, a Wilcoxon signed rank tests was then employed to investigate any significant differences between real and sham tDCS side effects.

3. Results

3.1 Results on the SDT Paradigm

The following tables presents the descriptive statistics of the tDCS effect on hit rates (Table 1), false alarm rates (Table 2), sensitivity A' (Table 3) and response bias β (Table 4) in across noise type (human and non-human), degree of expectation (low and high), stimulation conditions (real/sham), and electrode placements (treatment/AVH montage). Descriptive statistics for total rates are given in Appendix A, for noise type in Appendix B and degree of expectations in Appendix C.

Table 1.

Means are Shown Here as Percentages and (Standard Deviation) for Hit Rates Across Noise Type (Non-Human/Human)/Degree of Expectation (High/Low) Trials, Stimulation Condition (Real and Sham), and Electrode Placement (Treatment Montage and AVH Montage).

	Hit rates (%)							
	Human noise/ Low expectation		Human noise/ High expectation		Non-Human noise/ Low expectation		Non-Human noise/ High expectation	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Treatment Montage	72.79 (11.86)	74.45 (11.93)	73.48 (13.42)	71.72 (12.82)	87.69 (11.98)	83.01 (16.01)	88.67 (8.50)	90.79 (6.05)
AVH Montage	77.27 (10.75)	78.30 (13.21)	73.76 (16.03)	73.85 (12.64)	85.66 (12.02)	88.49 (9.61)	91.12 (6.18)	91.74 (5.15)
Total	74.97 (11.48)	76.32 (12.63)	73.62 (14.65)	72.76 (12.69)	86.70 (11.96)	85.68 (13.48)	89.87 (7.52)	91.25 (5.61)

Table 2.

Means are Shown Here as Percentages and (Standard Deviation) for False Alarm Rates Across Noise Type (Non-Human/Human)/Degree of Expectation (High/Low) Trials, Stimulation Condition (Real and Sham), and Electrode Placement (Treatment Montage and AVH Montage).

	False Alarm Rates (%)							
	Human noise/ Low Expectation		Human noise/ High Expectation		Non-Human noise/ Low Expectation		Non-Human noise/ High Expectation	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Treatment Montage	10.84 (9.82)	10.14 (8.50)	12.24 (10.12)	11.06 (13.18)	8.28 (5.79)	8.98 (9.53)	8.04 (7.41)	8.51 (8.02)
AVH Montage	14.62 (14.50)	11.92 (10.24)	13.39 (11.03)	12.66 (11.89)	9.22 (6.98)	10.44 (10.10)	8.73 (8.46)	11.18 (9.00)
Total	12.68 (12.38)	11.01 (9.37)	12.80 (10.52)	11.84 (12.51)	8.73 (6.37)	9.69 (9.77)	8.38 (7.89)	9.81 (8.56)

Table 3.

Means and (standard deviation) for A' *Across Noise Type (Non-Human/Human)/Degree of Expectation (High/Low) Trials, Stimulation Condition (Real and Sham), and Electrode Placement (Treatment Montage and AVH Montage).*

	A'							
	Human noise/ Low Expectation		Human noise/ High Expectation		Non-Human noise/ Low Expectation		Non-Human noise/ High Expectation	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Treatment Montage	.89 (.06)	.90 (.04)	.88 (.07)	.88 (.06)	.94 (.03)	.93 (.06)	.95 (.03)	.95 (.03)
AVH Montage	.89 (.05)	.90 (.04)	.88 (.06)	.88 (.06)	.93 (.04)	.94 (.04)	.95 (.03)	.95 (.03)
Total	.89 (.05)	.90 (0.04)	.88 (0.06)	.88 (.06)	.94 (.04)	.93 (.05)	.95 (.03)	.95 (.03)

Table 4.

Means and (standard deviation) for β Across Noise Type (Non-Human/Human)/Degree of Expectation (High/Low) Trials, Stimulation Condition (Real and Sham), and Electrode Placement (Treatment Montage and AVH Montage).

	β							
	Human noise/ Low Expectation		Human noise/ High Expectation		Non-Human noise/ Low Expectation		Non-Human noise/ High Expectation	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Treatment Montage	2.51 (1.29)	2.47 (1.33)	2.19 (1.25)	2.68 (1.34)	1.56 (1.11)	1.86 (1.19)	1.64 (1.02)	1.34 (.73)
AVH Montage	2.07 (1.30)	2.19 (1.44)	2.06 (1.30)	2.41 (1.38)	1.66 (1.16)	1.49 (1.12)	1.37 (.88)	1.09 (.71)
Total	2.30 (1.30)	2.33 (1.39)	2.12 (1.26)	2.55 (1.36)	1.61 (1.13)	1.68 (1.16)	1.51 (.96)	1.21 (.72)

3.1.1 Hit Rates.

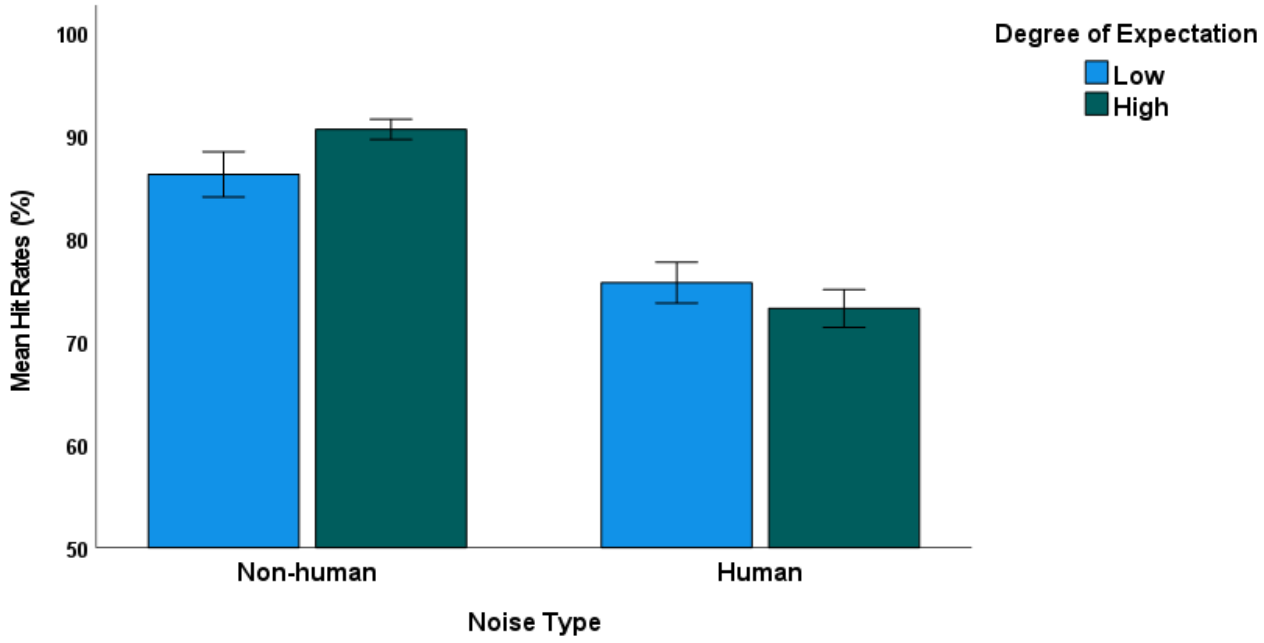
There was a significant main effect of **Noise type**, $F(1, 73) = 23.25$ $p < .001$, $\eta^2 = 0.24$, with hit rates being significantly lower for the human noise condition ($M = 74.5$, $SE = 0.9$) as compared to the non-human noise condition ($M = 88.4\%$, $SE = 0.7$).

In addition, a significant interaction effect was found for **Noise type** and **Degree of expectation**, $F(1, 73) = 4.46$, $p = .038$, $\eta^2 = 0.06$. As can be seen in Figure 6, hit rates were higher for high expectation trials ($M = 90.56\%$, $SE = 0.50$) as compared to low expectation trials ($M = 86.19\%$, $SE = 1.09$), when words were embedded in non-human noise. In turn, hit rates were higher for low expectation trials ($M = 75.65\%$, $SE = 1.01$) as compared to high expectation trials ($M = 73.19$, $SE = 0.92$), when words were embedded in human noise. Comparison in both the non-human sound condition $t(75) = 4.97$, $p <. 001$, and human sound condition $t(75) = -2.78$, $p = 001$, were significant.

There were no other statistically significant main or interaction effects on hit rates (all $Fs \leq 1.62$, all $ps \geq .239$, all $\eta^2 \leq .02$). The 2x2x2 ANCOVA, which collapsed the two electrode montage groups into one overall sample, yielded the same significant and non-significant effects.

Figure 6.

Interaction effect between degree of expectation and noise type on hit rate



3.1.2 False Alarm Rates.

There was no statistically significant main or interaction effects on false alarm rates (all $Fs \leq 2.73$, all $ps \geq .103$, all $\eta^2 \leq .04$). The 2x2x2 ANCOVA yielded the same results as the 2x2x2x2 ANCOVA.

3.1.3 Perceptual Sensitivity (A').

Noise type had a significant effect on perceptual sensitivity F (1, 73) = 32.25, p < .001, η^2 = .31, with participants showing better ability to discriminate whether a word was embedded during non-human noise (M= 0.94, SE <.01) as compared to human noise trials (M= 0.89, SE <.01).

There were no other statistically significant main or interaction effects on perceptual sensitivity (all $Fs \leq 2.41$, all $ps \geq .117$, all $\eta^2 \leq .03$). The 2x2x2 ANCOVA yielded the same results as the 2x2x2x2 ANCOVA.

3.1.4 Response Bias (β).

Stimulation condition had a significant main effect on response bias, $F(1, 73) = 4.86$, $p = .030$, $\eta^2 = .06$., indicating that the response bias β was significantly higher in sham stimulation ($M = 1.94$, $SE = 0.10$) than in real stimulation ($M = 1.88$, $SE = 0.09$), indicating a stronger tendency to say "no" during sham stimulation. Both real, $t(75) = 8.25$, $p < .001$, and sham tDCS, $t(75) = 8.67$, $p < .001$, differed significantly from 1.

Moreover, there was a significant main effect Noise type, $F(1, 73) = 4.64$, $p = .035$, $\eta^2 = .06$. The response bias was significantly higher during human noise trials ($M = 2.32$, $SE = 0.12$) than non-human noise items ($M = 1.50$, $SE = 0.08$), suggesting a stronger tendency to say "no" during human noise trials. Here, too, both human noise, $t(75) = 10.89$, $p < .001$, and non-human noise trials, $t(75) = 5.14$, $p < .001$ deviated significantly from 1.

There were no other statistically significant main or interaction effects on β (all $Fs \leq 2.36$, all $ps \geq .129$, all $\eta^2 \leq .03$). The 2x2x2 ANCOVA yielded the same results as the 2x2x2x2 ANCOVA.

3.2 Blinding Check and Adverse Effects

Fifty-eight (76%) out of 76 participants guessed correctly when being asked what session they believed real stimulation was given. The experimenter correctly guessed 54 times 71 when asked the same question. A non-parametric, binomial test revealed that both the observed proportion of correct guesses from both the participants ($p < .001$) and the

experimenter ($p < .001$.) significantly deviated from a random proportion of 0.50. This shows that the blinding for both participants and experimenter was unsuccessful.

The number of adverse side effects that was reported by the participants is shown in Table 5. A Wilcoxon signed rank tests revealed that the only significant difference between real and sham condition was found for tingling sensation ($p = < .001$) and itching sensations ($p = < .001$). That is, there was a larger portion of participants that reported tingling and itching sensations after real tDCS compared to sham tDCS (see Table 5). Even so, most side effects seemed to be similar across real and sham stimulation where the number of adverse side effects remained relatively low considering two sessions of tDCS (see Table 5).

Table 5

Number of reported adverse effects after real and sham tDCS.

	Real tDCS	Sham tDCS
Headache	20	22
Neck pain	8	10
Scalp pain	9	8
Tingling	53	38
Itching	48	33
Stinging	27	23
Redness	29	26
Drowsiness	22	27
Concentration difficulties	15	14
Acute mood changes	0	1
Other symptoms	1	2
Total	232	205

4. Discussion

The aim of this study was to test the possible effects of tDCS on auditory false perceptions in healthy individuals. Previous work suggests that AVH occur due to a simultaneous overactivation of the left TPC and reduced activity of the left DLPFC. Moreover, some but not all clinical studies found that AVH were reduced after placing the supposedly excitatory and inhibitory electrodes over the left DLPFC and TPC, respectively

(i.e., treatment montage; Bose et al., 2018; Brunelin et al., 2012; Mondino et al., 2014; Nawani et al., 2014; Rakesh et al., 2013; Shiozawa et al., 2013). Considering that the treatment montage shows some reductions in AVH, this study wanted to test if reversing the electrode placements (i.e., AVH montage) would lead to an inverted effect, that is increases in auditory false perceptions, in healthy participants (H_1).

In addition, the neurocognitive theoretical model of AVH suggests that at bottom-up level, the hyperactivity of the auditory network can be triggered by external conditions, such as a noisy environment (Waters et al., 2012). At top-down level, the hyperactivity in the TPC can be triggered by various types of cognitive and emotional factors (Waters et al., 2012). Based on Laloyaux et al. (2022), the current study hypothesised that auditory false perceptions should be particularly pronounced in the AVH montage, when participants expected a certain word and when the word was embedded in noise that resembles human speech (H_2).

4.1 Results

4.1.1 Stimulation Type.

The current study found one significant main effect of stimulation type on the SDT paradigm. That is, participants had a significant lower response bias during real tDCS as opposed to sham tDCS. As previously mentioned, AVH patients have a bias towards responding "yes" according to the signal detection theory (i.e., a liberal response bias; Alganami et al., 2017; Barkus et al., 2011; Bentall & Slade, 1985; Brookwell et al., 2013). Consequently, during real stimulation we expected to find a liberal response bias amongst participants receiving the AVH montage. Interestingly, however, our study showed that participants were relatively conservative and tended to lean towards "no" responses during both real and sham tDCS. In fact, most conditions showed a response bias well above 1.

These findings are not in line with our hypothesis that real tDCS in the AVH montage, should lead to increases in auditory false perceptions. Nevertheless, our study still produced some significant effects of real tDCS. A possible explanation for the general conservative responses amongst our participants could be down to the "easier" option of responding "no". For instance, in times of uncertainty it might have been "easier" to respond "no" there was no word, rather than claiming that there was. Moreover, the real tDCS might have triggered a slight shift in this uncertainty making participants less conservative in their responses. However, this study did not analyse level of uncertainty regarding the end-word and could therefore not confirm these suggestions.

4.1.2 Montages.

Except for the one stimulation effect on response bias, there was no other significant effect of tDCS. Moreover, all the significant findings in our study were found irrespective of tDCS montages. That is, there were no significant difference in SDT performance between participants receiving treatment montage and those receiving the AVH montage. The results in this study does therefore not support our hypothesis, as we expected to show that the AVH montage would increase auditory false perceptions.

Previous research showed that anodal tDCS over the left TPC leads to increases in auditory false perception, as compared to cathodal stimulation (Moseley et al., 2014). Additionally, anodal stimulation over the left TPC has been shown to increase the likelihood of misattributing the source of the perceptual information, as opposed to cathodal stimulation to the DLPFC (Mondino, Poulet, et al., 2016). Thus, both studies demonstrated some modulation effects of anodal stimulation on the TPC. A possible explanation for why we failed to observe any modulation effects between our montages could be due to the relatively different electrode placements, compared to previous research. Our study used anodal and cathodal stimulations of the left TPC and DLPFC, simultaneously. Meanwhile, Moseley et al.

(2014) used anodal or cathodal tDCS to the left TPC, whereas Mondino, Poulet, et al. (2016) gave some participants anodal stimulation to the left TPC and others cathodal stimulation to the left DLPFC. On the other hand, Kusztrits et al. (2021) employed the same AVH montage as our study. Similarly, their study revealed that this montage did not induce similar perceptual errors in healthy individuals as in patients.

4.1.3 Noise Type.

The current study revealed that noise type had a significant main effect on three of the SDT measures (i.e., hit rate, response bias, and perceptual sensitivity). Participants had significantly higher hit rates when words were embedded in a non-human noise as opposed to human noise. Accordingly, perceptual sensitivity was lower during human noise trials. Both findings suggest that participants struggled more to correctly distinguish the presence/absence of an end-word, when it is was embedded in human-noise. This is in line with Laloyaux et al. (2022) suggestion that speech like frequencies could trigger a more pronounced hyperactivity of the auditory networks, thus making performances worse under these conditions.

If the current study did indeed trigger a similar hyperactivity of the TPC region as in AVH patients, one would expect a stronger liberal response bias during the human noise condition as in Laloyaux et al. (2022). As mentioned above, other studies using different auditory SDT paradigms also reported a greater tendency to respond “yes” there is a word present (Alganami et al., 2017; Barkus et al., 2011; Bentall & Slade, 1985; Brookwell et al., 2013). Our study, however, showed the opposite: a significantly stronger conservative response biases in human noise trials as compared to non-human noise trials. Thus, participants had a stronger tendency to respond “no”. One explanation for these conservative responses could be that human like frequencies make it generally more difficult for the participants to distinguish words from noise, as demonstrated by the hit rate and perceptual

sensitivity results. Consequently, participants become more uncertain and adopt a more cautious, “if-in-doubt-say-no” strategy. However, this suggestion is speculative as research regarding noise types in on the SDT is a relatively new field. To establish further insight, future research should investigate how different noise types affect response biases in healthy participants.

4.1.4 Noise Type and Degree of Expectation.

Our study expected to find that a high semantic expectancy of words only increases the likelihood of detecting auditory false perceptions, if it is presented together with human noise, as in Laloyaux et al. (2022) study. We managed to find one interaction effect between noise type and degree of expectation on participants’ hit rates. That is, hit rates during human noise trials were significantly higher, when participants had low expectations that a certain word would follow. Whereas hit rates during non-human trials were significantly higher, when participants had high expectations that a certain word would follow. These findings are therefore not in line with Laloyaux et al. (2022) study. Moreover, these effects were observed regardless of tDCS montages. Our study does therefore not support the hypothesis that participants in the AVH montage should have particularly pronounced auditory false perceptions during high expectations end-words that are embedded in human noise. A possible explanation for the lack of support is that Laloyaux et al. (2022) tested participants with either a high or low proneness to AVH. Our sample, on the other hand, consisted of mostly individuals that are low, or perhaps, low to medium prone to AVH, as seen by the O-life questionnaire responses. Thus, we were dependent on the tDCS method to create a higher proneness to AVH in the AVH montage. Consequently, since our study did not show any montage effects there would have been much less variation to demonstrate noise/expectation effects as opposed to Laloyaux et al. (2022) study.

Nevertheless, our study did show that hit rates during human noise trials were significantly lower when participants had a high expectation that a certain word would follow. A possible explanation for these findings could be that the high expectation of words in the human noise condition created a level of uncertainty regarding the end-word. For instance, in the high expectation sentence: "The apple fell from the…End-word: tree", participant might have been thinking about the word "tree" when the noise appeared. In the non-human noise trials, high expectations of end-words would have aided participants in determining if the end-word is present or not. However, during human noise trials high expectations trials might have made participants uncertain as to whether they heard a word, or if they were thinking about that word instead. The human noise could have been similar in frequencies to the word they were thinking of. In turn, low expectation words in the human noise trials would stand out more from the general beliefs about the end-word, thus making it easier to detect the word when it appears. However, these suggestions are speculative and future studies should investigate how noise type and different expectation levels effect responses on the SDT in healthy participants.

4.2 Explanations for the Lack of tDCS Effects

All the above findings were found irrespective of tDCS montages, and there was no significant difference in SDT performance between participants receiving treatment montage and AVH montage. The results in this study does therefore not support either hypothesis, as we expected to show that the AVH montage increased auditory false perceptions (H_1) and that this effect would be particularly pronounced when participants expected a certain word and when that word was embedded in noise that resembles human speech (H_2).

There might be several explanations for why our study failed to support these hypotheses. One explanation could be that the tDCS electrodes are not triggering their indented neuronal

networks. After our study was designed, Marquardt et al. (2021) used a computer stimulation method that models how the electric current would flow in participants' heads, based on their anatomical scans. The model was simulated with the anode over the left TPC and the cathode over the left DLPFC, that is, the AVH montage in our study. Marquardt et al. (2021) showed that the peak of the electric field was typically strongest between the two electrodes. Thus, according to those simulations, the electrodes did not primarily stimulate the left DLPFC and left TPC. Rather, the strongest electric field was near Broca's area (Marquardt et al., 2021). The study speculated that this is likely because the two electrodes are relatively close. The AVH montage in our study (i.e., anodal over left TPC and cathodal over left DLPFC) could produce the same effects as in Marquardt et al. (2021) study. That is, the peak of the electric field could be between the anodal and cathodal electrodes. However, our study did not include any structural scans that could confirm this notion and, thus, future studies are required to investigate this.

Others have also shown that the success of the frontotemporal tDCS treatment depends on how much current flows to specific brain regions (Mondino et al., 2020). As previously mentioned, several studies aimed to use the tDCS treatment montage (i.e., the anode over the left TPC and the cathode over the left DLPFC) to reduce AVH. However, responses to the tDCS treatments are highly heterogenous amongst clinical patients (Mondino et al., 2020). It has been reported that patients who respond to tDCS treatments (i.e., have at least 50% reduction in AVH) shows a higher electric field strength within the left transverse temporal gyrus, as compared to non-responders (Mondino et al., 2020). Consequently, the key to increasing auditory false perceptions in healthy participants might depend on the current flow reaching the left transverse temporal gyrus. In any case, future studies should implement a computer stimulation method, as mentioned above, to further establish current flows of tDCS and how this relates to auditory false perceptions.

A second explanation as to why our study failed to support the hypotheses, could be that the tDCS are not producing its intended modulations effects on the left TPC and DLPFC. As previously mentioned, tDCS is not yet a completely understood method with different stimulation parameters, such as electrode placements, electrode type, electrode size, current strength, and durations affecting the outcome (Nitsche et al., 2008). For instance, it was earlier assumed that a current intensity of both 1 and 2 mA has an excitatory anodal and inhibitory cathodal effect on targeted brain regions (Nitsche et al., 2008). However, there are several studies that showed that the anodal and cathodal electrodes might not produce distinct excitatory and inhibitory modulation effects (Boggio et al., 2009; Civile et al., 2021; Elmer et al., 2009; Fregni et al., 2005; Nitsche et al., 2012; Ohn et al., 2008; Plewnia et al., 2013). For example, it has been reported that anodal stimulation of the TPC area both excites and supresses tinnitus loudness in clinical samples (Joos et al., 2014). Other studies showed that the cathodal electrode produces inhibitory effects in the primary motor cortex with 1 mA, whereas 2mA had an excitatory effect (Batsikadze et al., 2013; Mosayebi Samani et al., 2019). Consequently, perhaps trying to suppress the left DLPFC would work more optimally with a 1mA current. However, there are also several other studies that did not find inhibitory cathodal effects in cognitive tasks in general (Jacobson et al., 2011). This has also been shown when the cathodal tDCS is applied over the DLPFC (Dedoncker et al., 2016).

Further support for the notion that the anodal and cathodal electrodes do not produce distinct excitatory and inhibitory modulation effects can be seen on a cellular level. For instance, several studies investigated tDCS effect on gamma-aminobutyric acid (GABA), the main inhibitory neurotransmitter, and Glx, which is the combined measure of glutamate and glutamine that typically serves as a proxy for glutamate, the main excitatory neurotransmitter (S. Kim et al., 2014; Stagg et al., 2009). It has previously been assumed that Glx levels decrease after cathodal stimulation (Stagg et al., 2009) and increase after anodal stimulation

(Clark et al., 2011; Hunter et al., 2014). However, these changes were found primarily in the motor cortex, thus questioning, if these findings can be generalized to other brain areas as well. Marquardt et al. (2021) showed that tDCS over the left TPC and DLPFC triggered a weak increase in Glx levels independent of electrode placements. Moreover, other studies showed no significant changes in Glx levels during tDCS (Antonenko et al., 2017; Jalali et al., 2018; S. Kim et al., 2014). Taken together, the effects of tDCS on Glx are inconsistent across studies, making the specific functions of the anodal and cathodal electrodes somewhat inconsistent.

A third explanation that might explain the failure to produce any modulation effects in auditory false perceptions, is the relatively big and similar sized rectangular electrodes (7x5= 35cm^2) used in our study. For instance, we employed two electrodes that were identical in size, whereas Moseley et al. (2014) and Mondino, Poulet, et al. (2016) used two electrodes of different sizes: the cathodal electrode was bigger than the anodal electrode. The rationale behind using a larger electrode is to reduce its effect, thus creating a more focal effect of the other electrode (Nitsche et al., 2008). This might explain why both Moseley et al. (2014) and Mondino, Poulet, et al. (2016) managed to show some modulation effects of the left TPC that our study did not produce.

Moreover, we also used relatively big rectangular electrodes (7x5= 35cm^2) that could have produced not only modulating effects directly underneath the electrodes but also to immediate surrounding brain regions (Datta et al., 2009; Opitz et al., 2015). This might not be a problem in clinical samples where the aim is to alter excitability of larger regions. However, it is perhaps more crucial in a study that aims to explore the contribution of two specific brain areas that are close in parameters. Thus, larger electrodes can lead to a less focal stimulation on the targeted brain areas (Datta et al., 2009; Nitsche et al., 2008).

Consequently, the similar sized and relatively big electrodes might explain the lack of modulation effects on auditory false perceptions in our study. In light of these findings, future research might want to consider incorporating smaller electrodes and circular electrodes that shows potential in increasing the spatial focality of tDCS (Minhas, Bansal, et al., 2010; Minhas, Datta, et al., 2010). The current paper therefore suggests looking into high definition tDCS (HD-tDCS), that is, a multi-electrode approach that could improve both focality and intensity on the targeted brain regions and neuronal networks (Bikson et al., 2019; Datta et al., 2009; Dmochowski et al., 2011; Huang & Parra, 2019).

A fourth explanation is that the current strength of 2mA on the left TPC and DLPFC is not enough to reach its intended targets. Previous research has shown that when the tDCS current intensity is set to 2 mA only 10 % (i.e., 0.22V/m) of this current reaches the cortex (Miranda et al., 2006). Consequently, if the tDCS only produces a low electrical effect on the targeted brain areas in our study, it would question whether the current is strong enough to produce any modulating effects at all. Additionally, one quantitative review showed that single session tDCS is not strong enough to induce changes in cognitive tasks in healthy individuals (Horvath et al., 2015).

Alternatively, increasing the current strength, as well as having longer intervals might ensure a larger electrical effect on the targeted brain areas in our study. This is supported by meta-analyses showing that more frequent and intense sessions of tDCS can be superior in making sure that enough current reaches the cortex (Cheng et al., 2020; Kim et al., 2019). However, the 2 mA current intensity, as well as an interval of 20 minutes was set in accordance to some well-established safety guidelines prior to this current study (Bikson et al., 2016; Matsumoto & Ugawa, 2017). Changing these parameters might thus be unethical for this participant sample. Nevertheless, there are several studies that showed that a 1- 2mA current is strong enough to produce modulation effects in the prefrontal, parietal, and

temporal cortices (Kuo & Nitsche, 2012; Nitsche et al., 2012; Nitsche & Paulus, 2000; Nitsche et al., 2008). However, other studies contests these tDCS findings in general, suggesting there seems to be a bias toward publishing positive tDCS results and leaving out unpublicised studies with negative results (Medina & Cason, 2017).

A final explanation is that our study did not account for individual differences regarding the susceptibility to tDCS. Modelling studies suggested that tDCS current densities are dependent on the tissue properties between the electrodes and the brain (Kuo & Nitsche, 2012). Many previous studies showed that inter-individual differences in both micro- and macro-anatomical features (e.g., composition and thickness of the skull, gyration, and the volume of cortical regions) impact both the amount of current that enters the brain and how it spreads (Brunoni et al., 2021; J.-H. Kim et al., 2014; Opitz et al., 2015). At the same time, others show that differences in cognitive abilities (Jones & Berryhill, 2012), task difficulty (Hsu et al., 2016), personality traits (Cheng & Lee, 2016; Weidacker et al., 2016), and even genetic polymorphisms (Markett et al., 2015; Plewnia et al., 2013) have the potential to moderate the susceptibility to tDCS. To account for these individual differences, future studies could implement a similar computer stimulation method as in Marquardt et al. (2021) study. Their study managed to show that individuals who are more responsive to tDCS also displayed a higher electrical field as opposed to those who were less responsive, albeit in clinical samples (Mondino et al., 2020). Nevertheless, by including anatomical scans from each participant one could, perhaps, measure individual differences regarding the susceptibility to tDCS.

4.3 Limitations

This study has already addressed several shortcomings as to why the tDCS procedure failed to produce any significant findings on auditory false perceptions. Beyond this, some

additional limitations regarding the counterbalancing procedure, the estimated power, and blinding effects will be addressed.

Firstly, the data collected in this study was a combined effort with two other research projects, meaning there were three different data collections. A failure in the counterbalancing procedure of the first study resulted in participants always receiving SDT version A during real tDCS (and version B during sham tDCS). This was compensated for by participants always receiving version B during real tDCS in our study (and version A during sham). The third study had a balanced number of version A/B real/sham combinations. However, since the number of participants varied across the three studies, and one individual participant had to be excluded in our study, there were minor imbalances across the experimental conditions when all the data was combined. However, both versions of the SDT contained the same number of sentences (70), with similar semantic features and equal distribution of human/non-human noise and low/high expectation items. Thus, it is rather unlikely that the failure to properly counterbalance these tasks amongst participants explains the lack of significant montage or stimulation findings.

Secondly, our study also attempted to justify the sample size with a statistical power analysis. Research often relies on G*power to determine how much power a data sample of N have in finding any significant main or interaction effects (Faul et al., 2007). To our knowledge, however, G*power does not allow computing mixed between/within interactions in multifactorial designs. We thus ran a power analysis for a 2 x 2 x 2 ANOVA instead, with the two montage groups collapsed and the between-subject factor removed. Considering that we did not find any significant interaction effects with montages in either the 3-way or 4-way analyses, this strategy seems justified. The G*power analysis for the three-way ANOVA revealed sufficient power (.80) to detect a medium effect with a sample size of 76

participants. While not completely underpowered, is possible that we lacked statistical power to find small effects and a possible between/within multifactorial interaction.

Lastly, the blinding in this study was unsuccessful: both experimenter and participants guessed correctly above chance when real stimulation was given. There was a significant difference in tingling and itching sensation in the real versus sham stimulation condition. Thus, more tingling, and itching sensations might have given participants a clue as to when they received real stimulation. The experimenter, on the other hand, often based her guesses on the participants' thoughts and feelings expressed during testing. The experimenter's subjective recollection of the interaction with the participants reveals some tendencies from the participants to provide feedback after the stimulation process. Statements such as "I barely noticed it this time" or "it was way more intense the first time" made it relatively easier for me (the experimenter) to answer the blinding question correctly. A possible way to avoid this is for the researcher to give an answer immediately after tDCS stimulation, before removing the participants headphones. In this way, the experimenter would become less influenced by the experience of the participant and rather answer according to own observations.

However, the fact that participants seemed to be aware when they received real or sham stimulation could have an essential impact on the SDT performance. Another subjective observation from the experimenter was that some participants were under the impression that they were given real or sham stimulation during both sessions. Consequently, this would have made it less likely that the subjective beliefs about the stimulation type would have influenced the SDT outcomes. Moreover, the fact that participants were not aware that there was one real and one sham tDCS session from the beginning makes it less likely that the failed blinding could account for our results. Nevertheless, future studies could avoid a failed blinding by including more alternative responses that can account for these real/sham

conceptions. For example, instead of just “first session”, or “second session”, the study could include “both sessions” and “neither session” as well. In this way, the 50 % probability of answering correctly amongst participants would decrease. Another alternative for a successful blinding could be to use smaller electrodes (16 cm2) that are shown to be more tolerable producing less discomfort amongst participants (Turi et al., 2014). Moreover, using transcranial alternating current stimulation (tACS) or repetitive transcranial magnetic stimulation produces less itching, tingling, or burning sensations compared to tDCS (Ambrus et al., 2010; Ambrus et al., 2011).

4.4 Conclusion

In summary, there is no other study to our knowledge that investigated how simultaneous anodal stimulation to the left TPC and cathodal stimulation to the left DLPFC affect performances on an auditory SDT paradigm in healthy participants. However, the current study was unable to confirm that an AVH montage that mimics the supposed activation pattern of AVH would increase the rate of auditory false perceptions in healthy participants. In fact, there were no tDCS effect between the two montage groups whatsoever. These results are therefore inconsistent with previous research showing increases in auditory false perceptions with anodal stimulation on the left TPC (Mondino, Poulet, et al., 2016; Moseley et al., 2014). On the other hand, our findings are consistent with previous research showing that the AVH montage do not induce similar perceptual errors in healthy individuals as in patients (Kusztrits et al., 2021).

These lack of support for the hypotheses in our study is discussed in terms of the complexity of the tDCS protocols (i.e., electrode placements, electrode size, current strength, and durations) that might have limited our tDCS procedure. More importantly, these findings lend further support to the notion that the tDCS electrodes in the treatment montage might not

be primarily stimulating the left DLPFC and left TPC, as shown in Marquardt et al. (2021) study. Moreover, the findings in our study also promote previous research that shows that the success of tDCS depends on how much current flows to the left transverse temporal gyrus (Mondino et al., 2020). However, future research should implement a computer simulation model, based on anatomical scans from participants, to confirm these notions.

Additionally, results in our study supported the suggestion made by Laloyaux et al. (2022) that human noise frequencies make it harder for participants to detect a word embedded in the noise, as opposed to non-human noise frequencies. Even though these findings did not indicate increases in auditory false perceptions, they could suggest a vulnerability to these experiences. Surprisingly, however, the study did only reveal one interaction effect between noise type and degree of expectations that was not in line with Laloyaux et al. (2022). It was speculated that a high expectation of words in the human noise condition created a level of uncertainty regarding the end-word for our participants inatead. However, this needs to be confirmed by future studies that could implement measures of certainty regarding end-words.

4.5 Future Considerations

As previously mentioned, there is a huge variability in the tDCS protocols (i.e., electrode placements, electrode size, current strength, and durations) that might be affecting the outcomes of this study. An alternative approach to gain a deeper understanding of the tDCS effects could be to incorporate other physiological measures such as functional magnetic resonance imaging, magnetic resonance spectroscopy, electroencephalogram, and even genetic profiling. However, this would obviously make the set-up of the experiment more complicated, time-consuming, and expensive. Instead of investigating the complex protocols of tDCS, the current study wants to put forth another, perhaps more achievable

suggestion: tACS. Much like tDCS, tACS delivers a weak electric current using two or more electrodes that supposedly changes the membrane voltage of the underlying neurons (Brunoni et al., 2021). The difference is that tACS produces a low-intensity sinusoidal current with a selected frequency that interacts with the brain's natural neural oscillations (Ruffini et al., 2013). Thus, instead of voltage-controlled currents as in tDCS, tACS produces current-guided electronic circuits that can be adapted to the individuals skin and skull resistances (Brunoni et al., 2021). Moreover, the absence of a clear anode/cathode polarity in tACS, provides larger freedom concerning the direction of the current flow (Brunoni et al., 2021).

However, for tACS to be used as either a therapeutic tool in the treatment of AVH or to mimic these experiences in healthy participants, brain oscillations involved in AVH should be identified. Previous research shows that EEG can be used to establish oscillating frequencies between large neural networks, and / or within cortical networks (Uhlhaas & Singer, 2010). For instance, several dysfunctional brain oscillations in beta-, (12–30 Hz), gamma-, (Uhlhaas & Singer, 2010) and alpha frequencies (Sritharan et al., 2005) were shown in patients with schizophrenia. EEG studies have even provided additional support for a reduced functional connection between the left TPC and DLPFC during AVH (Garrity et al., 2007; Lynall et al., 2010). A generally persistent finding in schizophrenia patients is a reduced alpha frequency (Ahn et al., 2019) between the frontal and temporal lobes (Hinkley et al., 2011). Thus, targeting this frequency with tACS could be a possible treatment approach of AVH.

Once the targeted brain oscillation is established, future research should focus on specific tACS protocols to be used in the treatment of AVH. Only recently have the first well-structured double-blind randomized controlled trials examined the effect of tACS on AVH (Ahn et al., 2019). Ahn et al. (2019) used a three-electrode montage and placed the first electrode on F3 / Fp1 (DLPC), the second on T3 / P3 (TPC), as well as a return electrode on

Cz (midline central). The changes in alpha oscillation were then compared across three conditions: 10-Hz tACS, tDCS and sham. The study showed that tACS was able to increase alpha-oscillations, as compared to tDCS and sham. This in turn was associated with clinical improvements of AVH.

The above information suggests that increasing alpha frequencies between the TPC and DLPFC is a possible treatment option for AVH. Future studies could then attempt to induce similar aberrant brain oscillations in healthy participants. More specifically, the aim could be to modulate the alpha-oscillation frequency between the left TPC and the left DLPFC to induce auditory false perceptions in a non-clinical sample. The effects of tACS could then be established by employing the same SDT as our study. If tACS interferes with alpha oscillation between these two brain regions, then auditory false perceptions should increase. Future studies should, however, investigate both ethical considerations and the specific tACS protocols that are needed to reduce or diminish alpha oscillations. A possible solution is to stimulate higher gamma frequency between these two brain regions to reduce alpha oscillations (Boyle & Frohlich, 2013). A possible control condition could be to use tACS to increase alpha oscillation between the TPC and DLPFC. If tACS increases alpha oscillation between these brain regions, auditory false perceptions should not occur. However, this needs to be confirmed by future studies.

References

Ahn, S., Mellin, J. M., Alagapan, S., Alexander, M. L., Gilmore, J. H., Jarskog, L. F., & Fröhlich, F. (2019). Targeting reduced neural oscillations in patients with schizophrenia by transcranial alternating current stimulation. *Neuroimage*, *186*, 126-136. https://doi.org/10.1016/j.neuroimage.2018.10.056

Alganami, F., Varese, F., Wagstaff, G. F., & Bentall, R. P. (2017). Suggestibility and signal detection performance in hallucination-prone students. *Cogn Neuropsychiatry*, *22*(2), 159-174. https://doi.org/10.1080/13546805.2017.1294056

Allen, P., Amaro, E., Fu, C. H. Y., Williams, S. C. R., Brammer, M. J., Johns, L. C., & McGuire, P. K. (2007). Neural correlates of the misattribution of speech in schizophrenia. *Br J Psychiatry*, *190*(2), 162-169. https://doi.org/10.1192/bjp.bp.106.025700

Ambrus, G. G., Antal, A., & Paulus, W. (2010). Comparing cutaneous perception induced by electrical stimulation using rectangular and round shaped electrodes. *Clin Neurophysiol*, *122*(4), 803-807. https://doi.org/10.1016/j.clinph.2010.08.023

Ambrus, G. G., Zimmer, M., Kincses, Z. T., Harza, I., Kovács, G., Paulus, W., & Antal, A. (2011). The enhancement of cortical excitability over the DLPFC before and during training impairs categorization in the prototype distortion task. *Neuropsychologia*, *49*(7), 1974-1980. https://doi.org/10.1016/j.neuropsychologia.2011.03.026

Andreasen, N. C., & Flaum, M. (1991). Schizophrenia: The Characteristic Symptoms. *Schizophr Bull*, *17*(1), 27-49. https://doi.org/10.1093/schbul/17.1.27

Antal, A., Alekseichuk, I., Bikson, M., Brockmöller, J., Brunoni, A. R., Chen, R., Cohen, L. G., Dowthwaite, G., Ellrich, J., Flöel, A., Fregni, F., George, M. S., Hamilton, R., Haueisen, J., Herrmann, C. S., Hummel, F. C., Lefaucheur, J. P., Liebetanz, D., Loo, C. K., . . . Paulus, W. (2017). Low intensity transcranial electric stimulation: Safety,

ethical, legal regulatory and application guidelines. *Clin Neurophysiol*, *128*(9), 1774-1809. https://doi.org/10.1016/j.clinph.2017.06.001

Antonenko, D., Schubert, F., Bohm, F., Ittermann, B., Aydin, S., Hayek, D., Grittner, U., & Flöel, A. (2017). tDCS-Induced Modulation of GABA Levels and Resting-State Functional Connectivity in Older Adults. *J Neurosci*, *37*(15), 4065-4073. https://doi.org/10.1523/JNEUROSCI.0079-17.2017

Badcock, J. C., & Hugdahl, K. (2012). Cognitive mechanisms of auditory verbal hallucinations in psychotic and non-psychotic groups. *Neurosci Biobehav Rev*, *36*(1), 431-438. https://doi.org/10.1016/j.neubiorev.2011.07.010

Barkus, E., Smallman, R., Royle, N., Barkus, C., Lewis, S., & Rushe, T. (2011). Auditory false perceptions are mediated by psychosis risk factors. *Cogn Neuropsychiatry*, *16*(4), 289-302. https://doi.org/10.1080/13546805.2010.530472

Batsikadze, G., Moliadze, V., Paulus, W., Kuo, M. F., & Nitsche, M. A. (2013). Partially non-linear stimulation intensity-dependent effects of direct current stimulation on motor cortex excitability in humans. *J Physiol*, *591*(7), 1987-2000. https://doi.org/10.1113/jphysiol.2012.249730

Bentall, R. P., & Slade, P. D. (1985). Reality testing and auditory hallucinations: A signal detection analysis. *Br J Clin Psychol*, *24*(3), 159-169. https://doi.org/10.1111/j.2044-8260.1985.tb01331.x

Biggs, D., Hovey, N., Tyson, P. J., & MacDonald, S. (2010). Employer and employment agency attitudes towards employing individuals with mental health needs. *J Ment Health*, *19*(6), 505-516. https://doi.org/10.3109/09638237.2010.507683

Bikson, M., Esmaeilpour, Z., Adair, D., Kronberg, G., Tyler, W. J., Antal, A., Datta, A., Sabel, B. A., Nitsche, M. A., Loo, C., Edwards, D., Ekhtiari, H., Knotkova, H., Woods, A. J., Hampstead, B. M., Badran, B. W., & Peterchev, A. V. (2019).

Transcranial electrical stimulation nomenclature. *Brain Stimul*, *12*(6), 1349-1366. https://doi.org/10.1016/j.brs.2019.07.010

Bikson, M., Grossman, P., Thomas, C., Zannou, A. L., Jiang, J., Adnan, T., Mourdoukoutas, A. P., Kronberg, G., Truong, D., Boggio, P., Brunoni, A. R., Charvet, L., Fregni, F., Fritsch, B., Gillick, B., Hamilton, R. H., Hampstead, B. M., Jankord, R., Kirton, A., . . . Woods, A. J. (2016). Safety of Transcranial Direct Current Stimulation: Evidence Based Update 2016. *Brain Stimul*, *9*(5), 641-661. https://doi.org/10.1016/j.brs.2016.06.004

Binder, J. R., Frost, J. A., Hammeke, T. A., Bellgowan, P. S. F., Springer, J. A., Kaufman, J. N., & Possing, E. T. (2000). Human Temporal Lobe Activation by Speech and Nonspeech Sounds. *Cereb. Cortex*, *10*(5), 512-528. https://doi.org/10.1093/cercor/10.5.512

Bjorkly, S., & Grondahl, P. (2016). Psychosis and violence: Research findings relevant to forensic evaluation of accountability. *Tidsskrift for Norsk Psykologforening*, *53*(3), 212-219. Retrieved from: http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=psyc13&NEWS=N&AN=2016-46928-002.

Boggio, P. S., Zaghi, S., & Fregni, F. (2009). Modulation of emotions associated with images of human pain using anodal transcranial direct current stimulation (tDCS). *Neuropsychologia*, *47*(1), 212-217. https://doi.org/10.1016/j.neuropsychologia.2008.07.022

Bohlken, M. M., Hugdahl, K., & Sommer, I. E. C. (2017). Auditory verbal hallucinations: neuroimaging and treatment. *Psychol. Med*, *47*(2), 199-208. https://doi.org/10.1017/S003329171600115X

Bose, A., Shivakumar, V., Agarwal, S. M., Kalmady, S. V., Shenoy, S., Sreeraj, V. S., Narayanaswamy, J. C., & Venkatasubramanian, G. (2018). Efficacy of fronto-temporal transcranial direct current stimulation for refractory auditory verbal hallucinations in schizophrenia: A randomized, double-blind, sham-controlled study. *Schizophr Res*, *195*, 475-480. https://doi.org/10.1016/j.schres.2017.08.047

Bowie, C. R., & Harvey, P. D. (2006). Cognitive deficits and functional outcome in schizophrenia. *Neuropsychiatr Dis Treat*, *2*(4), 531-536. https://doi.org/10.2147/nedt.2006.2.4.531

Boyle, M. R., & Frohlich, F. (2013). EEG feedback-controlled transcranial alternating current stimulation. *the 6th International IEEE/EMBS Conference on Neural Engineering (NER)* 140-143. https://doi.org/10.1109/NER.2013.6695891.

Bricout, J. C., & Bentley, K. J. (2000). Disability status and perceptions of employability by employers. *Social Work Research*, *24*(2), 87-95. https://doi.org/10.1093/swr/24.2.87

Brookwell, M. L., Bentall, R. P., & Varese, F. (2013). Externalizing biases and hallucinations in source-monitoring, self-monitoring and signal detection studies: a meta-analytic review. *Psychol. Med*, *43*(12), 2465-2475. https://doi.org/10.1017/S0033291712002760

Brunelin, J., d'Amato, T., Brun, P., Bediou, B., Kallel, L., Senn, M., Poulet, E., & Saoud, M. (2006). Impaired verbal source monitoring in schizophrenia: An intermediate trait vulnerability marker? *Schizophr Res*, *89*(1), 287-292. https://doi.org/10.1016/j.schres.2006.08.028

Brunelin, J., Mondino, M., Gassab, L., Haesebaert, F., Gaha, L., Suaud-Chagny, M.-F., Saoud, M., Mechri, A., & Poulet, E. (2012). Examining Transcranial Direct-Current Stimulation (tDCS) as a Treatment for Hallucinations in Schizophrenia. *Am J Psychiatry*, *169*(7), 719-724. https://doi.org/10.1176/appi.ajp.2012.11071091

Brunoni, A. R., Amadera, J., Berbel, B., Volz, M. S., Rizzerio, B. G., & Fregni, F. (2011). A systematic review on reporting and assessment of adverse effects associated with transcranial direct current stimulation. *Int J Neuropsychopharmacol*, *14*(8), 1133-1145. https://doi.org/10.1017/S1461145710001690

Brunoni, A. R., Nitsche, M. A., & Loo, C. K. (2021). *Transcranial Direct Current Stimulation in Neuropsychiatric Disorders : Clinical Principles and Management* (2nd ed.). Springer International Publishing AG. https://doi.org/https://doi.org/10.1007/978-3-030-76136-3

Cheng, G. L. F., & Lee, T. M. C. (2016). Altering risky decision-making: Influence of impulsivity on the neuromodulation of prefrontal cortex. *Soc Neurosci*, *11*(4), 353-364. https://doi.org/10.1080/17470919.2015.1085895

Cheng, P. W. C., Louie, L. L. C., Wong, Y. L., Wong, S. M. C., Leung, W. Y., Nitsche, M. A., & Chan, W. C. (2020). The effects of transcranial direct current stimulation (tDCS) on clinical symptoms in schizophrenia: A systematic review and meta-analysis. *Asian journal of psychiatry*, *53*, 102392-102392. https://doi.org/10.1016/j.ajp.2020.102392

Chhabra, H., Sowmya, S., Sreeraj, V. S., Kalmady, S. V., Shivakumar, V., Amaresha, A. C., Narayanaswamy, J. C., & Venkatasubramanian, G. (2016). Auditory False Perception in Schizophrenia: Development and Validation of Auditory Signal Detection Task. *Asian J Psychiatr*, *24*, 23-27. https://doi.org/10.1016/j.ajp.2016.08.006

Chong, H. Y., Teoh, S. L., Wu, D. B.-C., Kotirum, S., Chiou, C.-F., & Chaiyakunapruk, N. (2016). Global economic burden of schizophrenia: a systematic review. *Neuropsychiatr Dis Treat*, *12*, 357-373. https://doi.org/10.2147/NDT.S96649

Civile, C., Quaglia, S., Waguri, E., Ward, M., McLaren, R., & McLaren, I. P. L. (2021). Using transcranial Direct Current Stimulation (tDCS) to investigate why faces are and

are not special. *Sci Rep*, *11*(1), 4380-4380. https://doi.org/10.1038/s41598-021-83844-3

Clark, V. P., Coffman, B. A., Trumbo, M. C., & Gasparovic, C. (2011). Transcranial direct current stimulation (tDCS) produces localized and specific alterations in neurochemistry: A 1H magnetic resonance spectroscopy study. *Neurosci Lett*, *500*(1), 67-71. https://doi.org/10.1016/j.neulet.2011.05.244

Daalman, K., Boks, M. P. M., Diederen, K. M. J., de Weijer, A. D., Blom, J. D., Kahn, R. S., & Sommer, I. E. C. (2011). The Same or Different? A Phenomenological Comparison of Auditory Verbal Hallucinations in Healthy and Psychotic Individuals. *J Clin Psychiatry*, *72*(3), 320-325. https://doi.org/10.4088/JCP.09m05797yel

Daalman, K., van Zandvoort, M., Bootsman, F., Boks, M., Kahn, R., & Sommer, I. (2011). Auditory verbal hallucinations and cognitive functioning in healthy individuals. *Schizophr Res*, *132*(2), 203-207. https://doi.org/10.1016/j.schres.2011.07.013

Daalman, K., Verkooijen, S., Derks, E. M., Aleman, A., & Sommer, I. E. C. (2012). The influence of semantic top-down processing in auditory verbal hallucinations. *Schizophr Res*, *139*(1-3), 82-86. https://doi.org/10.1016/j.schres.2012.06.005

Datta, A. M. S., Bansal, V. B. S., Diaz, J. B. S., Patel, J. M. S., Reato, D. M. S., & Bikson, M. P. (2009). Gyri-precise head model of transcranial direct current stimulation: Improved spatial focality using a ring electrode versus conventional rectangular pad. *Brain Stimul*, *2*(4), 201-207.e201. https://doi.org/10.1016/j.brs.2009.03.005

de Leede-Smith, S., & Barkus, E. (2013). A comprehensive review of auditory verbal hallucinations: lifetime prevalence, correlates and mechanisms in healthy and clinical individuals. *Front Hum Neurosci*, *7*, 367-367. https://doi.org/10.3389/fnhum.2013.00367

Dedoncker, J., Brunoni, A. R., Baeken, C., & Vanderhasselt, M.-A. (2016). A Systematic Review and Meta-Analysis of the Effects of Transcranial Direct Current Stimulation (tDCS) Over the Dorsolateral Prefrontal Cortex in Healthy and Neuropsychiatric Samples: Influence of Stimulation Parameters. *Brain Stimul*, *9*(4), 501-517. https://doi.org/10.1016/j.brs.2016.04.006

Demjaha, A., Lappin, J. M., Stahl, D., Patel, M. X., MacCabe, J. H., Howes, O. D., Heslin, M., Reininghaus, U. A., Donoghue, K., Lomas, B., Charalambides, M., Onyejiaka, A., Fearon, P., Jones, P., Doody, G., Morgan, C., Dazzan, P., & Murray, R. M. (2017). Antipsychotic treatment resistance in first-episode psychosis: prevalence, subtypes and predictors. *Psychol. Med*, *47*(11), 1981-1989. https://doi.org/10.1017/S0033291717000435

Diederen, K. M. J., Daalman, K., de Weijer, A. D., Neggers, S. F. W., van Gastel, W., Blom, J. D., Kahn, R. S., & Sommer, I. E. C. (2012). Auditory Hallucinations Elicit Similar Brain Activation in Psychotic and Nonpsychotic Individuals. *Schizophr Bull*, *38*(5), 1074-1082. https://doi.org/10.1093/schbul/sbr033

Dmochowski, J. P., Datta, A., Bikson, M., Su, Y., & Parra, L. C. (2011). Optimized multi-electrode stimulation increases focality and intensity at target. *J Neural Eng*, *8*(4), 046011-046011-046016. https://doi.org/10.1088/1741-2560/8/4/046011

Elmer, S., Burkard, M., Renz, B., Meyer, M., & Jancke, L. (2009). Direct current induced short-term modulation of the left dorsolateral prefrontal cortex while learning auditory presented nouns. *Behav Brain Funct*, *5*(1), 29-29. https://doi.org/10.1186/1744-9081-5-29

Evensen, S., Wisloff, T., Lystad, J. U., Bull, H., Ueland, T., & Falkum, E. (2016). Prevalence, Employment Rate, and Cost of Schizophrenia in a High-Income Welfare

Society: A Population-Based Study Using Comprehensive Health and Welfare Registers. *Schizophr Bull*, *42*(2), 476-483. https://doi.org/10.1093/schbul/sbv141

Fasseeh, A., Nemeth, B., Molnar, A., Horvath, M., Koczian, K., Gotze, A., & Kaló, Z. (2017). Indirect Costs and Caregiver Burden of Schizophrenia In European Countries – Results of A Systematic Literature Review. *Value in health*, *20*(9), A712. https://doi.org/10.1016/j.jval.2017.08.1883

Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using GPower 3.1: Tests for correlation and regression analyses. *Behav Res Methods*, *41*(4), 1149-1160. https://doi.org/10.3758/BRM.41.4.1149

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). GPower 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods*, *39*(2), 175-191. https://doi.org/10.3758/BF03193146

Fitzgerald, P. B., McQueen, S., Daskalakis, Z. J., & Hoy, K. E. (2014). A Negative Pilot Study of Daily Bimodal Transcranial Direct Current Stimulation in Schizophrenia. *Brain Stimul*, *7*(6), 813-816. https://doi.org/10.1016/j.brs.2014.08.002

Fregni, F., Boggio, P. S., Mansur, C. G., Wagner, T., Ferreira, M. J. L., Lima, M. C., Rigonatti, S. P., Marcolin, M. A., Freedman, S. D., Nitsche, M. A., & Pascual-Leone, A. (2005). Transcranial direct current stimulation of the unaffected hemisphere in stroke patients. *Neuroreport*, *16*(14), 1551-1555. https://doi.org/10.1097/01.wnr.0000177010.44602.5e

Fujita, J., Takahashi, Y., Nishida, A., Okumura, Y., Ando, S., Kawano, M., Toyohara, K., Sho, N., Minami, T., & Arai, T. (2015). Auditory verbal hallucinations increase the risk for suicide attempts in adolescents with suicidal ideation. *Schizophr Res*, *168*(1), 209-212. https://doi.org/10.1016/j.schres.2015.07.028

Garrison, J. R., Moseley, P., Alderson-Day, B., Smailes, D., Fernyhough, C., & Simons, J. S. (2017). Testing continuum models of psychosis: No reduction in source monitoring ability in healthy individuals prone to auditory hallucinations. *Cortex*, *91*, 197-207. https://doi.org/10.1016/j.cortex.2016.11.011

Garrity, A. G., Pearlson, G. D., McKiernan, K., Lloyd, D., Kiehl, K. A., & Calhoun, V. D. (2007). Aberrant "Default Mode" Functional Connectivity in Schizophrenia. *Am J Psychiatry*, *164*(3), 450-457. https://doi.org/10.1176/ajp.2007.164.3.450

Geddes, J., Freemantle, N., Harrison, P., & Bebbington, P. (2000). Atypical antipsychotics in the treatment of schizophrenia: systematic overview and meta-regression analysis. *British Medical Journal*, *321*(7273), 1371-1371. https://doi.org/https://doi.org/10.1136/bmj.321.7273.1371

Gould, R. A., Mueser, K. T., Bolton, E., Mays, V., & Goff, D. (2001). Cognitive therapy for psychosis in schizophrenia: an effect size analysis. *Schizophr Res*, *48*(2), 335-342. https://doi.org/10.1016/S0920-9964(00)00145-6

Guttesen, L. L., Albert, N., Nordentoft, M., & Hjorthøj, C. (2021). Repetitive transcranial magnetic stimulation and transcranial direct current stimulation for auditory hallucinations in schizophrenia: Systematic review and meta-analysis. *Journal of psychiatric research*, *143*, 163-175. https://doi.org/10.1016/j.jpsychires.2021.09.001

Hinkley, L. B. N., Vinogradov, S., Guggisberg, A. G., Fisher, M., Findlay, A. M., & Nagarajan, S. S. (2011). Clinical Symptoms and Alpha Band Resting-State Functional Connectivity Imaging in Patients With Schizophrenia: Implications for Novel Approaches to Treatment. *Biol Psychiatry*, *70*(12), 1134-1142. https://doi.org/10.1016/j.biopsych.2011.06.029

Hoaglin, D. C., & Iglewicz, B. (1987). Fine-Tuning Some Resistant Rules for Outlier Labeling. *Journal of the American Statistical Association*, *82*(400), 1147-1149. https://doi.org/10.1080/01621459.1987.10478551

Hoffman, R. E., Wu, K., Pittman, B., Cahill, J. D., Hawkins, K. A., Fernandez, T., & Hannestad, J. (2013). Transcranial Magnetic Stimulation of Wernicke's and Right Homologous Sites to Curtail "Voices": A Randomized Trial. *Biol Psychiatry*, *73*(10), 1008-1014. https://doi.org/10.1016/j.biopsych.2013.01.016

Horvath, J. C., Forte, J. D., & Carter, O. (2015). Quantitative Review Finds No Evidence of Cognitive Effects in Healthy Populations From Single-session Transcranial Direct Current Stimulation (tDCS). *Brain Stimul*, *8*(3), 535-550. https://doi.org/10.1016/j.brs.2015.01.400

Hoskin, R., Hunter, M. D., & Woodruff, P. W. R. (2014). The effect of psychological stress and expectation on auditory perception: A signal detection analysis. *Br J Psychol*, *105*(4), 524-546. https://doi.org/10.1111/bjop.12048

Hsu, T.-Y., Juan, C. H., & Tseng, P. (2016). Individual Differences and State-Dependent Responses in Transcranial Direct Current Stimulation. *Front Hum Neurosci*, *10*, 643-643. https://doi.org/10.3389/fnhum.2016.00643

Huang, Y., & Parra, L. C. (2019). Can transcranial electric stimulation with multiple electrodes reach deep targets? *Brain Stimul*, *12*(1), 30-40. https://doi.org/10.1016/j.brs.2018.09.010

Hugdahl, K. (2009). "Hearing voices": Auditory hallucinations as failure of top-down control of bottom-up perceptual processes. *Scand J Psychol*, *50*(6), 553-560. https://doi.org/10.1111/j.1467-9450.2009.00775.x

Hugdahl, K. (2015). Auditory hallucinations: A review of the ERC "VOICE" project. *World J Psychiatry*, *5*(2), 193-209. https://doi.org/10.5498/wjp.v5.i2.193

Hugdahl, K., Loberg, E.-M., Specht, K., Steen, V. M., van Wageningen, H., & Jorgensen, H. A. (2008). Auditory hallucinations in schizophrenia: the role of cognitive, brain structural and genetic disturbances in the left temporal lobe. *Front Hum Neurosci*, *1*, 6-6. https://doi.org/10.3389/neuro.09.006.2007

Hugdahl, K., & Sommer, I. E. (2018). Auditory Verbal Hallucinations in Schizophrenia from a Levels of Explanation Perspective. *Schizophr Bull*, *44*(2), 234-241. https://doi.org/10.1093/schbul/sbx142

Hunter, M. A., Coffman, B. A., Gasparovic, C., Calhoun, V. D., Trumbo, M. C., & Clark, V. P. (2014). Baseline effects of transcranial direct current stimulation on glutamatergic neurotransmission and large-scale network connectivity. *Brain Res*, *1594*, 92-107. https://doi.org/10.1016/j.brainres.2014.09.066

Jacobson, L., Koslowsky, M., & Lavidor, M. (2011). tDCS polarity effects in motor and cognitive domains: a meta-analytical review. *Exp Brain Res*, *216*(1), 1-10. https://doi.org/10.1007/s00221-011-2891-9

Jalali, R., Chowdhury, A., Wilson, M., Miall, R. C., & Galea, J. M. (2018). Neural changes associated with cerebellar tDCS studied using MR spectroscopy. *Exp Brain Res*, *236*(4), 997-1006. https://doi.org/10.1007/s00221-018-5170-1

Jardri, R., Cachia, A., Pins, D., & Thomas, P. (2013). *The Neuroscience of Hallucinations* (1. Aufl.,2013 ed.). New York, NY: Springer-Verlag. https://doi.org/10.1007/978-1-4614-4121-2

Jardri, R., Pouchet, A., Pins, D., & Thomas, P. (2011). Cortical Activations During Auditory Verbal Hallucinations in Schizophrenia: A Coordinate-Based Meta-Analysis. *Am J Psychiatry*, *168*(1), 73-81. https://doi.org/10.1176/appi.ajp.2010.09101522

Johannessen, J. O. (2002). Schizofreni - omfang og betydning. *Tidsskrift for den Norske Lægeforening*. https://tidsskriftet.no/2002/08/tema-schizofreni/schizofreni-omfang-og-betydning

Jones, K. T., & Berryhill, M. E. (2012). Parietal contributions to visual working memory depend on task difficulty. *Front Psychiatry*, *3*, 81-81. https://doi.org/10.3389/fpsyt.2012.00081

Joos, K., De Ridder, D., Van de Heyning, P., & Vanneste, S. (2014). Polarity Specific Suppression Effects of Transcranial Direct Current Stimulation for Tinnitus. *Neural Plast*, *2014*(2014), 930860-930868. https://doi.org/10.1155/2014/930860

Joyce, E. M., & Roiser, J. P. (2007). Cognitive heterogeneity in schizophrenia. *Curr Opin Psychiatry*, *20*(3), 268-272. https://doi.org/10.1097/YCO.0b013e3280ba4975

Kane, J. M., Agid, O., Baldwin, M. L., Howes, O., Lindenmayer, J.-P., Marder, S., Olfson, M., Potkin, S. G., & Correll, C. U. (2019). Clinical Guidance on the Identification and Management of Treatment-Resistant Schizophrenia. *J Clin Psychiatry*, *80*(2). https://doi.org/10.4088/JCP.18com12123

Kane, J. M., & Correll, C. U. (2016). The Role of Clozapine in Treatment-Resistant Schizophrenia. *JAMA Psychiatry*, *73*(3), 187-188. https://doi.org/10.1001/jamapsychiatry.2015.2966

Kim, J., Iwata, Y., Plitman, E., Caravaggio, F., Chung, J. K., Shah, P., Blumberger, D. M., Pollock, B. G., Remington, G., Graff-Guerrero, A., & Gerretsen, P. (2019). A meta-analysis of transcranial direct current stimulation for schizophrenia: "Is more better?". *J Psychiatr Res*, *110*, 117-126. https://doi.org/10.1016/j.jpsychires.2018.12.009

Kim, J.-H., Kim, D.-W., Chang, W. H., Kim, Y.-H., Kim, K., & Im, C.-H. (2014). Inconsistent outcomes of transcranial direct current stimulation may originate from

anatomical differences among individuals: Electric field simulation using individual MRI data. *Neurosci Lett*, *564*, 6-10. https://doi.org/10.1016/j.neulet.2014.01.054

Kim, S., Stephenson, M. C., Morris, P. G., & Jackson, S. R. (2014). tDCS-induced alterations in GABA concentration within primary motor cortex predict motor learning and motor memory: A 7T magnetic resonance spectroscopy study. *NeuroImage (Orlando, Fla.)*, *99*, 237-243. https://doi.org/10.1016/j.neuroimage.2014.05.070

Kinge, J. M., Sælensminde, K., Dieleman, J., Vollset, S. E., & Norheim, O. F. (2017). Economic losses and burden of disease by medical conditions in Norway. *Health Policy*, *121*(6), 691-698. https://doi.org/10.1016/j.healthpol.2017.03.020

Kolb, B., Whishaw, I. Q., & Teskey, G. C. (2019). *An introduction to brain and behavior* (sixth ed.). Worth Publishers.

Kompus, K., Westerhausen, R., & Hugdahl, K. (2011). The "paradoxical" engagement of the primary auditory cortex in patients with auditory verbal hallucinations: A meta-analysis of functional neuroimaging studies. *Neuropsychologia*, *49*(12), 3361-3369. https://doi.org/10.1016/j.neuropsychologia.2011.08.010

Koops, S., Blom, J. D., Bouachmir, O., Slot, M. I., Neggers, B., & Sommer, I. E. (2018). Treating auditory hallucinations with transcranial direct current stimulation in a double-blind, randomized trial. *Schizophr Res*, *201*, 329-336. https://doi.org/10.1016/j.schres.2018.06.010

Koops, S., van Dellen, E., Schutte, M. J. L., Nieuwdorp, W., Neggers, S. F. W., & Sommer, I. E. C. (2016). Theta Burst Transcranial Magnetic Stimulation for Auditory Verbal Hallucinations : Negative Findings From a Double-Blind-Randomized Trial. *Schizophr Bull*, *42*(1), 250-257. https://doi.org/10.1093/schbul/sbv100

Koops, S., van den Brink, H., & Sommer, I. E. C. (2015). Transcranial direct current stimulation as a treatment for auditory hallucinations. *Front Psychol*, *6*, 244-244. https://doi.org/10.3389/fpsyg.2015.00244

Kühn, S., & Gallinat, J. (2012). Quantitative meta-analysis on state and trait aspects of auditory verbal hallucinations in schizophrenia. https://doi.org/10.1093/schbul/sbq152

Kuo, M.-F., & Nitsche, M. A. (2012). Effects of Transcranial Electrical Stimulation on Cognition. *Clin EEG Neurosci*, *43*(3), 192-199. https://doi.org/10.1177/1550059412444975

Kusztrits, I., Marquardt, L., Hugdahl, K., & Hirnstein, M. (2021). Transcranial direct current stimulation (tDCS) enhances internal source monitoring abilities in healthy participants. *PloS one*, *16*(9), e0257010-e0257010. https://doi.org/10.1371/journal.pone.0257010

Kveraga, K., Ghuman, A. S., & Bar, M. (2007). Top-down predictions in the cognitive brain. *Brain Cogn*, *65*(2), 145-168. https://doi.org/10.1016/j.bandc.2007.06.007

Lakens, D. (2013). Calculating and reporting effect sizes to facilitate cumulative science : a practical primer for t-tests and ANOVAs. *Front Psychol*, *4*, 1-12. https://doi.org/10.3389/fpsyg.2013.00863

Laloyaux, J., De Keyser, F., Pinchard, A., Della Libera, C., & Larøi, F. (2019). Testing a model of auditory hallucinations: the role of negative emotions and cognitive resources. *Cogn Neuropsychiatry*, *24*(4), 256-274. https://doi.org/10.1080/13546805.2019.1629895

Laloyaux, J., Hirnstein, M., Specht, K., Giersch, A., & Larøi, F. (2022). Eliciting false auditory perceptions using speech frequencies and semantic priming: a signal detection approach. *Cogn Neuropsychiatry*, 1-18. https://doi.org/10.1080/13546805.2022.2031945

Lansky, L. M., Feinstein, H., & Peterson, J. M. (1988). Demography of handedness in two samples of randomly selected adults (N = 2083). *Neuropsychologia*, *26*(3), 465-477. https://doi.org/10.1016/0028-3932(88)90099-1

Laroi, F. (2012). How do auditory verbal hallucinations in patients differ from those in non-patients? *Front Hum Neurosci*, *6*, 25-25. https://doi.org/10.3389/fnhum.2012.00025

Larøi, F., Van der Linden, M., & Marczewski, P. (2004). The effects of emotional salience, cognitive effort and meta-cognitive beliefs on a reality monitoring task in hallucination-prone subjects. *Br J Clin Psychol*, *43*(3), 221-233. https://doi.org/10.1348/0144665031752970

Lawrie, S. M., Buechel, C., Whalley, H. C., Frith, C. D., Friston, K. J., & Johnstone, E. C. (2002). Reduced frontotemporal functional connectivity in schizophrenia associated with auditory hallucinations. *Biol Psychiatry*, *51*(12), 1008-1011. https://doi.org/10.1016/S0006-3223(02)01316-1

Lennox, B. R., Park, S. B. G., Medley, I., Morris, P. G., & Jones, P. B. (2000). The functional anatomy of auditory hallucinations in schizophrenia. *Psychiatry Res*, *100*(1), 13-20. https://doi.org/10.1016/S0925-4927(00)00068-8

Li, C.-s. R., Chen, M.-c., Yang, Y.-y., Chen, M.-c., & Tsay, P.-k. (2002). Altered performance of schizophrenia patients in an auditory detection and discrimination task: exploring the 'self-monitoring' model of hallucination. *Schizophr Res*, *55*(1), 115-128. https://doi.org/10.1016/S0920-9964(01)00203-1

Lynall, M.-E., Bassett, D. S., Kerwin, R., McKenna, P. J., Kitzbichler, M., Muller, U., & Bullmore, E. (2010). Functional connectivity and brain networks in schizophrenia. *J Neurosci*, *30*(28), 9477-9487. https://doi.org/10.1523/JNEUROSCI.0333-10.2010

Markett, S., Montag, C., Heeren, B., Saryiska, R., Lachmann, B., Weber, B., & Reuter, M. (2015). Voxelwise eigenvector centrality mapping of the human functional

connectome reveals an influence of the catechol-O-methyltransferase val158met polymorphism on the default mode and somatomotor network. *Brain Struct Funct*, *221*(5), 2755-2765. https://doi.org/10.1007/s00429-015-1069-9

Marquardt, L., Kusztrits, I., Craven, A. R., Hugdahl, K., Specht, K., & Hirnstein, M. (2021). A multimodal study of the effects of tDCS on dorsolateral prefrontal and temporo-parietal areas during dichotic listening. *Eur J Neurosci*, *53*(2), 449-459. https://doi.org/10.1111/ejn.14932

Matsumoto, H., & Ugawa, Y. (2017). Adverse events of tDCS and tACS: A review. *Clin Neurophysiol Pract*, *2*, 19-25. https://doi.org/10.1016/j.cnp.2016.12.003

Maxwell, S. E., Delaney, H. D., & Kelley, K. (2017). *Designing experiments and analyzing data: A model comparison perspective. (3rd ed.)*. Routledge.

McCarthy-Jones, S., Trauer, T., Mackinnon, A., Sims, E., Thomas, N., & Copolov, D. L. (2014). A New Phenomenological Survey of Auditory Hallucinations: Evidence for Subtypes and Implications for Theory and Practice. *Schizophr Bull*, *40*(1), 225-235. https://doi.org/10.1093/schbul/sbs156

McGrath, J., Saha, S., Chant, D., & Welham, J. (2008). Schizophrenia: A Concise Overview of Incidence, Prevalence, and Mortality. *Epidemiol Rev*, *30*(1), 67-76. https://doi.org/10.1093/epirev/mxn001

Medina, J., & Cason, S. (2017). No evidential value in samples of transcranial direct current stimulation (tDCS) studies of cognition and working memory in healthy populations. *Cortex*, *94*, 131-141. https://doi.org/10.1016/j.cortex.2017.06.021

Melle, I., Friis, S., Hauff, E., & Vaglum, P. (2000). Social Functioning of Patients With Schizophrenia in High-Income Welfare Societies. *Psychiatr Serv*, *51*(2), 223-228. https://doi.org/10.1176/appi.ps.51.2.223

Michie, P. T., Badcock, J. C., Waters, F. A. V., & Maybery, M. T. (2005). Auditory hallucinations: Failure to inhibit irrelevant memories. *Cogn Neuropsychiatry*, *10*(2), 125-136. https://doi.org/10.1080/13546800344000363

Millier, A., Schmidt, U., Angermeyer, M. C., Chauhan, D., Murthy, V., Toumi, M., & Cadi-Soussi, N. (2014). Humanistic burden in schizophrenia: A literature review. *J Psychiatr Res*, *54*, 85-93. https://doi.org/10.1016/j.jpsychires.2014.03.021

Minhas, P., Bansal, V., Patel, J., Ho, J. S., Diaz, J., Datta, A., & Bikson, M. (2010). Electrodes for high-definition transcutaneous DC stimulation for applications in drug delivery and electrotherapy, including tDCS. *J Neurosci Methods*, *190*(2), 188-197. https://doi.org/10.1016/j.jneumeth.2010.05.007

Minhas, P., Datta, A., & Bikson, M. (2010). Cutaneous perception during tDCS: Role of electrode shape and sponge salinity. *Clin Neurophysiol*, *122*(4), 637-638. https://doi.org/10.1016/j.clinph.2010.09.023

Miranda, P. C., Lomarev, M., & Hallett, M. (2006). Modeling the current distribution during transcranial direct current stimulation. *Clin Neurophysiol*, *117*(7), 1623-1629. https://doi.org/10.1016/j.clinph.2006.04.009

Mondino, M., Fonteneau, C., Simon, L., Dondé, C., Haesebaert, F., Poulet, E., & Brunelin, J. (2020). Advancing clinical response characterization to frontotemporal transcranial direct current stimulation with electric field distribution in patients with schizophrenia and auditory hallucinations: a pilot study. *Eur Arch Psychiatry Clin Neurosci*, *271*(1), 85-92. https://doi.org/10.1007/s00406-020-01149-4

Mondino, M., Haesebaert, F., Poulet, E., Suaud-Chagny, M.-F., & Brunelin, J. (2014). Fronto-temporal transcranial Direct Current Stimulation (tDCS) reduces source-monitoring deficits and auditory hallucinations in patients with schizophrenia. *Schizophr Res*, *161*(2), 515-516. https://doi.org/10.1016/j.schres.2014.10.054

Mondino, M., Jardri, R., Suaud-Chagny, M.-F., Saoud, M., Poulet, E., & Brunelin, J. (2016). Effects of Fronto-Temporal Transcranial Direct Current Stimulation on Auditory Verbal Hallucinations and Resting-State Functional Connectivity of the Left Temporo-Parietal Junction in Patients With Schizophrenia. *Schizophr Bull*, *42*(2), 318-326. https://doi.org/10.1093/schbul/sbv114

Mondino, M., Poulet, E., Suaud-Chagny, M.-F., & Brunelin, J. (2016). Anodal tDCS targeting the left temporo-parietal junction disrupts verbal reality-monitoring. *Neuropsychologia*, *89*, 478-484. https://doi.org/10.1016/j.neuropsychologia.2016.07.022

Morrison, A. P., Wells, A., & Nothard, S. (2000). Cognitive factors in predisposition to auditory and visual hallucinations. *Br J Clin Psychol*, *39*(1), 67-78. https://doi.org/10.1348/014466500163112

Mosayebi Samani, M., Agboada, D., Jamil, A., Kuo, M.-F., & Nitsche, M. A. (2019). Titrating the neuroplastic effects of cathodal transcranial direct current stimulation (tDCS) over the primary motor cortex. *Cortex*, *119*, 350-361. https://doi.org/10.1016/j.cortex.2019.04.016

Moseley, P., Aleman, A., Allen, P., Bell, V., Bless, J., Bortolon, C., Cella, M., Garrison, J., Hugdahl, K., Kozáková, E., Larøi, F., Moffatt, J., Say, N., Smailes, D., Suzuki, M., Toh, W. L., Woodward, T., Zaytseva, Y., Rossell, S., & Fernyhough, C. (2021). Correlates of Hallucinatory Experiences in the General Population: An International Multisite Replication Study. *Psychol Sci*, *32*(7), 1024-1037. https://doi.org/10.1177/0956797620985832

Moseley, P., Fernyhough, C., & Ellison, A. (2014). The role of the superior temporal lobe in auditory false perceptions: A transcranial direct current stimulation study.

Neuropsychologia, *62*, 202-208. https://doi.org/10.1016/j.neuropsychologia.2014.07.032

Nawani, H., Bose, A., Agarwal, S. M., Shivakumar, V., Chhabra, H., Subramaniam, A., Kalmady, S., Narayanaswamy, J. C., & Venkatasubramanian, G. (2014). Modulation of Corollary Discharge Dysfunction in Schizophrenia by tDCS: Preliminary Evidence. *Brain Stimul*, *7*(3), 486-488. https://doi.org/10.1016/j.brs.2014.01.003

Nieuwdorp, W., Koops, S., Somers, M., & Sommer, I. E. C. (2015). Transcranial magnetic stimulation, transcranial direct current stimulation and electroconvulsive therapy for medication-resistant psychosis of schizophrenia. *Curr Opin Psychiatry*, *28*(3), 222-228. https://doi.org/10.1097/YCO.0000000000000156

Nitsche, M. A., Koschack, J., Pohlers, H., Hullemann, S., Paulus, W., & Happe, S. (2012). Effects of frontal transcranial direct current stimulation on emotional state and processing in healthy humans. *Front Psychiatry*, *3*, 58-58. https://doi.org/10.3389/fpsyt.2012.00058

Nitsche, M. A., & Paulus, W. (2000). Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation. *J Physiol*, *527*(3), 633-639. https://doi.org/10.1111/j.1469-7793.2000.t01-1-00633.x

Nitsche, M. A. M. D., Cohen, L. G. M. D., Wassermann, E. M. M. D., Priori, A. M. D. P., Lang, N. M. D., Antal, A. P., Paulus, W. M. D., Hummel, F. M. D., Boggio, P. S. P., Fregni, F. M. D. P., & Pascual-Leone, A. M. D. P. (2008). Transcranial direct current stimulation: State of the art 2008. *Brain Stimul*, *1*(3), 206-223. https://doi.org/10.1016/j.brs.2008.06.004

Norsk helseinformatikk. (2021, 19 April). *Schizofreni - omfang og betydning*. https://nhi.no/sykdommer/psykisk-helse/schizofreni/schizofreni-forekomst/?page=all

Ohn, S. H., Park, C.-I., Yoo, W.-K., Ko, M.-H., Choi, K. P., Kim, G.-M., Lee, Y. T., & Kim, Y.-H. (2008). Time-dependent effect of transcranial direct current stimulation on the enhancement of working memory. *Neuroreport*, *19*(1), 43-47. https://doi.org/10.1097/WNR.0b013e3282f2adfd

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, *9*(1), 97-113. https://doi.org/10.1016/0028-3932(71)90067-4

Opitz, A., Paulus, W., Will, S., Antunes, A., & Thielscher, A. (2015). Determinants of the electric field during transcranial direct current stimulation. *Neuroimage*, *109*, 140-150. https://doi.org/10.1016/j.neuroimage.2015.01.033

Parlikar, R., Bose, A., & Venkatasubramanian, G. (2019). Schizophrenia and Corollary Discharge: A Neuroscientific Overview and Translational Implications. *Clin Psychopharmacol Neurosci*, *17*(2), 170-182. https://doi.org/10.9758/cpn.2019.17.2.170

Paulik, G., Badcock, J. C., & Maybery, M. T. (2007). Poor intentional inhibition in individuals predisposed to hallucinations. *Cogn Neuropsychiatry*, *12*(5), 457-470. https://doi.org/10.1080/13546800701394329

Plewnia, C., Zwissler, B., Längst, I., Maurer, B., Giel, K., & Krüger, R. (2013). Effects of transcranial direct current stimulation (tDCS) on executive functions: Influence of COMT Val/Met polymorphism. *Cortex*, *49*(7), 1801-1807. https://doi.org/10.1016/j.cortex.2012.11.002

Pondé, P. H., de Sena, E. P., Camprodon, J. A., de Araújo, A. N., Neto, M. F., DiBiasi, M., Baptista, A. F., Moura, L. M., & Cosmo, C. (2017). Use of transcranial direct current stimulation for the treatment of auditory hallucinations of schizophrenia - a systematic review. *Neuropsychiatr Dis Treat*, *13*, 347-355. https://doi.org/10.2147/NDT.S122016

Purves, D., & Brannon, E. M. (2013). *Principles of cognitive neuroscience* (2nd ed.). Sinauer Associates.

Quam, R. M., Martínez, I., Rosa, M., & Arsuaga, J. L. (2017). Evolution of Hearing and Language in Fossil Hominins. In (pp. 201-231). Cham: Springer International Publishing. https://doi.org/10.1007/978-3-319-59478-1_8

Rakesh, G., Shivakumar, V., Subramaniam, A., Nawani, H., Amaresha, A. C., Narayanaswamy, J. C., & Venkatasubramanian, G. (2013). Monotherapy with tDCS for Schizophrenia: A Case Report. *Brain Stimul*, *6*(4), 708-709. https://doi.org/10.1016/j.brs.2013.01.007

Rasmussen, T., & Milner, B. (1977). THE ROLE OF EARLY LEFT-BRAIN INJURY IN DETERMINING LATERALIZATION OF CEREBRAL SPEECH FUNCTIONS. *Ann N Y Acad Sci*, *299*(1), 355-369. https://doi.org/10.1111/j.1749-6632.1977.tb41921.x

Ruffini, G., Wendling, F., Merlet, I., Molaee-Ardekani, B., Mekonnen, A., Salvador, R., Soria-Frisch, A., Grau, C., Dunne, S., & Miranda, P. C. (2013). Transcranial Current Brain Stimulation (tCS): Models and Technologies. *IEEE Trans Neural Syst Rehabil Eng*, *21*(3), 333-345. https://doi.org/10.1109/TNSRE.2012.2200046

Sher, L., & Kahn, R. S. (2019). Suicide in Schizophrenia: An Educational Overview. *Medicina*, *55*(7), 361. https://www.mdpi.com/1648-9144/55/7/361

Shergill, S. S., Murray, R. M., & McGuire, P. K. (1998). Auditory hallucinations: a review of psychological treatments. *Schizophr Res*, *32*(3), 137-150. https://doi.org/10.1016/S0920-9964(98)00052-8

Shiozawa, P., da Silva, M. E., Cordeiro, Q., Fregni, F., & Brunoni, A. R. (2013). Transcranial Direct Current Stimulation (tDCS) for the Treatment of Persistent Visual and

Auditory Hallucinations in Schizophrenia: A Case Study. *Brain Stimul*, *6*(5), 831-833. https://doi.org/10.1016/j.brs.2013.03.003

Simons, C. J., Tracy, D. K., Sanghera, K. K., O'Daly, O., Gilleen, J., Dominguez, M. D., Krabbendam, L., & Shergill, S. S. (2010). Functional magnetic resonance imaging of inner speech in schizophrenia. *Biological psychiatry (1969)*, *67*(3), 232-237. https://doi.org/https://doi.org/10.1016/j.biopsych.2009.09.007

Soriano, M. F., Jiménez, J. F., Román, P., & Bajo, M. T. (2009). Inhibitory processes in memory are impaired in schizophrenia: Evidence from retrieval induced forgetting. *Br J Psychol*, *100*(4), 661-673. https://doi.org/10.1348/000712609X418912

Sritharan, A., Line, P., Sergejew, A., Silberstein, R., Egan, G., & Copolov, D. (2005). EEG coherence measures during auditory hallucinations in schizophrenia. *Psychiatry Res*, *136*(2), 189-200. https://doi.org/10.1016/j.psychres.2005.05.010

Stagg, C. J., Best, J. G., Stephenson, M. C., O'Shea, J., Wylezinska, M., Kincses, Z. T., Morris, P. G., Matthews, P. M., & Johansen-Berg, H. (2009). Polarity-Sensitive Modulation of Cortical Neurotransmitters by Transcranial Stimulation. *J Neurosci*, *29*(16), 5202-5206. https://doi.org/10.1523/JNEUROSCI.4432-08.2009

Stanislaw, H., & Todorov, N. (1999). Calculation of signal detection theory measures. *Behavior research methods, instruments, & computers*, *31(1)*, 137-149. https://doi.org/https://doi.org/10.3758/BF03207704

Stephane, M., Kuskowski, M., McClannahan, K., Surerus, C., & Nelson, K. (2010). Evaluation of speech misattribution bias in schizophrenia. *Psychol. Med*, *40*(5), 741-748. https://doi.org/10.1017/S003329170999081X

Stępnicki, P., Kondej, M., & Kaczor, A. A. (2018). Current Concepts and Treatments of Schizophrenia. *Molecules*, *23*(8), 2087. https://doi.org/10.3390/molecules23082087

Tandon, R., Keshavan, M. S., & Nasrallah, H. A. (2008). Schizophrenia, "Just the Facts" What we know in 2008. 2. Epidemiology and etiology. *Schizophr Res*, *102*(1), 1-18. https://doi.org/10.1016/j.schres.2008.04.011

Thair, H., Holloway, A. L., Newport, R., & Smith, A. D. (2017). Transcranial Direct Current Stimulation (tDCS): A Beginner's Guide for Design and Implementation. *Front Neurosci*, *11*, 641-641. https://doi.org/10.3389/fnins.2017.00641

Thomas, E. H. X., Rossell, S. L., Tan, E. J., Neill, E., Van Rheenen, T. E., Carruthers, S. P., Sumner, P. J., Louise, S., Bozaoglu, K., & Gurvich, C. (2019). Do schizotypy dimensions reflect the symptoms of schizophrenia? *Aust N Z J Psychiatry*, *53*(3), 236-247. https://doi.org/10.1177/0004867418769746

Thornicroft, G. P., Brohan, E. M., Rose, D. P., Sartorius, N. P., & Leese, M. P. (2009). Global pattern of experienced and anticipated discrimination against people with schizophrenia: a cross-sectional survey. *Lancet*, *373*(9661), 408-415. https://doi.org/10.1016/S0140-6736(08)61817-6

Turi, Z., Ambrus, G. G., Ho, K.-A., Sengupta, T., Paulus, W., & Antal, A. (2014). When Size Matters: Large Electrodes Induce Greater Stimulation-related Cutaneous Discomfort Than Smaller Electrodes at Equivalent Current Density. *Brain Stimul*, *7*(3), 460-467. https://doi.org/10.1016/j.brs.2014.01.059

Uhlhaas, P. J., & Singer, W. (2010). Abnormal neural oscillations and synchrony in schizophrenia. *Nat Rev Neurosci*, *11*(2), 100-113. https://doi.org/10.1038/nrn2774

Vercammen, A., & Aleman, A. (2010). Semantic Expectations Can Induce False Perceptions in Hallucination-Prone Individuals. *Schizophr Bull*, *36*(1), 151-156. https://doi.org/10.1093/schbul/sbn063

Waters, F., Allen, P., Aleman, A., Fernyhough, C., Woodward, T. S., Badcock, J. C., Barkus, E., Johns, L., Varese, F., Menon, M., Vercammen, A., & Laroi, F. (2012). Auditory

Hallucinations in Schizophrenia and Nonschizophrenia Populations: A Review and Integrated Model of Cognitive Mechanisms. *Schizophr Bull*, *38*(4), 683-692. https://doi.org/10.1093/schbul/sbs045

Waters, F., Badcock, J., Michie, P., & Maybery, M. (2006). Auditory hallucinations in schizophrenia: Intrusive thoughts and forgotten memories. *Cogn Neuropsychiatry*, *11*(1), 65-83. https://doi.org/10.1080/13546800444000191

Waters, F. A. V., Badcock, J. C., Maybery, M. T., & Michie, P. T. (2003). Inhibition in schizophrenia: association with auditory hallucinations. *Schizophr Res*, *62*(3), 275-280. https://doi.org/10.1016/S0920-9964(02)00358-4

Weidacker, K., Weidemann, C. T., Boy, F., & Johnston, S. J. (2016). Cathodal tDCS improves task performance in participants high in Coldheartedness. *Clin Neurophysiol*, *127*(9), 3102-3109. https://doi.org/10.1016/j.clinph.2016.05.274

Whiteford, H. A. P., Degenhardt, L. P., Rehm, J. P., Baxter, A. J. M. P. H., Ferrari, A. J. B., Erskine, H. E. B., Charlson, F. J. M. P. H., Norman, R. E. P., Flaxman, A. D. P., Johns, N. B. A., Burstein, R. B. A., Murray, C. J. L. P., & Vos, T. P. (2013). Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. *Lancet*, *382*(9904), 1575-1586. https://doi.org/10.1016/S0140-6736(13)61611-6

Woods, A. J., Antal, A., Bikson, M., Boggio, P. S., Brunoni, A. R., Celnik, P., Cohen, L. G., Fregni, F., Herrmann, C. S., Kappenman, E. S., Knotkova, H., Liebetanz, D., Miniussi, C., Miranda, P. C., Paulus, W., Priori, A., Reato, D., Stagg, C., Wenderoth, N., & Nitsche, M. A. (2015). A technical guide to tDCS, and related non-invasive brain stimulation tools. *Clin Neurophysiol*, *127*(2), 1031-1048. https://doi.org/10.1016/j.clinph.2015.11.012

World Health Organization. (2019). *International statistical classification of diseases and related health problems* (11 ed.). https://doi.org/ https://icd.who.int/

Yang, K., Longo, L., Narita, Z., Cascella, N., Nucifora, F. C., Coughlin, J. M., Nestadt, G., Sedlak, T. W., Mihaljevic, M., & Wang, M. (2021). A multimodal study of a first episode psychosis cohort: potential markers of antipsychotic treatment resistance. *Molecular psychiatry*, 1-8. https://doi.org/: https://doi.org/10.1101/2021.05.03.442450

Yee, L., Korner, A. J., McSwiggan, S., Meares, R. A., & Stevenson, J. (2005). Persistent hallucinosis in borderline personality disorder. *Compr Psychiatry*, *46*(2), 147-154. https://doi.org/10.1016/j.comppsych.2004.07.032

Yeom, S.-K., Fazli, S., Müller, K.-R., & Lee, S.-W. (2014). An efficient ERP-based brain-computer interface using random set presentation and face familiarity. *PLoS One*, *9*(11), e111157-e111157. https://doi.org/10.1371/journal.pone.0111157

Appendix A

A1

Means (%) and (standard deviation) for hit rate total and false alarm rate total across all stimulation conditions (real and sham) and electrode montages (treatment montage and AVH montage)

	Hit rate total (%)		**False alarm rate total (%)**	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	85.13 (6.79)	84.19 (8.01)	6.73 (6.59)	6.60 (8.86)
Montage 2	86.58 (7.22)	87.66 (6.08)	8.51 (8.02)	8.45 (8.77)
Total	85.83 (7.00)	85.88 (7.30)	7.60 (7.33)	7.50 (8.70)

A2

*Means and (standard deviation) for sensitivity (*A'*) total and response bias (β) total across all stimulation conditions (real and sham) and electrode montages (treatment montage and AVH montage)*

	A' total		**β total**	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	.94 (.03)	.94 (.03)	2.80 (1.58)	3.24 (1.88)
Montage 2	.94 (.02)	.94 (.02)	2.52 (1.95)	2.41 (1.73)
Total	.94 (.03)	.94 (.03)	2.67 (1.76)	2.84 (1.85)

Appendix B

B1

Means are shown as percentages and (standard deviation) for hit rates in the noise condition, stimulation conditions and electrode placements.

	Hit rates (%)			
	Human noise		Non-Human noise	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	76.75 (8.50)	76.24 (9.28)	90.78 (8.32)	89.06 (7.87)
Montage 2	79.46 (10.35)	79.64 (9.93)	90.63 (6.93)	92.07 (3.08)
Total	78.07 (9.48)	77.89 (9.69)	90.70 (7.63)	90.52 (6.18)

B2

Means are shown as percentages and (standard deviation) for false alarm rates in the noise condition, stimulation conditions and electrode placements.

	False alarm rates (%)			
	Human noise		Non-Human noise	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	9.74 (7.94)	9.36 (9.75)	6.92 (4.68)	7.69 (7.42)
Montage 2	12.30 (11.34)	11.08 (9.87)	7.84 (5.96)	9.05 (7.89)
Total	10.99 (9.76)	10.18 (9.78)	7.37 (5.32)	8.36 (7.63)

B3

Means and (standard deviation) for perceptual sensitivity (A') in the noise condition, stimulation condition and electrode placement

	A'			
	Human noise		Non-Human noise	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	.90 (.04)	.90 (.04)	.96 (.03)	.95 (.03)
Montage 2	.91 (.03)	.91 (.03)	.95 (.02)	.95 (.02)
Total	.90 (.04)	.91 (.04)	.95 (.02)	.95 (.03)

B4

Means and (standard deviation) for response bias (β) in the noise condition, stimulation condition and electrode placement

	β			
	Human noise		Non-Human noise	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	2.31 (1.00)	2.47 (1.08)	1.35 (.70)	1.56 (.83)
Montage 2	1.99 (1.15)	2.14 (1.19)	1.34 (.75)	1.18 (.56)
Total	2.15 (1.08)	2.31 (1.14)	1.35 (.72)	1.18 (.56)

Appendix C

C1

Means are shown as percentages and (standard deviation) for hit rates in the degree of expectation condition, stimulation conditions and electrode placements.

	Hit rates (%)			
	Low expectation		High expectation	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	84.27 (9.11)	82.39 (10.87)	85.30 (6.70)	85.47 (7.47)
Montage 2	85.41 (7.83)	86.49 (6.18)	86.49 (7.61)	86.85 (5.77)
Total	84.82 (8.47)	84.39 (9.08)	85.88 (7.13)	86.14 (6.69)

C2

Means are shown as percentages and (standard deviation) for false alarm rates in the degree of expectation condition, stimulation conditions and electrode placements.

	False alarm rates (%)			
	Low expectancy		High expectancy	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	8.33 (5.89)	7.82 (7.05)	8.33 (6.82)	8.59 (9.452)
Montage 2	10.27 (9.12)	9.46 (8.48)	9.19 (6.62)	10.27 (8.33)
Total	9.28 (7.65)	8.62 (7.77)	8.75 (6.69)	9.40 (8.94)

C3

Means and (standard deviation) for perceptual sensitivity (A') in the degree of expectation condition, stimulation condition and electrode placement

	A'			
	Low expectancy		High expectancy	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	.93 (.03)	.93 (.03)	.93 (.03)	.94 (.03)
Montage 2	.93 (.03)	.94 (.02)	.94 (.02)	.93 (.03)
Total	.93 (.03)	.94 (.03)	.94 (.03)	.94 (.03)

C4

Means and (standard deviation) for response bias (β) in the degree of expectation condition, stimulation condition and electrode placement

	β			
	Low expectancy		High expectancy	
	Real tDCS	Sham tDCS	Real tDCS	Sham tDCS
Montage 1	1.85 (.94)	2.11 (1.09)	1.81 (.82)	1.87 (.90)
Montage 2	1.73 (1.01)	1.72 (.88)	1.68 (.95)	1.60 (.88)
Total	1.79 (.97)	1.92 (1.00)	1.75 (.88)	1.74 (.90)