Discrimination of Auditory Signals in Schizotypy

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Statement of Sources

I declare that this thesis is my own work and that, to the best of my knowledge and belief, it does not contain material from published sources without proper acknowledgement, nor does it contain material which has been accepted for the award of any other higher degree or graduate diploma in any university.

Signed:

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Abstract

The resting state hypothesis is a recent theoretical account of auditory verbal (AVH) hallucinations in schizophrenia, which is an alternative to the conventional explanation of the forward model. This study aimed to test one component of the hypothesis, termed the rest-stimulus interaction, using electroencephalography. Using schizotypy as a proxy for schizophrenia, 28 psychology students were recruited into high/low schizotypy groups. Event-related potentials (ERP) were recorded under two passive auditory conditions that specifically excluded the motor component associated with speaking in real time and that is intrinsic to the forward model. ERP peaks N1 and P2 were analysed using a hierarchical regression approach, with schizotypy and hallucination experience separately tested as predictors and controlling for depression, anxiety, and stress. Schizotypy was associated with a difference in the amplitude of the initial attentional response (represented by N1) between the two conditions, but in the opposite direction to that predicted. This finding suggests that there may be an alternative mechanism to both the resting state hypothesis and the forward model. However, as expected, both schizotypy and hallucination experience predicted a difference in forward processing (represented by P2 amplitude) between the conditions. This finding supports the resting state hypothesis. These results provide initial support for the rest-stimulus interaction and present some challenge to the forward model. Future research needs to replicate these findings in a clinical population and include measurement of resting brain activity.

Schizophrenia is a complex and heterogeneous syndrome, experienced by approximately 0.5% of the Australian population (Saha, Chant, Welham, & McGrath, 2005). Auditory verbal hallucinations (AVH) are perhaps the most characteristic and distressing symptom, with more than 70% of individuals diagnosed with schizophrenia estimated to hear hallucinatory voices (Mawson, Cohen, & Berry, 2010). Generally negative, critical, and persistent, AVH frequently cause considerable distress and disruption to daily life, with 'command' hallucinations (telling the person to harm themselves or another person) considered a primary driving force for the elevated rates of suicide and self-harm associated with schizophrenia (Hor & Taylor, 2010). Current treatments may ameliorate some symptoms but are unable to completely control schizophrenia and medication may even be counter-indicated for some patients (Harrow & Jobe, 2007). This poor outlook is due to an inadequate understanding of the mechanisms involved in AVH and schizophrenia in the brain. This study examines a potential mechanism involved in AVH, the neural correlates of the misattribution of auditory stimuli.

Models of AVH

Historically, theoretical accounts of AVH have largely been concerned with defining the location and mechanics of processing and integrating auditory stimuli in the brain (Javitt, 2009). Generally, AVH have been conceptualised as resulting either from 'bottom-up' processes (how a percept is constructed from incoming auditory stimuli) or from 'top-down' processes (how the percept is interpreted through incorporation of previous knowledge or experience), or a combination of both. An example of a bottom-up model concerns a particular electrophysiological signal known as mismatch negativity (MMN), which arises in response to the auditory presentation of an unexpected stimulus (varying in type, intensity, or frequency) in a

sequence of repetitive stimuli. MMN is a preattentive auditory change-detection response, occurring before the stimulus reaches conscious awareness (Javitt, 2000). In schizophrenia, reduced MMN has consistently been found in response to speech sounds as well as simple tones and has been associated with poor auditory perception, discrimination, and working memory, with deficits worsening with illness chronicity (Javitt, 2000, 2009; Näätänen & Kähkönen, 2009).

The Forward Model

Several top-down models are based on the proposition that monitoring of the source of a sensory signal is impaired in schizophrenia, due to abnormalities in a mechanism known as the forward model (Ditman & Kuperberg, 2009; Wang, Metzak, & Woodward, 2011). In a healthy individual, prediction of the sensory sequelae of the person's own actions is thought to be accomplished by a copy of the motor signal (termed the efference copy) being sent directly to the relevant somatosensory cortex (Ford & Mathalon, 2005; Figure 1). The efference copy arrives before the sensory signal that results from the motor movement. In a process known as predictive coding, arrival of the efference copy produces an electrical discharge in the sensory cortex (termed corollary discharge). This has the effect of attenuating the response to the later-arriving sensory signal (Feinberg & Guazzelli, 1999). However, if there are delays in conduction of the efference copy, predictive coding and corollary discharge cannot occur. Thus, the sensory consequences of the brain's own action are not suppressed and therefore may be mistakenly processed as and attributed to an external agency (Stephane, Friston, & Frith, 2009).

Applied to AVH in schizophrenia, disruption of functional connectivity between frontal and auditory areas has been hypothesised to cause delays to corollary discharge, so that the brain's own auditory stimulus (thought) invokes

higher order processing and is misattributed to the speech of another person (Wang, et al., 2011; Whitford, Ford, Mathalon, Kubicki, & Shenton, 2012). Consistent empirical evidence has recently accumulated in support of the forward model and a number of studies (discussed below) have focused on the experience of AVH in schizophrenia.

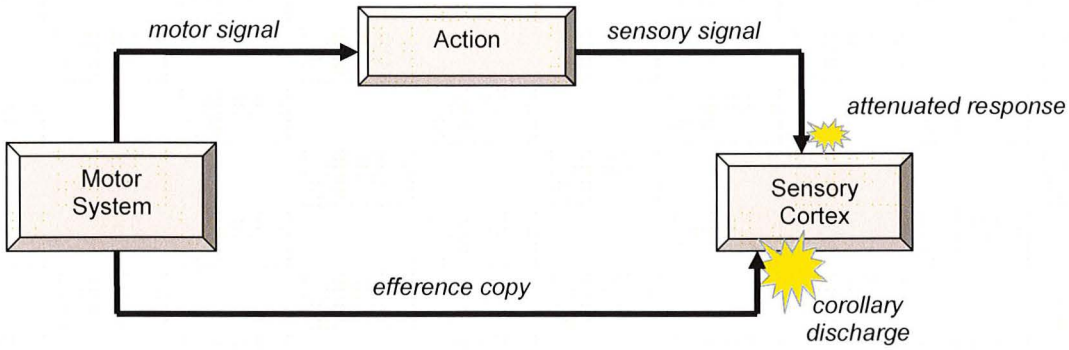

Figure 1. Schematic representation of corollary discharge in the forward model.

The difficulty of tickling oneself is a familiar instance of the brain's suppression of the sensory sequelae its own actions. A study conducted by Blakemore, Wolpert, and Frith (2000) with a healthy sample found that self-administered tactile stimuli resulted in significantly less tickly sensations than the same stimuli applied robotically. Using functional magnetic resonance imaging (fMRI) with healthy participants, Blakemore et al. implicated the right anterior cerebellum in generating the signal that attenuated the response of the somatosensory and anterior cingulate cortices to self-administered tactile stimuli. The researchers then examined sensory suppression and its mechanisms in a clinical psychosis sample. They found that whereas healthy controls and psychosis patients without AVH and/or delusions of control reported that self-induced tactile sensation was less intense, tickly, and pleasant than experimenter-induced sensation, psychosis patients with AVH and/or delusions of control reported no such decrease. The authors

concluded that this failure to suppress perception of self-produced sensation suggests an abnormal forward model mechanism (Blakemore et al.).

Convergently, other researchers were using electroencephalography (EEG) to study forward model deficits in schizophrenia. In an EEG auditory event-related potential (ERP) waveform, the first negative peak, approximately 100 ms post-stimulus, is known as N1 or N100 and is generally taken to reflect the initial, preconscious, attentional response in primary auditory cortex to the physical features of a sound stimulus as salient and requiring further processing (Bösel & Tamm, 2003; Näätänen, Kujala, & Winkler, 2011). The second positive peak in the ERP waveform, at approximately 200 ms post-stimulus, is known as P2 or P200 and is generally taken to represent an early stage of encoding and forward processing (passing the signal to secondary auditory cortex to begin processing its meaning; Bösel & Tamm). Latency and amplitude of both peaks are closely related to volume and intensity of the auditory stimulus (Bösel & Tamm) and amplitude of the auditory N1/P2 complex has been found to be maximal at fronto-central electrode sites (Tremblay, Kraus, McGee, Ponton, & Otis, 2001). The N1 and P2 peaks have consistently been found to be reduced in individuals with schizophrenia (Foxe et al., 2011; Salisbury, Collins, & McCarley, 2010) and also to have a high heritability index (Ahveninen et al., 2006).

Using EEG to study the N1 response, Judith Ford and colleagues conducted a series of studies to examine the role of sensory suppression in AVH. In healthy controls, they found that N1 amplitude was smaller in response to an actively spoken vowel ("Talk" condition") than in response to a passively heard vowel ("Listen" condition), but that this difference was not found in schizophrenia (Ford, Mathalon, Heinks, et al., 2001; Ford, Roach, Faustman, & Mathalon, 2007). A related study

observed that in healthy controls N1 amplitude was reduced in response to deliberate inner speech compared to silence but, again, not in schizophrenia (Ford, Mathalon, Kalba, et al., 2001). These results were taken to support the notion of a deficit in suppression of self-generated signals in AVH due to a failure of corollary discharge. Agency (being the producer of the sound) and expectancy (knowing what sound to expect) might offer alternative explanations for the suppression of N1 amplitude, but a further study found that the effect of these was too weak to account for reduced suppression in both control and schizophrenia groups and did not account for the differences between the groups in N1 suppression (Ford, Gray, Faustman, Roach, & Mathalon, 2007). From these and comparable studies, Ford's team have concluded that an abnormal cortical response to a self-generated auditory stimulus in schizophrenia may be associated with a dysfunction of corollary discharge and that, consequently, a person's spontaneous internal dialogue may be misattributed to an external source (Ford, Perez, & Mathalon, 2012).

Other researchers have conducted investigations of white matter through diffusion tensor imaging (DTI). DTI is a magnetic resonance imaging technique measuring the nonrandom movement of water molecules in soft tissue (Jones & Leemans, 2011) and has been used to assess axonal (white matter) connectivity between distributed cortical (grey matter) regions (Whitford, Kubicki, & Shenton, 2011). Clear evidence of volume deficits and compromised signalling in white matter tracts have been cited in support of the thesis that schizophrenia arises from faulty connectivity (Friston & Frith, 1995; Whitford, Kubicki, et al.). For example, in a study involving individuals with schizophrenia, first-degree relatives, and healthy controls, reduced integrity of the arcuate fasciculus was linearly associated with predisposition to positive symptoms of psychosis (Knöchel et al., 2012).

Another study used DTI analysis of the arcuate fasciculus to predict cortical suppression (represented by N1 amplitude) in response to hearing a recorded syllable played back at different latencies following a button-press (Whitford, Mathalon, et al., 2011). This study found that immediate delivery of the auditory stimulus was associated with abnormal suppression in the schizophrenia group, consistent with previous studies. However, there was no difference from the control group following a 50 millisecond delay and the extent to which N1 suppression became normalised correlated with the extent of compromised integrity in the arcuate fasciculus. The study concluded that patients with schizophrenia experience delayed corollary discharge due to white matter deficits (Whitford, Mathalon, et al.).

Despite these findings, as pointed out by Konrad and Winterer (2008), it is by no means clear whether defects in white matter tracts constitute a primary factor in schizophrenia or are secondary to dysfunction in grey matter structures. Indeed, a recent review by leaders in the field concluded that the anomalies that have been identified across a wide range of research studies are so diverse that a single pathophysiology underlying schizophrenia cannot be specified (Mathalon & Ford, 2012).

Efforts have been made to integrate bottom-up and top-down approaches. Most recently, Waters et al. (2012) proposed an integrated model of cognitive mechanisms. According to this, an internal auditory stimulus (e.g., inner speech) exceeds the threshold for perception (bottom-up) in the auditory cortex due to hyperactivation of language-related areas and, due to the intensity of the resultant sensory experience, the stimulus is processed and interpreted (top-down) as externally generated. The model suggests that the form and content of an auditory experience is influenced by factors such as attention, emotion, and prior experience,

while meaning is mediated by state and trait characteristics including, for example, negative affect, poor insight, and delusional beliefs (Waters et al.). However, process-driven models offer explanations of specific aspects of abnormal auditory processing in AVH but fail to account for key characteristics of auditory hallucinations. According to Northoff and Qin (2011), process theories have not accounted for why anomalies such as auditory cortex hyperactivation or failure of N1 suppression should occur at all, why hallucinatory voices have consistent characters but do not sound like the person's own voice, and why AVH content is overwhelmingly self-related.

A Default Mode of Brain Function

The increasing sophistication of neuroimaging of the brain has encouraged systems-level approaches to modelling cortical functioning. Of particular relevance is the identification of neural circuits that regularly covary in activation temporally, due either to intrinsic fluctuation or in response to an applied stimulus (Fox et al., 2005; Raichle et al., 2001). Perhaps the most exciting discovery was the recognition of a set of midline regions that spontaneously and coherently anticorrrelate with an increase or decrease of activity in task-related circuits (Shulman et al., 1997). The observed increase in cortical activity when the brain was not engaged in task-related activity was taken to represent a baseline state, consequently becoming known as the default mode and the associated regions as the default mode network (DMN; Raichle et al.). Researchers also found that maintenance of the at-rest state consumes up to 80% of the brain's metabolic resources, while less than 5% more has been accounted for by task-related processing (Raichle & Mintun, 2006). Many studies since have confirmed the existence of the DMN (see Whitfield-Gabrieli & Ford, 2012, for a review) and, unexpectedly, it appears that the brain principally engages in intrinsic,

highly organised, and functional activity, and that it is process-oriented evoked activity that is atypical (Damoiseaux et al., 2006; Rosazza & Minati, 2011).

The DMN has been robustly associated with self-referential processing, stimulus-independent thought, affective control, episodic memory, and the maintenance of conscious awareness (Gusnard & Raichle, 2001; Philippi, 2011). It appears to be a critical control system performing integrative, moderating, and mediatory operations across the cortex, including modulating attentional processes and coordinating task-related processing (Damoiseaux et al, 2006; Fox et al., 2005). Aberrant functioning of the DMN has been strongly associated with a range of psychopathologies, generating several excellent reviews and meta-analyses (especially, Broyd et al., 2008; Rosazza & Minati, 2011; Whitfield-Gabrieli & Ford, 2012), and a number of fMRI studies have provided evidence of hyperactivation and hyperconnectivity of the DMN in schizophrenia.

For example, in schizophrenia compared to healthy controls, poor performance on working memory tasks has been associated with reduced suppression of DMN activation (Pomerol-Clotet et al., 2008; Whitfield-Gabrieli et al., 2009). Another study revealed differences in patterns of regional cortical activation between a schizophrenia group and healthy controls in response to an auditory oddball task and rest condition, leading to hyperactivation of the auditory cortex in schizophrenia being attributed to the influence of the DMN (Swanson et al., 2010). Again, a study of resting-state networks showed that individuals with AVH, compared to healthy controls, exhibited hyperactivity in speech-related areas of the temporal lobe and reduced connectivity between the auditory cortex and the anterior cingulum and precuneus (Wolf et al., 2011). The report concluded that AVH result from misattribution of inner speech, due to abnormal activation of the auditory cortex and

disrupted modulation of auditory processing by networks that included the DMN (Wolf et al.).

There appears to have been little investigation of the DMN using ERPs. However, intracerebral EEGs acquired during pre-surgical assessment of epilepsy were consistent with fMRI findings, showing that key regions associated with the DMN deactivate during task-related processing (Jerbi et al., 2010) and identifying characteristic coherence of cortical activity at specific frequencies within the DMN (Ko, Darvas, Poliakov, Ojemann, & Sorensen, 2011). The scalp distribution of EEG spectra relating to resting state networks, including the DMN, have also been characterised (Jann, Kottlow, Dierks, Boesch, & Koenig, 2010). There is little with regard to schizophrenia specifically, although researchers have found that EEG microstates (transient, subsecond periods of stable brain state) characterising the resting state are different in schizophrenia compared to healthy controls (Kindler, Hubl, Strik, Dierks, & Koenig, 2011; Nishida et al., 2013).

The Resting State Hypothesis of AVH

Synthesising such findings and taking a systems-level approach, Northoff and Qin (2011) put forward a new model termed the resting state hypothesis of AVH. This is predicated on the influence of abnormal DMN resting state activity on the functioning of the DMN as a control network. The first component of the resting state hypothesis, called the rest-rest interaction, proposes that elevated resting DMN activity causes elevated resting state activation in the auditory cortex (illustrated in the left-hand peaks of Figure 2). This is deemed to imply reduced capacity of the auditory cortex for responding differentially to internal and external auditory signals (illustrated in the right-hand peaks of Figure 2). This leads to processing errors in the second component of the resting state hypothesis, called the rest-stimulus interaction.

Here, a thought or auditory memory is forward processed by primary auditory cortex as if it were an externally-generated sound and is treated as similarly salient.

The resting state hypothesis attributes the self-relatedness of AVH to the over-elaboration of self-referential thoughts and credits the non-self voice to a recombination of remembered voices, both arising from the supervening role of the DMN (Northoff & Qin; Northoff, Qin, & Nakao, 2010). The hypothesis thus accounts for core features of AVH that process-based models do not explain. However, while Northoff and Qin drew on existing evidence to support their model, the model itself has not been tested.

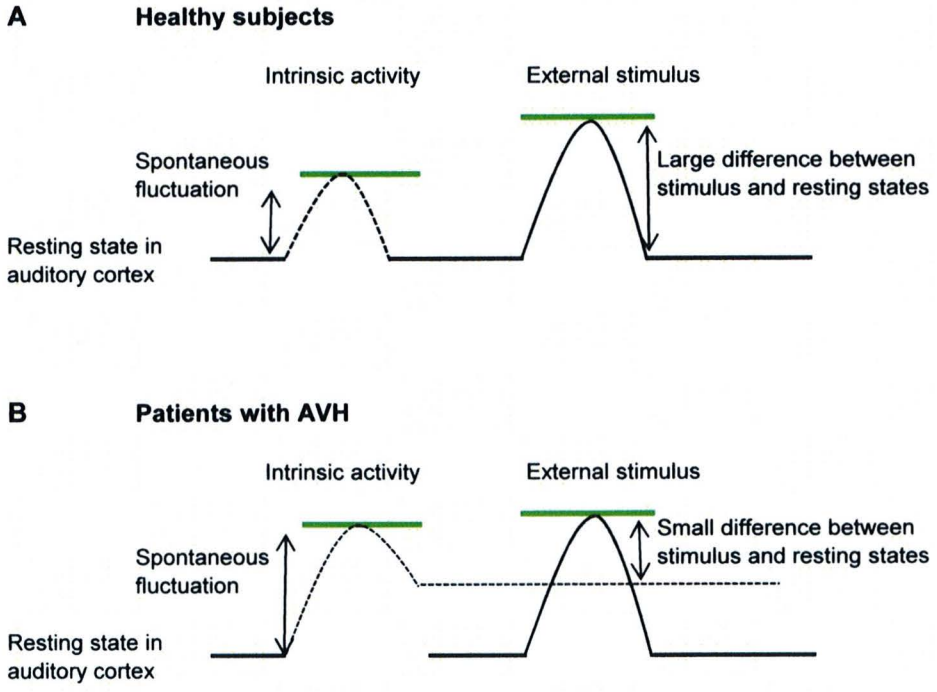

Figure 2. Amplitude of auditory cortex resting state fluctuations compared to stimulus activation in (A) healthy individuals and (B) individuals with AVH. Left-hand peaks indicate intrinsic, spontaneous, activation in the resting state is abnormally elevated in AVH. Right-hand peaks indicate that activation induced by an external auditory stimulus has a reduced effect in AVH. Based on Northoff & Qin (2011).

Rationale for this Study

The research reported here investigated the role of the DMN in the misattribution of auditory signals in AVH, by testing the rest-stimulus interaction component of the resting state hypothesis (Northoff & Qin, 2011). As described above, the conventional explanation is the forward model, represented by the corollary discharge hypothesis. Corollary discharge has received some support from behavioural research and from electrophysiological studies that compare a 'speaking in real time' condition with a 'listening to a recording of own voice' condition (e.g., Ford, Roach, et al., 2007). However, those investigations only examined N1 amplitude and did not consider possible causes of ERP suppression in the absence of motor signals triggering corollary discharge, such as the MMN mechanism (Graux et al., 2013) or temporal cueing (self-initiation of a stimulus provides a timing cue; Sowman, Kuusik, & Johnson, 2012), or the potential influence of the DMN.

The resting state hypothesis predicts that elevated resting state activity reduces the capacity of the auditory cortex to discriminate internally and externally generated auditory signals; a thought or auditory memory is forward processed as if it is as salient as an external sound. An ERP study offers a relatively silent environment within which to administer an auditory paradigm and the fine temporal resolution permits dissection of the components of the cortical response (Sakkalis, 2011). The ERP suppression paradigm was adapted from Ford's laboratory, employing the syllable 'ah' as the vocal auditory stimulus and analysing N1 amplitude as a measure of the initial cortical response (Ford, Gray et al., 2007; Ford, Mathalon, Heinks, et al., 2001; Ford, Roach et al., 2007).

However, to test the prediction, it is necessary to exclude the electromyographic signals (electrical signals emitted by muscles) associated with

speech which may promote corollary discharge. For the present study, rather than comparing conditions of speaking in real time and listening to a recording of the own voice, the paradigm compared passive listening to a recording of the own voice with passive listening to a recording of the voice of another person.[1] The paradigms of Ford and colleagues were also extended by analysing P2 amplitude as a measure of forward processing (Bösel & Tamm, 2003), which to our knowledge has not been carried out previously.

Schizotypy as a Proxy for Schizophrenia

A recognised difficulty in schizophrenia research is the number of potential confounding factors that must be accounted for, including the number of years since diagnosis, the effects of medication, the high incidence of substance use among individuals with schizophrenia, and the impact of social and cognitive decline (Fonseca-Pedrero et al., 2008). However, there is a well established dimensional relationship between schizotypy and schizophrenia (van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009) and research has demonstrated the genetic and neurophysiological correspondence between them (Fanous et al., 2007; Nelson, Seal, Pantelis, & Phillips, 2013).

For example, a DTI study (Nelson et al., 2011), found increased scores on a measure of schizotypy to be correlated with reduced connectivity in white matter tracts, while a voxel-based morphometry study found higher schizotypy to be associated with larger grey matter volumes in posterior cortical regions (Modinos et al., 2010). These deficits are consistent with abnormalities found in schizophrenia (Bora et al., 2011). Electrophysiological anomalies have also been found that are that are similar in schizotypy and schizophrenia, including in auditory ERPs (Koychev,

[1] ERPs were also recorded while a participant was speaking the syllable in real time (as a corollary discharge comparison condition) and thinking the syllable. Technical issues prevented analysis of these data, which will not be presented in this thesis.

2011; Koychev, Deakin, Haenschel, & El-Deredy, 2011; Shin et al., 2010). Finally, in an fMRI study of adolescents using a self-reflection task, scores on the Schizotypal Personality Questionnaire correlated with changes in patterns of activation of key DMN regions; these findings were consistent with similar studies with schizophrenia (Debbané et al., 2014).

Therefore, given the difficulties of recruiting individuals with schizophrenia within a short timeframe, the extent of schizophrenia-related confounds likely to influence results in a small study group, and the exploratory nature of the paradigm, the present study opted to use schizotypy as a proxy for schizophrenia.

Aims and Hypotheses

As described above, the DMN in the resting state has consistently demonstrated elevated activity in individuals with schizophrenia and schizotypy and this underpins the resting state hypothesis of AVH. Hyperactivity in the DMN is therefore assumed for this study. Using EEG to measure cortical activity in response to auditory verbal stimuli, this study aimed to test the rest-stimulus interaction by comparing groups high and low in schizotypal characteristics. It was expected that individuals low in schizotypy would respond to a novel voice as more salient than the own voice, while the high schizotypy group would respond to the own voice as more salient.

To test this, we hypothesized that as level of schizotypy decreased there would be increased N1 and P2 amplitudes to hearing the voice of another person compared to hearing the own voice (reflecting effective suppression of internal auditory signals), but as level of schizotypy increased, there would be increased N1 and P2 amplitude to hearing the own voice compared to hearing the voice of another person (reflecting difficulty discriminating internal from external signals).

Method

Participants

Twenty eight participants with normal hearing were recruited from among first year psychology students at the University of Tasmania and awarded two hours of course credit. Participants were required to be between 18 and 55 years of age as age changes can alter brain function (Tremblay et al., 2001). Participants were selected according to their scores on the Schizotypal Personality Questionnaire Brief Revised version (SPQ-BR; Cohen, Matthews, Najolia, & Brown, 2010). In accordance with a power analysis (Appendix H), the study aimed to recruit 15 participants in each of two groups. A total of 155 students completed the SPQ-BR as part of class work. Since there are no formal cut-offs recognised for the SPQ-BR, the top 15% of individuals, recording scores over 95 points, and the bottom 15%, recording scores under 60 points, were invited to participate further. Of these, 33 individuals volunteered to complete the full study; four later withdrew and one was excluded for technical reasons.

As shown in Table 1, the recruitment strategy resulted in a statistically significant difference in SPQ-BR scores between the high schizotypy group (M = 103.5, range 96 to 118) and the low schizotypy group (M = 51.4, range 35 to 58). The high schizotypy group comprised 15 participants (5 males), with mean age 25.6 years (range 18.4 – 52) and the low schizotypy group comprised 13 participants (5 males), with mean age 24.1 years (range 18.5 - 50.4). Approval for the study was granted by the Human Research Ethics Committee at the University of Tasmania (H0012495). All participants read an information sheet (Appendix B) and signed a consent form (Appendix C) prior to participation.

Materials and Measures

Demographics questionnaire. A demographics questionnaire (Appendix E) recorded participant age and gender and screened for heavy drug or alcohol use over the previous year, hearing impairment, and substantial developmental, neurological, or psychiatric condition that might influence cortical activity. No participant was excluded on these grounds.

Schizotypal Personality Questionnaire, Brief Revised version (SPQ-BR). The SPQ-BR is a self-report measure of schizotypal traits, which has better psychomentric properties than most scales (Fonseca-Pedrero et al., 2008). It is best represented by three super-ordinate factors termed Cognitive-Perceptual, Disorganised, and No Close Friends/Constricted Affect (Cohen et al., 2010). There is also a factor of Social Anxiety but items related to this factor were excluded in the present study to control for social or testing anxiety as a potential confound. For the three-factor scale, α for internal consistency was .97.

Depression Anxiety Stress Scales (DASS). To assess for depression, anxiety, and stress, the 21-item short form of the DASS was administered (DASS-21; Lovibond & Lovibond, 1995; Appendix F); as recommended by Lovibond and Lovibond, scores on the DASS-21 were doubled for equivalence with the DASS long form. Both full and short versions of the DASS have excellent and well-tested psychometric properties across clinical and nonclinical samples. For example, Henry and Crawford (2005) assessed the DASS-21 with a nonclinical adult sample, finding that the subscales were valid with α reliabilities of .88 for depression , .82 for anxiety, and .90 for stress.

Auditory Hallucination-like Experience Scale (AHES). A self-report instrument that distinguishes subclinical hallucinatory experience was required in

order to assess susceptibility to AVH against schizotypy. The Auditory Hallucination-like Experience Scale (AHES; Sugimori, Asai, & Tanno, 2009; Appendix G) is a measure constructed specifically for use across clinical and nonclinical samples and consists of four Factors. Sugimori et al. reported good reliability and validity for the AHES, with the two groups in their Japanese study providing Cronbach's alpha coefficients of .96 and .93 for the total scale.[2] The present study confirmed internal consistency with an α score of .96 for the total scale, and for Factors 1 to 4 alpha was .86, .87, .88, and .88, respectively.

Since schizotypy was used as a proxy for schizophrenia, performance on the AHES was employed to investigate differences between the groups specifically on proneness to auditory hallucination. Factor 1 was selected, as it is the AHES component that relates most closely to AVH (Asai, Sugimori, & Tanno, 2011). As expected, the high schizotypy group reported more auditory hallucination-like experiences than the low schizotypy group on the total scale and Factor 1 scores (*p*s <.001), with large effect sizes (adjusted $\eta_p^2 \geq .48$).

Auditory stimuli. The auditory paradigm was adapted from Ford, Roach, et al. (2007) and produced using the Neuroscan STIM audio system unit and Stim^2 Gentask software (Compumedics Neuroscan, 2003). The paradigm consisted of four independent conditions: Hearing the recording of the voice of another person uttering the syllable (listen-other); hearing a recording of one's own voice uttering the syllable (listen-self); hearing oneself speak the syllable in real time (Speak); and imagining the syllable (Think).[3] For the listen-other condition, a recording of the voice of an adult Australian male uttering the syllable was recorded as a waveform

[2] The AHES was back-translated from Japanese for an earlier study (Ranson, 2011). However, it has yet to be validated with an English-speaking population.

[3] Due to technical difficulties, analysis of data from the Speak and Think conditions is not presented in this study.

file (.WAV) and incorporated into Stim^2 Gentask for playing back to the participant within the paradigm. The experimental speaker and participants were trained to articulate the syllable 'ah' to within 120 to 220 ms and at 54 ± 2 dB (measured using a hand-held sound-level meter at approximately 1 m), and using minimal tongue, throat, and jaw movement. A microphone was used to record the participant uttering a syllable, which was then recorded as a waveform file and incorporated into the paradigm for playback during administration of the listen-self condition. The recorded auditory stimuli were played at the recorded volume. There was no significant difference between the groups for syllable length (high schizotypy M = 0.170 ms; low schizotypy M = 0.160 ms), $F(1,26) = 0.19$, $p = .67$.

Adapting the methodology of Ford's ERP suppression studies, for both the listen-other and listen-self conditions, the instruction word "*START*" appeared for one second to indicate that the paradigm was commencing. Following an interval of one second, the instruction word "rest" appeared for one second. Following an interval of one second, a black cross appeared for one second to indicate the start of a rest period. The screen then remained stimulus-free for 15 seconds, to allow cortical activity to adjust to the low-stimulus environment. Next, the instruction word "listen" appeared for one second, followed by an interval of 500 ms. Then a yellow cross appeared for 500 ms to indicate that an auditory stimulus would be administered. After 500 ms, the sound file containing the syllable 'ah' was played through the audio system and lasted for one second, followed by an interval of one second. This sequence of cross and syllable was repeated five times. Then the instruction word "rest" appeared in black, followed by an interval of one second. Next, a black cross appeared for one second followed by a stimulus-free interval of two seconds. This pattern of a black cross followed by a stimulus-free period was

repeated five times. The sequence of listen and rest blocks was repeated ten times. Finally, "*END thank you!*" appeared on the screen to signify the termination of the paradigm.

The length of the intertrial interval of 3000 ms was selected to inhibit possible response decrements due to refractoriness to the stimulus (Rosburg, Zimmerer, & Huonker, 2010) and was randomly varied by ± 200 ms, to inhibit practice effects. A total of 50 rest and 50 listen trials were recorded per participant in each condition.

EEG Recording

Equipment. The EEG system consisted of NeuroScan Quikcaps with silver/silver chloride electrodes, a Compumedics SynAmps headbox, and a desktop computer. The cap held 32 electrodes (FP1, FP2, F7, F3, FZ, F4, F8, FT7, FC3, FCZ, FC4, FT8, T7, C3, CZ, C4, T8, TP7, CP3, CPZ, CP4, TP8, P7, P3, PZ, P4, P8, O1, OZ, and O2) arranged according to the international 10/20 system (Towle et al., 2003). Linked mastoids provided the reference signal and FZ acted as the ground electrode. Impedances were maintained at 10kΩ or less. Vertical and horizontal electro-oculographic (EOG) activity was recorded. Continuous data were recorded and digitised using Scan version 4.5 software, with a sampling rate of 1,000 Hz and band-pass filter parameters set at .15 Hz to 100 Hz. Using $STIM^2$ software, instructions were presented on a desk-top computer monitor and auditory stimuli administered in stereo via free-standing desk-top speakers placed either side of the monitor.

Procedure. Data recording took place in the Cognitive Neuroscience Laboratory on the Sandy Bay campus of the University of Tasmania. Sessions were conducted during the early evening or at weekends, in order to ensure an environment with consistent and low levels of extraneous noise. On arrival, a

participant was provided with an information sheet describing the study and the nature of the paradigm, and then completed the ethics consent form and questionnaires. The participant recorded the syllable and then the EEG cap was positioned on the participant's head. The auditory paradigm was completed while the participant was seated alone in a darkened room.

Prior to commencing the paradigm, participants were instructed to sit quietly with their eyes open and focused on the computer screen, to not think of anything in particular, and to not go to sleep. The total task time was 28 minutes, with the conditions administered in the order listen-other, listen-self, Speak, and Think. When the paradigm had been completed, the participant the cap was removed and the participant debriefed.

Data preprocessing and analysis. Data were corrected for electro-oculographic (EOG) activity (Semlitsch, Anderer, Schuster, & Presslich, 1986), applying the automated Compumedics Neuroscan algorithm (Compumedics NeuroScan, 2006), and criteria were set to reject signals exceeding ±100 μV as these were likely to constitute environmental or physiological artefacts. The cleaned data was then visually inspected to remove any epochs that appeared to contain idiosyncratic artefacts not corrected by the automated cleaning procedure. Following procedures of Ford, Roach, et al. (2007) to maximise measurement of N1, a band-pass filter was applied to extract frequencies between 2 Hz and 8 Hz. Epochs were then computed for each auditory stimulus response, from -250 ms (following Ford, Roach, et al.) to 450 ms (extending Ford, Roach, et al.) of stimulus onset. Baseline correction was applied to each epoch, using activity in the 100 ms preceding stimulus onset, following Ford, Roach, et al. For each participant, intact epochs were then averaged per electrode per condition.

N1 and P2 were determined a priori as the peaks of interest, as described earlier. N1 was classified as the peak negative waveform between approximately 50 and 175 ms post-stimulus onset (following Ford, Roach, et al., 2007), and P2 as the peak positive-going waveform arising approximately 150 to 250 ms post-stimulus onset (Ferreira-Santos et al., 2012). Through peak detection, mean amplitude per participant for N1 and P2 were calculated for each electrode per condition. Previous literature has found that N1 and P2 amplitudes are maximal at frontal-midline electrode sites (Ferreira-Santos et al.; Foxe et al., 2011) and these sites are routinely used by Ford's team to investigate corollary discharge (Ford, Gray, et al., 2007; Ford et al., 2001; Ford, Roach, et al.). N1 and P2 data analysis in the present study was restricted to electrode sites FZ, FCZ, and CZ, in order to minimise multiple comparisons. Given that the findings across sites were very similar, for parsimony the findings at FCZ will be reported in the main text, with data for FZ and CZ presented in Appendix J. These activity values were exported to SPSS version 21 for statistical analysis.

Statistical analysis

In order to assess differences between the groups, scores on the demographic and clinical measures were analysed using univariate ANOVA, with the fixed factor of Group (high schizotypy and low schizotypy), while Fisher's exact chi-squared test assessed whether the groups differed on gender. Correlation analysis evaluated the strength and direction of relationships among the principal variables of interest. Initial exploration of the EEG mean amplitude data was carried out by visually assessing differences between the groups and conditions on N1 and P2 amplitudes through generation of grand mean ERP waveforms for each group per condition, using Scan 4.5 software. This confirmed that maximal peak amplitudes arose at

frontal-midline sites (FCZ and CZ) and indicated discrepancies between the conditions.

To further investigate the perceived waveform discrepancies, between-groups ANOVAs were conducted to compare the groups on N1 and P2 peak amplitudes. While initial results were nonsignificant, the numbers within the groups were too small to allow multiple testing to account for the effects of depression, anxiety, and stress. Therefore, to increase power, a regression approach that collapsed the two groups was chosen for data analysis. The difference between the conditions in N1 and P2 peak amplitudes was then computed as listen-self minus listen-other for each participant.

Separate hierarchical regression analyses were conducted for N1 and P2, with listen-self minus listen-other amplitude difference as the dependent variable. Since schizotypy was the characteristic that distinguished the study groups, the first set of analyses assessed the ability of schizotypy to predict the N1 and P2 amplitude differences. However, depression, anxiety and stress have been shown to influence ERP responses (Engels et al., 2010; Lupien, McEwen, Gunnar, & Heim, 2010; Sheline, Price, Yan, & Mintun, 2010) and, in this study, DASS-21 subscale scores correlated highly with the SPQ-BR score (Table 2). Therefore, the model controlled for the influence of these variables on the ability of schizotypy to predict the amplitude differences. Since limitations of the sample size ($n = 28$) called for a minimum number of independent variables in each regression (Tabachnick & Fidell, 2013), depression, anxiety, and stress scores were entered in Step 1 in separate regressions. Score on the SPQ-BR was entered in Step 2 for each equation.

Next, the specific impact of susceptibility to auditory hallucination on N1 and P2 amplitude differences was represented by score on AHES Factor 1 (termed here

Hallucination) and a set of regressions were run with hallucination in place of schizotypy . To assess whether hallucination predicted N1 and P2 amplitude differences over and above the effect of depression, anxiety, and stress, these mood variables were, again, separately controlled for in Step 1 of the model, with hallucination entered in Step 2.

Assumptions relating to hierarchical regression were met, following Tabachnick and Fidell (2013). Deviations from normality, homoscedasticity, and linearity were not considered sufficient to invalidate the analyses. There was no evidence of multicollinearity (all correlations were below .90), Durbin-Watson statistics were within the acceptable range (greater than 1 and less than 3), and, at $p < .001$, Mahalanobis distances were less than 13.816. Outliers identified on residuals scatterplots were retained as there was no evidence to suggest that they were not valid in the population. In each of the statistical analyses reported below, statistical significance was set at $p \leq .05$ and a trend towards significance was set at $p \leq .07$. Description of effect size as, for example, small, medium, or large, is based on Cohen (1988). Adjusted R^2 ($\overline{R}^2$) was used for reporting all regression results, as provided by SPSS, in view of the small sample size.

Results

Demographic and Clinical Data

Univariate ANOVAs were conducted with the demographic and clinical variables. Table 1 displays the means, standard deviations, and group difference statistics for schizotypy (SPQ-BR scores); experience of hallucination (AHES Factor 1 subscale scores); depression, anxiety and stress (DASS-21 subscale scores); age, and gender. There was no significant difference between the groups on age or gender. As expected, the difference between the groups on schizotypy was highly

Table 1

Statistical Comparisons Between the High and Low Schizotypy Groups on the Main Study Variables

	Schizotypy								
	High (n = 15)			Low (n = 13)					
Variable	*M*	*SD*	95% CI	*M*	*SD*	95% CI	Test statistic	*p* value	η_p^2
N1 amplitude difference at FCZ	0.71	2.0	[-0.38, 1.8]	-0.47	1.4	[-1.3, 0.1]	$F(1, 26) = 3.21$	.085	
P2 amplitude difference at FCZ	0.82	4.6	[-1.8, 3.4]	0.11	1.5	[-0.8, 0.4]	$F(1, 26) = 0.28$	.604	
SPQ-BR; Schizotypy	103.5	6.7	[99.8, 107.2]	51.4	5.4	[48.1, 54.6]	$F(1, 26) = 506.19$	.000	.95
AHES Factor 1; Hallucination	35.9	8.6	[31.2, 40.7]	20.9	4.3	[18.3, 23.5]	$F(1, 26) = 32.43$	.000	.54
DASS; Depression	15.5	9.3	[10.3, 20.6]	3.7	2.3	[2.3, 5.1]	$F(1, 26) = 19.57$	.000	.43
DASS; Anxiety	14.3	8.4	[9.6, 18.9]	1.9	2.7	[0.2, 3.5]	$F(1, 26) = 25.99$	.000	.50
DASS; Stress	20.4	8.2	[15.9, 25.0]	6.2	4.7	[3.3, 9.0]	$F(1, 26) = 30.29$	.000	.54
Age	25.6	11.6	[20.0, 31.2]	24.1	9.1	[18.1, 30.0]	$F(1, 26) = 0.15$	.700	.01
Gender	10 females, 5 males			8 females, 5 males			Fisher's exact	1.00 (two-sided)	

Note. Amplitude difference = listen-self minus listen-other. CI = confidence interval. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety). AHES = Auditory Hallucination-like Experiences Scale. Hallucination = lifetime extent of auditory hallucination experience, assessed by the Auditory Hallucination-like Experiences Scale Factor 1 subscale score. Depression, anxiety, and stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short form. To convert to full-scale scores, DASS-21 scores were multiplied by 2, as recommended by Lovibond & Lovibond (1995).

significant, with a very large effect size. The high schizotypy group reported significantly greater levels of depression, anxiety, and stress than the low schizotypy group, with large effect sizes. Correlations between the key study variables (schizotypy, hallucination, depression, anxiety, stress, and N1 and P2 amplitude differences at FCZ) are shown in Table 2.

Table 2

Intercorrelations for Scores on the SPQ-BR (Excluding the Factor for Social Anxiety), AHES Factor 1 Subscale, the DASS-21 Subscales (Depression, Anxiety, and Stress), and the N1 and P2 Amplitude Differences at Electrode FCZ

Measure	1	2	3	4	5	6	7
1. SPQ-BR Schizotypy	-						
2. AHES Factor 1 Hallucination	.72***	-					
3. DASS-21 Depression	.68***	.62***	-				
4. DASS-21 Anxiety	.67***	.80***	.74***	-			
5. DASS-21 Stress	.68***	.78***	.66***	.86***	-		
6. N1 amplitude difference at FCZ	.37*	.14	.43**	.24	.14	-	
7. P2 amplitude difference at FCZ	.17	.19	.12	-.06	-.15	.30	-

Note. * $p < .05$. ** = $p < .01$. *** = $p < .001$. Amplitude difference = listen-self minus listen-other. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety). Hallucination = lifetime extent of auditory hallucination experience, assessed by the Auditory Hallucination-like Experiences Scale Factor 1 subscale score. Depression, anxiety, and stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short form.

Visual inspection of ERP waveforms

Initial exploration of the data was carried out by visual inspection of the ERP grand mean waveforms for each group per condition. The whole-head grand mean waveforms are shown in Figure 3, indicating that N1 and P2 peak amplitudes were maximal on the midline at FCZ and CZ. The detailed waveforms for electrode site FCZ are shown in Figure 4.

Figure 3. Grand mean waveforms for each group per condition and for all electrode sites, showing the N1 and P2 peaks.

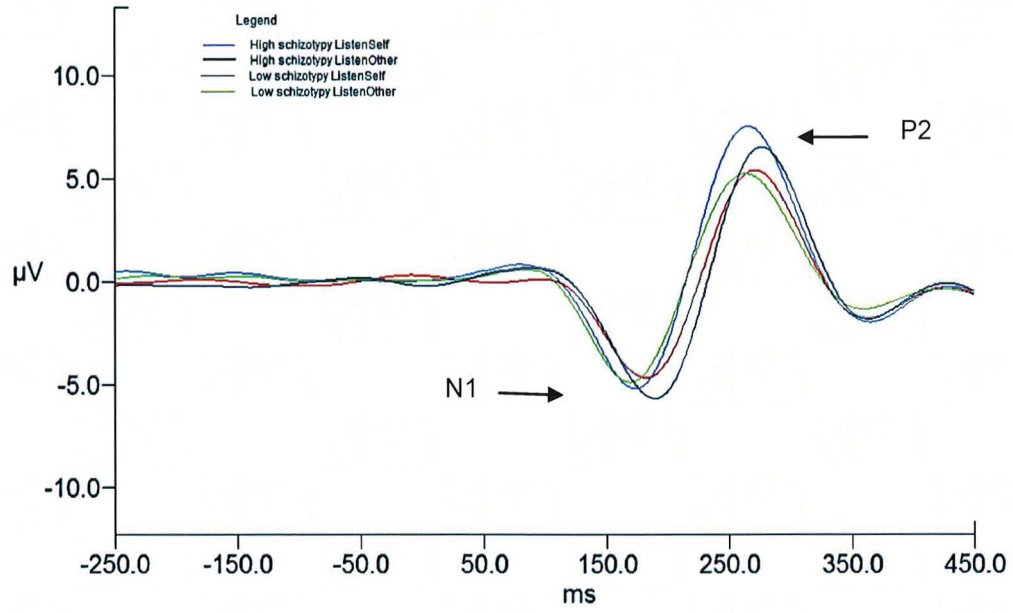

Figure 4. Grand mean waveforms for each group per condition at electrode site FCZ, showing the N1 and P2 peaks.

Analysis of Variance

A mixed within-between subjects analysis of variance was conducted to compare the impact of schizotypy (high and low) on the N1 and P2 amplitude differences when listening to the own voice or the voice of another person. All comparisons were nonsignificant.

Regression Analysis

The results presented below and in Tables 3-6, concern the N1 amplitude difference and P2 amplitude difference arising at electrode site FCZ. Results for electrode sites FZ and CZ are provided in Appendix I. Suppression relationships between the predictor variables (schizotypy and hallucination) and the controlled variables (depression , anxiety, and stress) emerged as a factor in the analyses. As described by Tabachnick and Fidell (2013), suppression arises when one independent variable enhances the predictive effect of another independent variable, by removing variance that is irrelevant to prediction of the dependent variable (here,

N1 or P2 amplitude difference). In classical suppression, the beta weight of the predictor variable is larger than the absolute value of the correlation between the predictor and dependent variables (Tabachnick & Fidell). Reciprocal suppression occurs when the relationship with the dependent variable improves for both independent variables, and negative suppression occurs when the beta weight of the predictor is larger than the correlation value but has the opposite sign (Tabachnick & Fidell).

Ability of Schizotypy to Predict N1 Amplitude Difference

The dependent variable of N1 amplitude difference was derived by subtracting the N1 amplitude in the listen-other condition from the N1 amplitude in the listen-self condition. As shown in Figure 5, if the value of N1 amplitude difference is negative, then there is greater suppression of N1 amplitude in response to the listen-other stimulus than to the listen-self stimulus. This represents a stronger cortical response when listening to a recording of the own voice than listening to a recording of the voice of another person. Conversely, if the value of the N1 amplitude difference is positive, then there is greater suppression of N1 in listen-self than in listen-other. This represents a reduced cortical response when listening to a recording of the own voice, compared to listening to a recording of the voice of another person. Table 3 presents the regression results for the N1 amplitude difference, listen-self minus listen-other, including beta weights, significance tests, and adjusted R-squared for each step.

Direct effect of schizotypy on N1 amplitude. Simple linear regression revealed that schizotypy was significantly associated with the N1 amplitude difference, $\overline{R}^2 = .10$, $F(1, 26) = 4.06$, $p = .05$, $\beta = .37$.

Controlling for the effect of stress. A hierarchical multiple regression

examined the effect of stress and schizotypy on the N1 amplitude difference between listen-self and listen-other. To test whether schizotypy added any significant variance over and above that of stress, stress was entered in Step 1 and schizotypy

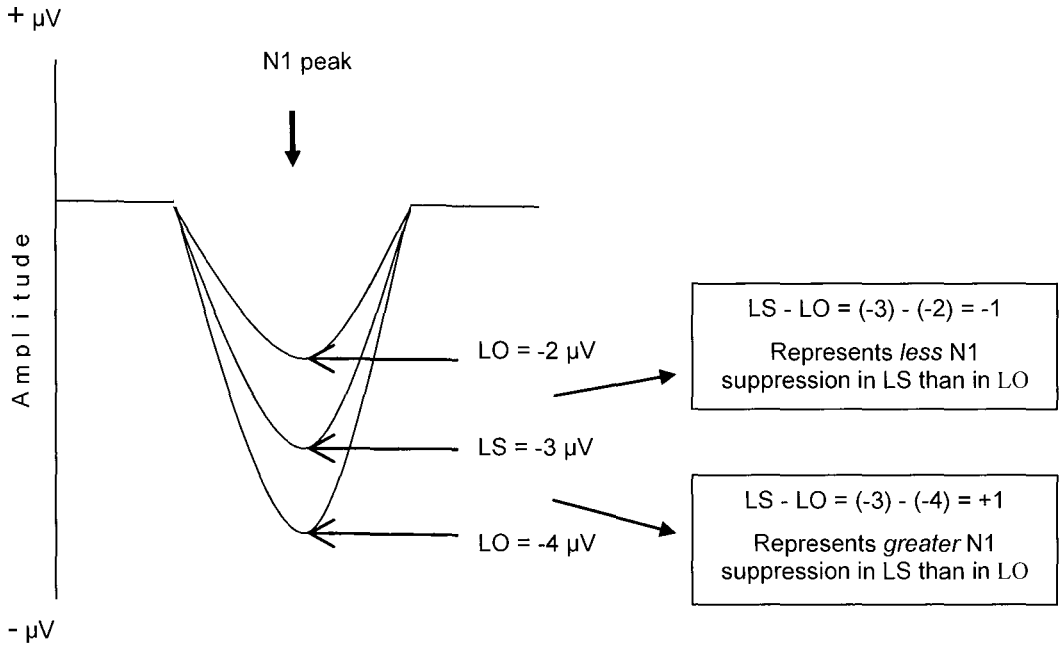

Figure 5. The calculation of the direction of N1 amplitude difference between the listen-self (LS) and listen-other (LO) conditions. N1 amplitude difference was computed as listen-self minus listen-other.

entered in Step 2. After Step 1, stress did not significantly predict differential N1 amplitude to listen-self versus listen-other. After Step 2, schizotypy was a significant predictor of differential N1 amplitude, explaining 14% of unique variance in N1 amplitude difference. The positive beta weight (.51) of this relationship reflects that as schizotypy total score increased, N1 amplitude increased to listen-other compared to listen-self. There was a classical suppression relationship between schizotypy and stress, with schizotypy predicting a higher proportion of the variance in N1 amplitude difference in the presence of stress than under the simple linear regression, although the significance level did not change.

Table 3

Hierarchical Regression Analyses Predicting N1 Amplitude Difference From Schizotypy, Controlling Separately for Depression, Anxiety, and Stress, at Electrode FCZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.69	0.47		-1.46	.16	[-1.66, 0.28]
	Depression	0.09	0.04	.43	2.43	.02	[0.01, 0.16]
	$\bar{R}^2$ = .15, $F(1, 26) = 5.91$, $p = .02$						
Step 2	Constant	-1.25	1.07		-1.16	.26	[-3.46, 0.97]
	Depression	0.07	0.05	.34	1.38	.18	[-0.03, 0.17]
	Schizotypy	0.01	0.02	.14	0.58	.57	[-0.02, 0.04]
	$\bar{R}^2$ = .13; $F(2, 25) = 3.05$, $p = .07$				$\Delta\bar{R}^2$ = .01, $\Delta F(1, 25) = 0.34$, $p = .57$		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-2.45	0.47		-0.52	.61	[-1.22, 0.73]
	Anxiety	0.05	0.04	.24	1.25	.22	[-0.03, 0.13]
	$\bar{R}^2$ = .02, $F(1, 26) = 1.56$, $p = .22$						
Step 2	Constant	-1.81	1.14		-1.59	.12	[-4.16, 0.53]
	Anxiety	0.00	0.05	-.02	-0.07	.94	[-0.11, 0.10]
	Schizotypy	0.03	0.02	.38	1.51	.14	[-0.01, 0.06]
	$\bar{R}^2$ = .07; $F(2, 25) = 1.96$, $p = .16$				$\Delta\bar{R}^2$ = .08, $\Delta F(1, 25) = 2.28$, $p = .14$		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.19	0.60		-0.32	.75	[-1.43, 1.05]
	Stress	0.03	0.04	.14	0.72	.48	[-0.05, 0.10]
	$\bar{R}^2$ =.00, $F(1, 26) = 0.52$, $p = .48$						
Step 2	Constant	-2.00	1.06		-1.89	.07	[-4.18, 0.18]
	Stress	-0.04	0.05	-.20	-0.82	.42	[-0.13, 0.06]
	Schizotypy	0.03	0.02	.51	2.02	.05	[0.00, 0.07]
	$\bar{R}^2$ = .09; $F(2, 25) = 2.34$, $p = .12$				$\Delta\bar{R}^2$ = .14, $\Delta F(1, 25) = 4.09$, $p = .05$		

Note. $n = 28$ in all analyses. CI = confidence interval. $\bar{R}^2$ = Adjusted R^2.
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety).

Controlling for the effect of depression. In a two-step hierarchical multiple regression to examine the effect of depression and schizotypy on N1 amplitude differences between listen-self and listen-other, depression was entered in Step 1 and schizotypy entered in Step 2. After Step 1, depression was a significant predictor of N1 amplitude differences, explaining 15% of the variance. The positive beta weight of this relationship (.43) reveals that increased depression is associated with increased N1 amplitude to listen-other compared to listen-self. After entry of schizotypy at Step 2, neither schizotypy nor depression were significant predictors of N1 amplitude. This result suggests that schizotypy and depression share variance on N1 amplitude difference to a large extent.

Controlling for the effect of anxiety. Results for the two-step hierarchical regression examining the effect of anxiety in Step 1, and anxiety and schizotypy in Step 2 reveal no significant predictors of N1 amplitude difference at either step.

Ability of Schizotypy to Predict P2 Amplitude Difference

The dependent variable of P2 amplitude difference entered into the regression equations was derived from subtracting P2 amplitude listen-other from P2 amplitude listen-self. As illustrated in Figure 6, a negative value of the P2 amplitude difference represents less forward processing of the stimulus in listen-self than in to listen-other, which indicates a higher level of processing in response to hearing the other voice than hearing the own voice. A positive value represents more forward processing in listen-self compared to listen-other, indicating a higher level of processing of the own voice stimulus compared to the other voice. Table 4 presents the regression results for the P2 amplitude difference, listen-self minus listen-other, including beta weights, significance tests, and adjusted R-squared for each step.

Direct effect of schizotypy on P2 amplitude. Simple linear regression revealed that the association between schizotypy and the P2 amplitude difference was nonsignificant, $\overline{R}^2 = 0$, $F(1, 26) = 0.78$, $p = .39$, $\beta = .17$.

Controlling for the effect of stress. A hierarchical multiple regression examined the effect of stress and schizotypy on P2 amplitude difference between

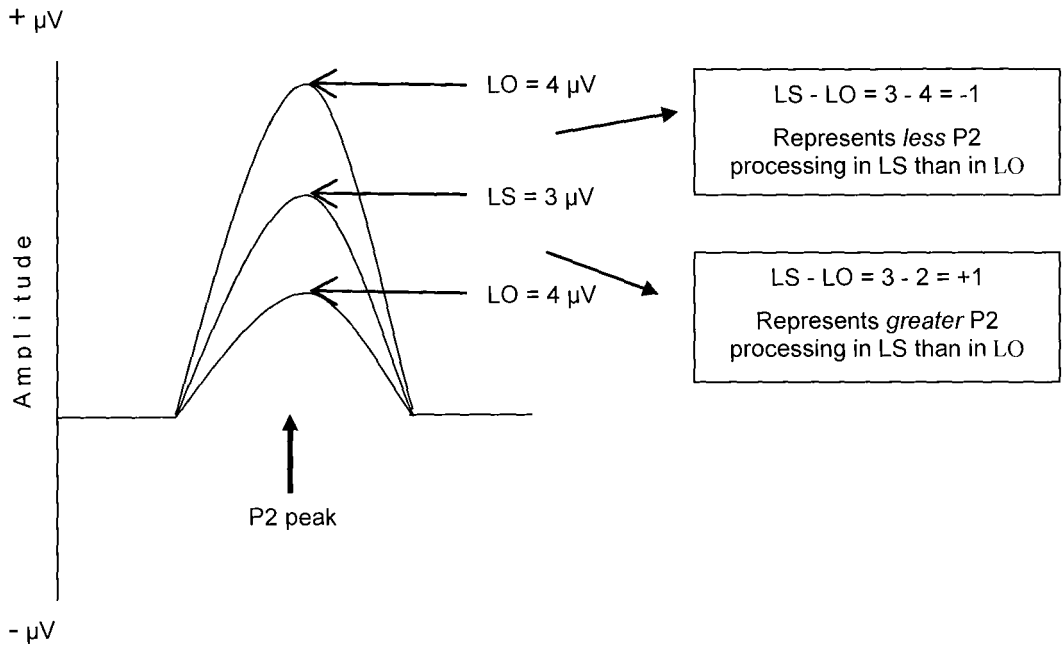

Figure 6. The calculation of the direction of P2 amplitude difference between the listen-self (LS) and listen-other (LO) conditions. P2 amplitude difference was computed as listen-self minus listen-other.

listen-self and listen-other. Stress was entered in Step 1 and schizotypy entered in Step 2 to test whether schizotypy added any significant variance over and above that of stress. After Step 1, stress did not significantly predict P2 amplitude difference between listen-self and listen-other. After Step 2, schizotypy was a significant predictor of P2 amplitude difference, explaining 14% of unique variance in P2 amplitude. The positive beta weight (.51) of this relationship reflected that as schizotypy score increased, P2 amplitude increased to listen-self compared to listen-

Table 4

Hierarchical Regression Analyses Predicting P2 Amplitude Difference From Schizotypy, Controlling Separately for Depression, Anxiety, and Stress, at Electrode FCZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.03	1.00		0.03	.98	[-2.04, 2.09]
	Depression	0.05	0.08	.12	0.62	.54	[-0.11, 0.20]
	$\overline{R}^2$ = 0, $F(1, 26)$ = 0.83, p = .54						
Step 2	Constant	-1.23	2.28		-0.54	.60	[-5.93, 3.47]
	Depression	0.00	0.10	.01	0.04	.97	[-0.21, 0.22]
	Schizotypy	0.02	0.03	.16	0.61	.55	[-0.05, 0.09]
	$\overline{R}^2$ = 0*; F (2, 25) = 0.38, p = .69				$\Delta\overline{R}^2$ = .02, $\Delta F(1, 25)$ = 0.38, p = .55		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.68	0.94		0.73	.47	[-1.24, 2.61]
	Anxiety	-0.02	0.08	-.06	-0.30	.77	[-0.18, 0.14]
	$\overline{R}^2$ = .00, $F(1, 26)$ = 0.90, p = .77						
Step 2	Constant	-2.39	2.26		-1.06	.30	[-7.05, 2.27]
	Anxiety	-0.13	0.10	-.32	-1.23	.23	[-0.33, 0.09]
	Schizotypy	0.05	0.03	.39	1.49	.15	[-0.02, 0.12]
	$\overline{R}^2$ = .01; F (2, 25) = 1.15, p = .33				$\Delta\overline{R}^2$ = .08, $\Delta F(1, 25)$ = 2.21, p = .15		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	1.23	1.16		1.06	.30	[-1.16, 3.61]
	Stress	-0.05	0.07	-.15	-0.78	.44	[-0.20, 0.09]
	$\overline{R}^2$ = .00, $F(1, 26)$ = 0.61, p = .44						
Step 2	Constant	-2.29	2.04		-1.12	.27	[-6.50, 1.92]
	Stress	-0.18	0.09	-.50	-1.99	.06	[-0.36, 0.01]
	Schizotypy	0.07	0.03	.51	2.04	.05	[0.00, 0.13]
	$\overline{R}^2$ = .10; F (2, 25) = 2.42, p = .11				$\Delta\overline{R}^2$ = .14, $\Delta F(1, 25)$ = 4.16, p = .05		

Note. n = 28 in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety).

other. There was a reciprocal suppression relationship between schizotypy and stress, with schizotypy predicting a higher proportion of the variance in P2 amplitude difference in the presence of stress than in the simple linear regression, and stress showed a trend towards significance in the presence of schizotypy . While the absolute beta values of schizotypy and stress were virtually equal, stress was signed opposite to schizotypy , which indicated that, as level of stress increased, P2 amplitude was greater to listen-other than to listen-self.

Controlling for the effect of anxiety. Results for the two-step hierarchical regression examining the effect of anxiety in Step 1 and anxiety and schizotypy in Step 2, revealed no significant predictors of P2 amplitude difference at either step. However, although statistically nonsignificant, there was evidence of reciprocal suppression with regression weights and *p* values improving for both schizotypy and anxiety; the regression weights were oppositely signed.

Controlling for the effect of depression. Results for the two-step hierarchical regression examining the effect of depression in Step 1 and depression and schizotypy in Step 2, revealed no significant predictors of P2 amplitude difference at either step.

Ability of Hallucination Experience to Predict N1 Amplitude Difference

The dependent variable of N1 amplitude difference was derived by subtracting the N1 amplitude in the listen-other condition from the N1 amplitude in the listen-self condition. The potential outcomes of this calculation are illustrated in Figure 5 and described above. Table 5 presents the regression results for the N1 amplitude difference, listen-self minus listen-other, including beta weights, significance tests, and adjusted R-squared for each step.

Direct effect of hallucination experience on N1 amplitude. Simple linear regression revealed that the association between hallucination and the P2 amplitude difference was nonsignificant, $\overline{R}^2 = 0$, $F(1, 26) = 0.49$, $p = .49$, $\beta = .14$.

Controlling for the effect of depression. A hierarchical multiple regression examined the effect of hallucination experience on N1 amplitude difference while controlling for depression. Depression made a significant contribution at Step 1, explaining 15% of the variance in N1 amplitude difference between listen-self and listen-other. After entry of hallucination in Step 2, only depression made a significant unique contribution in the final model. The positive beta value indicated that as depression increased, N1 amplitude to listen-self was greater than to listen-other. However, there was some indication of a suppression relationship between hallucination and depression, with the absolute value of the oppositely-signed regression weights increasing for both hallucination and depression after Step 2 and, although remaining statistically nonsignificant, the *p* value improving for Hallucination.

Controlling for the effect of anxiety. Hierarchical regression examining the effect of anxiety in Step 1 and anxiety and hallucination in Step 2, revealed no significant predictor of N1 amplitude difference at either step.

Controlling for the effect of stress. Hierarchical regression examining the effect of stress in Step 1 and stress and hallucination in Step 2, revealed no significant predictor of N1 amplitude difference at either step.

Ability of Hallucination Experience to Predict P2 Amplitude Difference

The dependent variable of P2 amplitude difference was derived by subtracting the P2 amplitude in the listen-other condition from the P2 amplitude in the listen-self condition. The potential outcomes of this calculation are described above. Table 6

Table 5

Hierarchical Regression Analyses Predicting N1 Amplitude Difference From Experience of Auditory Hallucination, Controlling Separately for Depression, Anxiety, and Stress, at Electrode FCZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.69	0.47		-1.46	.16	[-1.66, 0.28]
	Depression	0.09	0.04	.43	2.43	.02	[0.01, 0.16]
	$\overline{R}^2$ = .15, $F(1, 26)$ = 5.91, p = .02						
Step 2	Constant	0.15	1.00		0.15	.88	[-1.91, 2.21]
	Depression	0.11	0.05	.56	2.49	.02	[0.02, 0.21]
	Hallucination	-0.04	0.04	-.22	-0.95	.35	[-0.12, 0.05]
	$\overline{R}^2$ = .15; F (2, 25) = 3.39, p = .05				$\Delta\overline{R}^2$ = .03, $\Delta F(1, 25)$ = 0.90, p = .35		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-2.45	0.47		-0.52	.61	[-1.22, 0.73]
	Anxiety	0.05	0.04	.24	1.25	.22	[-0.03, 0.13]
	$\overline{R}^2$ = .02, $F(1, 26)$ = 1.56, p = .22						
Step 2	Constant	0.32	1.30		0.25	.81	[-2.36, 2.99]
	Anxiety	0.07	0.07	.36	1.11	.28	[-0.06, 0.21]
	Hallucination	-0.03	0.06	-.15	-0.47	.65	[-0.14, 0.09]
	$\overline{R}^2$ = .0*; F (2, 25) = 0.86, p = .43				$\Delta\overline{R}^2$ = .01, $\Delta F(1, 25)$ = 0.22, p = .65		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.19	0.60		-0.32	.75	[-1.43, 1.05]
	Stress	0.03	0.04	.14	0.72	.48	[-0.05, 0.10]
	$\overline{R}^2$ = .00, $F(1, 26)$ = 0.52, p = .48						
Step 2	Constant	-0.40	1.17		-0.34	.74	[-2.82, 2.02]
	Stress	0.02	0.06	.09	0.28	.78	[-0.10, 0.14]
	Hallucination	0.01	0.06	.07	0.21	.84	[-0.10, 0.13]
	$\overline{R}^2$ = 0*; F (2, 25) = 0.27, p = .76				$\Delta\overline{R}^2$ = 0, $\Delta F(1, 25)$ = 0.04, p = .84		

Note. n = 28 in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).

Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Hallucination = lifetime extent of auditory hallucination experience, assessed by Factor 1 of the Auditory Hallucination-like Experience Scale.

presents the regression results for the P2 amplitude difference, listen-self minus listen-other, including beta weights, significance tests, and adjusted R-squared for each step.

Direct effect of hallucination experience on P2 amplitude. Simple linear regression revealed that the association between hallucination and the P2 amplitude difference was nonsignificant, $\overline{R}^2 = 0$, $F(1, 26) = 0.95$, $p = .34$, $\beta = .19$.

Controlling for the effect of stress. A hierarchical multiple regression examined the effect of stress and hallucination experience on P2 amplitude difference between listen-self and listen-other. Stress was entered in Step 1 and hallucination entered in Step 2 to test whether hallucination experience added any significant variance over and above that of stress. After Step 1, the effect of stress on P2 amplitude difference was nonsignificant. After Step 2, hallucination was a highly significant predictor of P2 amplitude difference, explaining 24% of unique variance in P2 amplitude. The positive beta weight (.79) of this relationship reflected that, as hallucination score increased, P2 amplitude increased to listen-self in comparison to listen-other. There was a reciprocal suppression relationship between hallucination and stress, with hallucination predicting a higher proportion of the variance in P2 amplitude difference in the presence of stress than in the simple linear regression. As a result of reciprocal suppression, stress also emerged as a significant predictor of P2 amplitude difference in the presence of Hallucination. While the absolute beta values of hallucination and stress were virtually equal, stress was signed opposite to Hallucination, which indicated that, as level of stress increased, P2 amplitude was greater to listen-other than to listen-self.

Controlling for the effect of anxiety. A hierarchical multiple regression examined the effect of anxiety and hallucination experience on P2 amplitude

Table 6

Hierarchical Regression Analyses Predicting P2 Amplitude Difference From Experience of Auditory Hallucination, Controlling Separately for Depression, Anxiety, and stress, at Electrode FCZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.03	1.00		0.03	.98	[-2.04, 2.09]
	Depression	0.05	0.08	.12	0.62	.54	[-0.11, 0.20]
	$\overline{R}^2$ = .00, $F(1, 26) = 0.38$, $p = .54$						
Step 2	Constant	-1.36	2.14		-0.63	.53	[-5.77, 3.06]
	Depression	0.00	0.10	.01	0.03	.98	[-0.20, 0.20]
	Hallucination	0.06	0.09	.18	0.73	.47	[-0.11, 0.24]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.46$, $p = .64$				$\Delta\overline{R}^2$ = .02, $\Delta F(1, 25) = 0.54$, $p = .47$		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.68	0.94		0.73	.47	[-1.24, 2.61]
	Anxiety	-0.02	0.08	-.06	-0.30	.77	[-0.18, 0.14]
	$\overline{R}^2$ = .00, $F(1, 26) = 0.09$, $p = .77$						
Step 2	Constant	-4.03	2.39		-1.69	.10	[-8.94, 0.89]
	Anxiety	-0.23	0.12	-.58	-1.89	.07	[-0.47, 0.02]
	Hallucination	0.22	0.11	.65	2.12	.04	[0.01, 0.44]
	$\overline{R}^2$ = .09; $F(2, 25) = 2.31$, $p = .12$				$\Delta\overline{R}^2$ = .15, $\Delta F(1, 25) = 4.51$, $p = .04$		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	1.23	1.16		1.06	.30	[-1.16, 3.61]
	Stress	-0.05	0.07	-.15	-0.78	.44	[-0.20, 0.09]
	$\overline{R}^2$ = .00, $F(1, 26) = 0.61$, $p = .44$						
Step 2	Constant	-3.58	1.97		-1.82	.08	[-7.63, 0.47]
	Stress	-0.27	0.10	-.77	-2.79	.01	[-0.48, 0.07]
	Hallucination	0.27	0.09	.79	2.87	<.01	[0.08, 0.47]
	$\overline{R}^2$ = .21; $F(2, 25) = 4.49$, $p = .02$				$\Delta\overline{R}^2$ = .24, $\Delta F(1, 25) = 8.21$, $p = <.01$		

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Hallucination = lifetime extent of auditory hallucination experience, assessed by Factor 1 of the Auditory Hallucination-like Experience Scale.

difference between listen-self and listen-other. Anxiety was entered in Step 1 and hallucination entered in Step 2 to test whether hallucination experience added any significant variance over and above that of anxiety. After Step 1, anxiety did not significantly predict P2 amplitude difference between listen-self and listen-other. After Step 2, hallucination was a significant predictor of P2 amplitude difference, explaining 15% of unique variance in P2 amplitude. The positive beta weight (.65) of this relationship reflected that as hallucination score increased, P2 amplitude increased to listen-self compared to listen-other. There was a reciprocal suppression relationship between hallucination and anxiety, with hallucination predicting a higher proportion of the variance in P2 amplitude difference in the presence of anxiety than in the simple linear regression, and anxiety showed a trend towards significance in the presence of Hallucination. While the absolute beta values of hallucination and anxiety were similar, anxiety was signed opposite to Hallucination, which indicated that, as level of anxiety increased, P2 amplitude was greater to listen-other than to listen-self.

Controlling for the effect of depression. Results for the two-step hierarchical regression examining the effect of depression in Step 1 and depression and hallucination in Step 2, revealed no significant predictors of P2 amplitude difference at either step.

Discussion

The ERP research reported here tested the rest-stimulus interaction, a component of the resting state hypothesis of AVH (Northoff & Qin, 2011). Using schizotypy as a proxy for schizophrenia and in the absence of a motor signal (and resultant corollary discharge), we examined whether schizotypy and experience of auditory hallucinations were associated with difficulty discriminating internal from

external auditory signals. In line with prediction, increased schizotypy and auditory hallucination experience were associated with increased P2 amplitude to internally generated (listen-self) signals compared to externally generated (listen-other) signals. However, this was only observed when controlling for stress and anxiety in regression analyses, but not when controlling for depression. It was also predicted that increased schizotypy and auditory hallucination experience would be associated with increased N1 amplitude to internally generated (listen-self) signals compared to externally generated (listen-other) signals, but this was not found. Rather, increased schizotypy was associated with increased N1 amplitude to externally generated signals, but only when controlling for stress.

The N1 Amplitude Difference

The rest-stimulus interaction proposes that, in schizophrenia, over-activation of the auditory cortex in a task-free state means that auditory cortex does not have the capacity to adequately limit its response to internally-generated stimuli (e.g., thoughts). To test this, the first hypothesis was that, as schizotypy increased, the amplitude of the initial attentional response (indexed by N1) would also increase when listening to a recording of the own voice compared to listening to the recording of the voice of another person. This was not confirmed. Instead, increased schizotypy score was associated with increased N1 amplitude to other-voice signals compared to own-voice signals, both directly and when controlling for stress.

This finding also contradicts previous ERP research into the corollary discharge mechanism. Convincing evidence has been found that, in healthy controls, N1 amplitude is reduced (suppressed) to self-generated auditory signals (a 'talk' condition) compared to external signals (a 'listen' condition) and that this N1 suppression effect is reduced in schizophrenia (Ford, Roach, et al., 2007). Thus, in

that study, participants with schizophrenia displayed relatively greater N1 amplitudes to internal signals than controls (Ford, Roach, et al.). Importantly, however, the present study excluded the overt motor signals caused by speaking that are thought to trigger corollary discharge and attenuate the cortical response to the auditory signal (Feinberg & Guazzelli, 1999). Consequently, the association between higher schizotypy and greater N1 amplitude to externally generated auditory signals was found in the absence of the corollary discharge mechanism. This suggests the presence of a different, underlying mechanism associated with schizotypy that is, as yet, unknown.

The significant association of schizotypy with increased N1 amplitude to external signals was enhanced after controlling for stress in the hierarchical regression, indicating that the inclusion of stress suppressed irrelevant variance in the regression. The opposite direction of the beta weights indicated that, while higher schizotypy predicted increased N1 amplitude difference, the effect was stronger when stress was lower. Schizotypy was not a significant predictor when controlling for anxiety or depression. This indicated that anxiety and depression shared more variance with schizotypy in predicting N1 amplitudes than did stress. Indeed, shared variance eliminated the statistically-significant predictive power of depression when schizotypy was added to the regression equation.

Depression, anxiety, and stress are all well known to affect brain function, as are medications used to treat their clinically-significant symptoms (Stahl, 2008; Sylvester et al., 2012). But the situation is complex and the influences of stress and depression in the N1 amplitude difference regression models are intriguing. Stress is considered a vulnerability marker in the schizophrenia spectrum, which includes schizotypy (Herzig & Mohr, 2013). Therefore, the significant difference between the

high and low schizotypy groups was expected. However, the impact of exposure to stress on cortical functioning is not straightforward, depending on factors such as the age at which the stressor is experienced and whether deleterious effects are offset to any extent by positive influencers (Lupien et al., 2010). Interestingly, the impact of stress here appears to be indirect, since the factor of stress did not offer a unique explanation for variance in the N1 amplitude difference, instead removing variance that was irrelevant to the effect of schizotypy on the amplitude difference.

The three superordinate factors of the SPQ-BR used here measure several constructs (cognitive-perceptual, disorganized, and socially-isolated traits; Cohen et al., 2010). It is possible that one or more constructs may have influenced the effect of schizotypy on the N1 response, over and above the effect of the SPQ-BR subfactor of usual perceptual experiences. To more closely examine the effect of susceptibility to AVH on N1 amplitude, Factor 1 of the AHES was used as a measure of previous auditory hallucination experiences. However, the first hypothesis was not supported when using hallucination experience as a predictor, suggesting that the SPQ-BR taps a characteristic that Factor 1 of the AHES does not. Hallucination was not found to be a significant predictor of N1 amplitude of listen-self relative to listen-other, either directly or when controlling for stress, anxiety, or depression.

However, depression remained a significant predictor of N1 amplitude and this effect was slightly enhanced by suppression when schizotypy was entered into the regression equation. As depression increased, there was a relative increase in N1 amplitude to listen-self compared to listen-other. Although a recent review found that most studies investigating auditory N1 amplitude in patients with depression did not detect any difference from nondepressed controls, with just four studies

identifying reduced auditory N1 amplitude in patients (Bruder, Kayser, & Tenke, 2009), it is possible that the situation is different for depression associated with the schizophrenia spectrum, including schizotypy. This is especially as the severity of depression and its temporal relationship with the development of psychotic symptoms inform diagnosis along the schizophrenia spectrum (American Psychiatric Association, 2013).

Although the N1 hypothesis was not supported and the specific finding appears at first sight to be inconsistent with prior studies, the general finding of a statistically significant discrepancy in the N1 amplitude difference is nevertheless in keeping with previous research. Deficits in N1 amplitude have been reliably measured in schizophrenia, first-degree relatives of schizophrenia probands (symptomatic individuals), and in individuals high in schizotypal traits (Ahveninen et al., 2006; Ford, Roach, et al., 2007; Foxe et al., 2011). The discrepancy revealed in the present study may reflect a different mechanism to those previously identified. Moreover, it may reflect a normal variability associated with nonclinical levels of schizotypy that is not necessarily continuous with clinical deficits.

The P2 Amplitude Difference

The second prediction proposed that, as schizotypy increased, the amplitude difference in forward processing between listening to the own voice and listening to the other voice would also increase, indexed by P2. Specifically, it was predicted that as schizotypy increased, there would be an increase in P2 amplitude to internal (listen-self) signals compared to external (listen-other) signals. This proposition was supported by schizotypy when the effect of stress was taken into account, with P2 amplitude increasing to listen-self as schizotypy increased. The suppression relationship between schizotypy and stress was reciprocal. Stress showing a trend

towards statistical significance in the presence of schizotypy, with an oppositely-signed beta weight, such that the P2 amplitude difference increased as level of stress decreased. Schizotypy was not a significant predictor after controlling for depression or anxiety, and depression and anxiety were also nonsignificant as predictors.

The P2 hypothesis was supported by hallucination when the effect of either stress or anxiety was taken into account, with P2 amplitude to listen-self compared to listen-other increasing as hallucination increased. Both controlled variables showed reciprocal suppression and beta weights oppositely-signed to hallucination. Stress emerged as a significant predictor of P2 amplitude difference, while anxiety showed a trend towards significance. Thus, P2 amplitude difference increased as schizotypy increased and as stress or anxiety decreased. However, substantial shared variance between schizotypy and depression obscured any relationship of either variable with P2.

There has been little research interest in P2 amplitude in schizophrenia to date (Salisbury et al., 2010) and findings have been mixed. However, most studies have identified deficits in auditory P2 in schizophrenia, in first-degree relatives of schizophrenia probands, and in individuals high in schizotypal traits (Ferreira-Santos et al., 2012; Ogura et al., 2008; Salisbury et al.; Stekelenburg, Maes, Van Gool, Sitskoorn, & Vroomen, 2013). Although these studies were measuring different parameters through different tasks, the P2 amplitude difference associated with level of schizotypy and hallucination experience found in the present study reflects a similar discrepancy. However, since this study used a nonclinical sample, the P2 discrepancy associated with level of schizotypy and extent of hallucination experience may reflect normal variability.

Relationship Between N1 and P2 Amplitude Differences

Generally, where a stimulus has been perceived (before reaching conscious awareness) as nonsalient and N1 is therefore reduced, forward processing is also limited. This would be reflected in a smaller P2 amplitude to a person's own thought (less salient) than to an external voice (more salient; Tremblay et al., 2001). However, N1 and P2 have each been found to consist of endogenous components (subjective mental operations, such as attention) as well as exogenous components (physical characteristics of the sound stimulus, such as loudness), which may influence their amplitudes differentially (Salisbury et al., 2010). The rest-stimulus interaction suggests that, in schizophrenia, internal auditory verbal signals elicit a salience response as if they are external and therefore will be forward processed as if external vocal stimuli. From this, the premise in this study was that as schizotypy increased there would be an increase in both N1 and P2 amplitude to the listen-self condition compared to the listen-other condition, reflecting less suppression of internal compared to external signals.

In the event, higher schizotypy was associated with a greater N1 response to listening to the voice of another person than listening to the own voice, which was against the predicted direction of the effect. However, both higher schizotypy and greater hallucination experience were associated with a greater P2 response to listening to the own voice as opposed to listening to the other voice, as predicted. The finding suggests that P2 was more dependent on mechanisms other than the N1 response and the disjunction between the peaks suggests that separate endogenous factors for each peak played a greater role than shared exogenous factors in this study.

Theoretical Implications

The results of the study provide limited support for the rest-stimulus interaction hypothesis, but the interaction could not be tested with sufficient rigour. We need to test this theory more fully by examining DMN function and by including a clinical schizophrenia sample. It is important to note that we did not find the association of reduced suppression to internal signals with schizotypy, as has been reported in the ERP literature when comparing speaking in real time to a listening condition, and that has been used to support the corollary discharge theory (Ford, Roach, et al., 2007). The fact that we find dissimilar findings for N1 and P2 amplitude, in the absence of a motor condition which would generate a corollary discharge, suggests that other processes may also interfere with the discrimination between internal and external signals. It is important to note that this study does not refute the corollary discharge theory, and future research should test both models by including an internal motor condition (speaking in real time) as well as own-voice and other-voice listening conditions.

The influences of depression, anxiety, and stress identified here are complex, but to be expected. All are common in individuals who experience hallucinations, particularly in schizophrenia (Paulik, Badcock, & Maybery, 2006), and altered functional connectivity in the DMN has been found associated with anxiety in fMRI studies (Sylvester et al., 2012). Although Paulik et al., using the Launay–Slade Hallucination Scale-Revised (LSHS-R; Bentall & Slade, 1985) scale and the DASS-21 (Lovibond & Lovibond, 1995), found anxiety was most related to predisposition to hallucinate, in this study schizotypy and hallucination experience appear to share the most variance with depression and the least with stress. With larger samples, future work might examine specific factors within the SPQ-BR to better characterise

these relationships. In addition to the discussion above on the impact of these three factors on N1 and P2 in this study, considerable interactions between depression and anxiety have been identified by previous research. For example, the effects of depression on brain function were moderated by varying degrees of co-occurring anxiety, with the direction of influence exerted by anxiety dependent on whether the anxiety was based in arousal or apprehension (Engels et al., 2010). However, research conducted in this area has largely used fMRI and focused on emotion-related tasks (Engels et al), making it difficult to assess the implications for N1 amplitude in the present study.

Limitations and Future Research

There were several important limitations to this study that need to be considered in future research. First, the sample size was too small, despite G-power analysis suggesting that a sample of 30 would be sufficient to match the effect size found by Ford, Roach, et al. (2007). Research of this nature commonly uses small samples due to difficulties in recruitment, but this is likely to contribute to inconsistency of results across studies (Salisbury et al., 2010). The formula $N \geq 50 + 8m$ (where m is the number of predictors; Tabachnik & Fidell, 2013) would seem more robust. Given that five predictors in a single model would have been useful, a minimum sample of 90 would be indicated for the study reported here. Three of these predictors were depression, anxiety, and stress. These clearly distinguished the high and low schizotypy groups and are known to be important correlates of schizophrenia. Given the suppression relationships, the substantial shared variances disclosed in the regression analyses, and the disparity of effects between N1 and P2, a larger sample would permit the correlations to be more closely dissected in future work.

Second, activation of the DMN at rest was assumed to be elevated in high schizotypy, based on previous research into schizophrenia and schizotypy (Broyd et al., 2009; Debbané et al., 2014; Nelson et al., 2013; van Os et al., 2009), and was not tested. Moreover, most research into the DMN has been conducted using fMRI and more needs to be done to identify characteristic EEG components relating to the DMN (Whitfield-Gabrieli & Ford, 2012), both in schizophrenia and schizotypy. As well, the DMN is largely characterised by abnormally high activation in a cortical task-free state. Any future study needs to include a resting state (task-free, eyes-closed) condition alongside the ERP suppression task. As well, this might be complemented by frequency domain analyses to investigate the associations between the DMN and alpha, beta, and gamma power previously demonstrated in studies using using fMRI (Chen, Feng, Zhao, Yin, & Wang, 2008; Mantin, Perrucci, Del Gratta, Romani, & Corbetta, 2007).

Third, P2 has not been researched as extensively as other ERP components. As evidence of processing of auditory stimuli prior to conscious awareness, the results presented here suggest that further investigation would be timely, including attempts to replicate results.

Fourth, as originally conceived, the study included two conditions (speaking in real time and thinking) that are not reported here due to technical difficulties. Analysis of these would have helped address some of the outstanding questions, particularly teasing out propositions to differentiate the rest-stimulus interaction and the corollary discharge model. Future work needs to resolve these technical issues prior to replication or extension of the study.

Finally, the participants here formed a nonclinical sample. While schizotypy is normally distributed in the general population and constitutes part of the

schizophrenia spectrum, it does not necessarily confer clinically-relevant deficits. The recruitment strategy was originally designed for a between-groups ANOVA approach, which lead to an artificial bimodal distribution of schizotypy and hallucination experience scores that may have influenced regression results. While schizotypy proved to be a useful discriminator, to better evaluate the rest-stimulus interaction a future study would do well to include a schizophrenia group.

Conclusions

This study tested predictions of the rest-stimulus interaction component of the resting state hypothesis, which hypothesizes that with increasing schizotypy there will be a reduced capacity to discriminate internal from external signals, such that there will be increased N1 and P2 amplitude to the listen-self condition compared to listen-other. Hierarchical regression analyses provided partial confirmation of these predictions, revealing that as schizotypy or hallucination experience increased, there was an increase in P2 amplitude to the listen-self relative to listen-other signal. However, this was observed when controlling for stress or anxiety but not depression. Future research needs to test this model whilst including an explicit test of DMN function, using a motor signal condition (hearing oneself speak in real time), an internal condition (listen-self), and an external condition (listen-other), in order to test competing predictions of corollary discharge and resting state models. The predictions need also to be tested in a clinical schizophrenia sample. Despite several important limitations in the current study, which qualify the conclusion that can be drawn, initial findings suggest further investigation of the resting state hypothesis is warranted.

References

Ahveninen, J., Jääskeläinen, I. P., Osipova, D., Huttunen, M. O., Ilmoniemi, R. J., Kaprio, J., . . . Cannon, T. D. (2006). Inherited auditory-cortical dysfunction in twin pairs discordant for schizophrenia. *Biological Psychiatry, 60*, 612-620. doi:10.1016/j.biopsych.2006.04.015

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th. ed.). Arlington, VA: Author.

Asai, T., Sugimori, E., & Tanno, Y. (2011). Auditory verbal hallucination in schizophrenic patients and the general population: The sense of agency in speech. In M. S. Payne (Ed.), *Hallucinations: Types, stages and treatments* (pp. 33-59). Hauppauge NY: Nova Science.

Bentall, R. P., & Slade, P. D. (1985). Reliability of a scale measuring disposition towards hallucination: A brief report. *Personality & Individual Differences, 6*, 527–529.

Bora, E., Fornito, A., Radua, J., Walterfang, M., Seal, M., Wood, S. J., . . . Pantelis, C. (2011). Neuroanatomical abnormalities in schizophrenia: A multimodal voxelwise meta-analysis and meta-regression analysis. *Schizophrenia Research, 127*, 46-57. doi:10.1016/j.schres.2010.12.020

Bösel, R., & Tamm, S. (2003). Brain activity measurement. In R. Fernandez-Ballesteros (Ed.), *Encylopedia of psychological assessment* (pp. 145-150). London: Sage.

Blakemore, S. J., Wolpert, D., & Frith, C. (2000). Why can't you tickle yourself? *Neuroreport, 11*, 11-16.

Broyd, S. J., Demanuele, C., Debener, S., Helps, S. K., James, C. J., & Sonuga-Barke, E. J. S. (2009). Default-mode brain dysfunction in mental disorders: A

systematic review. *Neuroscience & Biobehavioral Reviews, 33*, 279-296. doi:10.1016/j.neubiorev.2008.09.002

Bruder, G. E., Kayser, J., & Tenke, C. E. (2009). Event-related brain potentials in depression: Clinical, cognitive and neurophysiologic implications. In S. J. Luck & E. S. Kappenman (Eds.), *The Oxford handbook of event-related potential components* (pp. 563-592). New York: Oxford University Press.

Chen, A. C. N., Feng, W., Zhao, H., Yin, Y., & Wang, P. (2008). EEG default mode network in the human brain: Spectral regional field powers. *Neuroimage, 41,* 561-574. doi:10.1016/j.neuroimage.2007.12.064

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd. ed.). Mahwah, NJ: Erlbaum.

Cohen, A. S, Matthews, R. A., Najolia, G. N., & Brown, L. A. (2010). Towards a more psychometrically sound brief measure of schizotypal traits: Introducing the SPQ-Brief Revised. *Journal of Personality Disorders, 24*, 516-537.

Compumedics Neuroscan. (2003). *Stim^2. A comprehensive library of sensory, cognitive and neuropsychological tasks.* El Paso TX: Author.

Compumedics Neuroscan (2006). *SCAN 4.4 - Vol. II EDIT 4.4. Offline analysis of acquired data.* El Paso TX: Author.

Damoiseaux, J. S., Rombouts, S. A. R. B., Barkhof, F., Scheltens, P., Stam, C. J., Smith, S. M., & Beckmann, C. F. (2006). Consistent resting-state networks across healthy subjects. *Proceedings of the National Academy of Sciences, 103,* 13848-13853. doi: 10.1073/pnas.0601417103

Debbané, M., Vrtčka, P., Lazouret, M., Badoud, D., Sander, D., & Eliez, S. (2014). Self-reflection and positive schizotypy in the adolescent brain. *Schizophrenia Research, 152*, 65-72. doi.org/10.1016/j.schres.2013.06.027

Ditman, T., & Kuperberg, G. R. (2009). A source-monitoring account of auditory verbal hallucinations in schizophrenia. *Harvard Review of Psychiatry, 13*, 280-299. doi:10.1080/10673220500326391

Dragovic, M. (2004). Categorization and validation of handedness using latent class analysis. *Acta Neuropsychiatrica, 16,* 212-218.

Engels, A. S., Heller, W., Spielberg, J. M., Warren, S. L., Sutton, B. P., Banich, M. T., & Miller, G. A. (2010). Co-occurring anxiety influences patterns of brain activity in depression. *Cognitive Affective & Behavioral Neuroscience, 10*, 141-156. doi:10.3758/CABN.10.1.141

Fanous, A. H., Neale, M. C., Gardner, C.O., Webb, B. T., Straub, R. E., O'Neill, F. A., . . . Kendler, K. S. (2007). Significant correlation in linkage signals from genome-wide scans of schizophrenia and schizotypy. *Molecular Psychiatry, 12*, 958–965. doi:10.1038/sj.mp.4001996

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-191.

Feinberg, I., & Guazzelli, M. (1999). Schizophrenia - a disorder of the corollary discharge systems that integrate the motor systems of thought with the sensory systems of consciousness. *British Journal of Psychiatry, 174*, 196-204.

Ferreira-Santos, F., Silveira, C., Almeida, P. R., Palha, A., Barbosa, F., & Marques-Teixeira, J. (2012). The auditory P200 is both increased and reduced in schizophrenia? A meta-analytic dissociation of the effect for standard and target stimuli in the oddball task. *Clinical Neurophysiology, 123*, 1300-1308. doi:10.1016/j.clinph.2011.11.036

Fonseca-Pedrero, E., Paíno, M., Lemos-Giráldez, S., García-Cueto, E., Campillo-Álvarez, Á., Villazón-García, Ú., & Muñiz, J. (2008). Schizotypy assessment: State of the art and future prospects. *International Journal of Clinical & Health Psychology, 8*, 577-593.

Ford, J. M., Gray, M., Faustman, W. O., Roach, B. J., & Mathalon, D. H. (2007). Dissecting corollary discharge dysfunction in schizophrenia. *Psychophysiology, 44*, 522-529. doi: 10.1111/j.1469-8986.2007.00533.x

Ford, J. M., & Mathalon, D. H. (2005). Corollary discharge dysfunction in schizophrenia: Can it explain auditory hallucinations? *International Journal of Psychophysiology, 58*, 170-189. doi:10.1016/j.ijpsycho.2005.01.014

Ford, J. M., Mathalon, D. H., Heinks, T., Kalba, S., Faustman, W. O., & Roth, W. T. (2001). Neurophysiological evidence of corollary discharge dysfunction in schizophrenia. *American Journal of Psychiatry, 158*, 2069-2071.

Ford, J. M., Mathalon, D. H., Kalba, S., Whitfield, S., Faustman, W. O., & Roth, W. T. (2001). Cortical responsiveness during inner speech in schizophrenia: An event-related potential study. *American Journal of Psychiatry, 158*, 1914-1916.

Ford, J. M., Perez, V. B., & Mathalon, D. H. (2012). Neurophysiology of a possible fundamental deficit in schizophrenia. *World Psychiatry, 11*, 58-60.

Ford, J. M., Roach, B. J., Faustman, W. O., & Mathalon, D. H. (2007). Synch before you speak: Auditory hallucinations in schizophrenia. *American Journal of Psychiatry, 164*, 458-466.

Fox, M. D., Snyder, A. Z., Vincent, J. L., Corbetta, M., van Essen, D., & Raichle, M. E. (2005). The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proceedings of the National Academy of Sciences, 102*, 9673-9678. doi:10.1073/pnas.0504136102

Foxe, J. J., Yeap, S., Snyder, A. C., Kelly, S. P., Thakore, J. H., & Molholm, S. (2011). The N1 auditory evoked potential component as an endophenotype for schizophrenia: high-density electrical mapping in clinically unaffected first-degree relatives, first-episode, and chronic schizophrenia patients. *European Archives of Psychiatry & Clinical Neuroscience, 261*, 331-339. doi:10.1007/s00406-010-0176-0

Friston, K. J., & Frith, C. D. (1995). Schizophrenia: A disconnection syndrome? *Clinical Neuroscience, 3*, 89-97.

Graux, J., Gomot, M., Roux, S., Bonnet-Brilhault, F., Camus, V., & Bruneau, N. (2013). My voice or yours? An electrophysiological study. *Brain Topography, 26*, 72-82. doi: 10.1007/s10548-012-0233-2

Gusnard, D. A., Raichle, M. E. (2001). Searching for a baseline: Functional imaging and the resting human brain. *Nature Reviews Neuroscience, 2*, 685-694.

Harrow, M., & Jobe, T. H. (2007). Factors involved in outcome and recovery in schizophrenia patients not on antipsychotic medications: A 15-year multifollow-up study. *Journal of Nervous & Mental Diseases, 195*, 406-414. doi:10.1097/01.nmd. 0000253783.32338.6e

Henry, J. D., & Crawford, J. R. (2005). The 21 Item version of the Depression Anxiety Stress Scales (DASS-21): Normative data and psychometric evaluation in a large non-clinical sample. *British Journal of Clinical Psychology, 44*, 227-239. Retrieved from http://www2. psy.unsw.edu.au/users/jdhenry/publications/DASS21.pdf

Herzig, D. A., & Mohr, C. (2013). Stressing schizotypy: The modulating role of stress-relieving behaviours and intellectual capacity on functional hemispheric asymmetry. *Laterality, 18*, 152-178. doi.org/10.1080/1357650X.2011.638638

Hor, K., & Taylor, M. (2010). Suicide and schizophrenia: A systematic review of rates and risk factors. *Journal of Psychopharmacology, 24*, 81–90. doi:10.1177/1359786810385490

Jann, K., Kottlow, M., Dierks, T., Boesch, C., & Koenig, T. (2010). Topographic electrophysiological signatures of fMRI resting state networks. *PLoS One, 5*(9), e12945. doi:10.1371/journal.pone.0012945

Javanbakht, A., Liberzon, I., Amirsadri, A., Gjini, K., & Boutros, N. N. (2011). Event-related potential studies of posttraumatic stress disorder: a critical review and synthesis. *Biology of Mood & Anxiety Disorders, 1*, 5. doi:10.1186/2045-5380-1-5

Javitt, D. C. (2000). Intracortical mechanisms of mismatch negativity dysfunction in schizophrenia. *Audiology & Neurootology, 5*, 207–215.

Javitt, D. C. (2009). When doors of perception close: Bottom-up models of disrupted cognition in schizophrenia. *Annual Review of Clinical Psychology, 5*, 249-275. doi:10.1146/annurev.clinpsy.032408.153502

Jerbi. K., Vidal, J. R., Ossandon, T., Dalal, S. S., Jung, J., Hoffmann, D., . . . Lachaux, J.-P. (2010). Exploring the electrophysiological correlates of the default-mode network with intracerebral EEG. *Frontiers in Systems Neuroscience, 4*, 27. doi: 10.3389/fnsys.2010.00027

Jones, D. K., & Leemans, A. (2011). Diffusion Tensor Imaging. *Methods in Molecular Biology, 711*, 127-144. doi:10.1007/978-1-61737-992-5_6

Kemp, A. H., Benito, L. P., Quintana, D. S., Clark, C. R., McFarlane, A., Mayur, P., . . . Williams, L. M. (2010). Impact of depression heterogeneity on attention: An auditory oddball event related potential study. *Journal of Affective Disorders, 123*, 202-207. doi:10.1016/j.jad.2009.08.010

Ko, A. L., Darvas, F., Poliakov, A., Ojemann, J. & Sorensen, L. B. (2011). Quasi-periodic fluctuations in default mode network electrophysiology. *Journal of Neuroscience, 31*, 11728-11732. doi:10.1523/JNEUROSCI.5730-10.2011

Konrad, A., & Winterer, G. (2008). Disturbed structural connectivity in schizophrenia - primary factor in pathology or epiphenomenon? *Schizophrenia Bulletin, 34*, 72-92. doi:10.1093/schbul/sbm034

Koychev, I. G. (2011). *Neuropsychological and electrophysiological biomarkers of the schizophrenia spectrum* (Doctoral dissertation, University of Manchester). Retrieved from http://www.manchester.ac.uk/ escholar/uk-ac-man-scw:125370

Knöchel, C., O'Dwyer, L., Alves, G., Reinke, B., Magerkurth, J., Rotarska-Jagiela, A., . . . Oertel- Knöchel, V. (2012). Association between white matter fiber integrity and subclinical psychotic symptoms in schizophrenia patients and unaffected relatives. *Schizophrenia Research, 140*, 129-135. doi:10.1016/j.schres.2012.06.001

Lovibond, S.H., & Lovibond, P.F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd ed.). Sydney: Psychology Foundation. Scales retrieved from http://www2.psy. unsw.edu.au/groups/dass/down.htm

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2010). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*, 434-445. doi:10.1038/nrn2639

Mantini, D., Perrucci, M. G., Del Gratta, C., Romani, G. L., & Corbetta, M. (2007). Electrophysiological signatures of resting state networks in the human brain. *Proceedings of the National Academy of Sciences, 104,* 13170-13175. doi:10.1073/pnas.0700668104

Mathalon, D. H., & Ford, J. M. (2012). Neurobiology of schizophrenia: Search for the elusive correlation with symptoms. *Frontiers in Human Neuroscience, 6*, 136. doi:10.3389/fnhum.2012.00136

Mawson, A., Cohen, K., & Berry, K. (2010). Reviewing evidence for the cognitive model of auditory hallucinations: The relationship between cognitive voice appraisals and distress during psychosis. *Clinical Psychology Review, 30*, 248-258. doi:10.1016/j.cpr.2009.11.006

Modinos, G., Mechelli, A., Ormel, J., Groenewald, N. A., Aleman, A., & McGuire, P.K. (2010). Schizotypy and brain structure: A voxel-based morphometry study. *Psychological Medicine, 40*, 1423-131. doi:10.1017/S0033291709991875

Näätänen, R., & Kähkönen, S. (2009). Central auditory dysfunction in schizophrenia as revealed by the mismatch negativity (MMN) and its magnetic equivalent MMNn: A review. *International Journal of Neuropsychopharmacology, 12*, 125–135. doi:10.1017/S1461145708009322

Näätänen, R., Kujala, T., & Winkler, I. (2011). Auditory processing that leads to conscious perception: A unique window to central auditory processing opened by the mismatch negativity and related responses. *Psychophysiology, 48*, 4-22. doi:10.1111/j.1469-8986.2010.01114.x

Nelson, M. T., Seal, M. L., Pantelis, C., & Phillips, L. J. (2013). Evidence of a dimensional relationship between schizotypy and schizophrenia: A systematic review. *Neuroscience & Biobehavioral Reviews, 37*, 317-327. doi.org/10.1016/j.neubiorev.2013.01.004

Nelson, M. T., Seal, M. L., Phillips, L. J., Merritt, A. H., Wilson, R., & Pantelis, C. (2011). An investigation of the relationship between cortical connectivity and

schizotypy in the general population. *Journal of Nervous & Mental Disease, 199*, 348-353. doi:10.1097/nmd.0b013e318217514b

Northoff, G., & Qin, P. (2011). How can the brain's resting state activity generate hallucinations? A 'resting state hypothesis' of auditory verbal hallucinations. *Schizophrenia Research, 127*, 202-214. doi:10.1016/j.schres.2010.11.009

Northoff, G., Qin, P., & Nakao, T. (2010). Rest-stimulus interaction in the brain: A review. *Trends in Cognitive Sciences, 33*, 277-284. doi:10.1016/j.tins.2010.02.006

Ogura, C., Nageishi, Y., Matsubayashi, M., Omura, F., Kishimoto, A., & Shimokochi, M. (2008). Abnormalities in event-related potentials, N100, P200, P300 and slow wave in schizophrenia. *Psychiatry & Clinical Neurosciences, 45*, 57-65. doi:10.1111/j.1440-1819.1991.tb00506.x

van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. (2009). A systematic review and meta-analysis of the psychosis continuum: Evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychological Medicine, 39*, 179-195. doi:10.1017/S0033291708003814

Paulik, G., Badcock, J. C., & Maybery, M. T. (2006). The multifactorial structure of the predisposition to hallucinate and associations with anxiety, depression and stress. *Personality & Individual Differences, 41*, 1067-1076. doi:10.1016/j.paid.2006.04.012

Philippi, C. L. (2011). *The dynamic self: Exploring the critical role of the default mode network in self-referential processing* (doctoral dissertation). Retrieved from http://ir.uiowa.edu/etd/1254.

Pomerol-Clotet, E., Salvador, R., Sarró, S., Gomar, J., Vila, F., Martínez, Á., . . . McKenna, P. J. (2008). Failure to deactivate in the prefrontal cortex in schizophrenia: Dysfunction of the default mode network? *Psychological Medicine, 38*, 1185-1193. doi:10.1017/SO033291708003565

Raichle, M. E., MacLeod, A. M., Snyder, A. Z., Powers, W. J., Gusnard, D. J., & Shulman, G. L. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences, 98*, 676-682. doi:10.1073/pnas.98.2.676

Raichle, M. E., & Mintun, M. A. (2006). Brain work and brain imaging. *Proceedings of the National Academy of Sciences, 29,* 449-476. doi:10.1146/annurev.neuro. 29.051605.112819

Ranson, V. A. (2011). *Testing the resting state hypothesis of auditory verbal hallucinations in schizophrenia* (Unpublished undergraduate thesis). University of New England, Armidale, Australia.

Rosazza, C., & Minati, L. (2011). Resting-state brain networks: Literature review and clinical applications. *Neurological Sciences, 32,* 773-785. doi:10.1007/s10072-011-0636-y

Rosburg, T., Zimmerer, K., & Huonker, R. (2010). Short-term habituation of auditory evoked potential and neuromagnetic field components in dependence of the interstimulus interval. *Experimental Brain Research, 205*, 559-570. doi: 10.1007/s00221-010-2391-3

Saha, S., Chant, D., Welham, J., & McGrath, J. (2005). A systematic review of the prevalence of schizophrenia. *PLoS Medicine, 2* (5), e141. Retrieved from http://tinyurl.com/36795c

Sakkalis , V. (2011). Review of advanced techniques for the estimation of brain connectivity. *Computers in Biology & Medicine, 41*, 1110-1117. doi:10.1016/j.compbiomed.2011.06.020

Salisbury, D. F., Collins, K. C., & McCarley, R. W. (2010). Reductions in the N1 and P2 auditory event-related potentials in first-hospitalized and chronic schizophrenia. *Schizophrenia Bulletin, 36*, 991-1000. doi:10.1093/schbul/sbp003

Semlitsch, H. V., Anderer, P., Schuster, P., & Presslich, O., (1986). A solution for reliable and valid reduction of ocular artifacts applied to the P300 ERP, *Psychophysiology, 23,* 695-703. doi:10.1111/j.1469-8986.1986.tb00696.x

Sheline, Y. L., Price, J. L., Yan, Z., & Mintun, M. A. (2010). Resting-state functional MRI in depression unmasks increased connectivity between networks via the dorsal nexus. *Proceedings of the National Academy of Sciences, 107*, 11020-11025. doi:10.1073/pnas.1000446107

Shin, Y. W., Krishnan, G., Hetrick, W. P., Brenner, C. A., Shekhar, A. Malloy, F. W., & O'Donnell, B. F. (2010). Increased temporal variability of auditory event related potentials in schizophrenia and schizotypal personality disorder. *Schizophrenia Research, 124*, 110-118. doi:10.1016/j.schres.2010.08.008

Shulman, G. L., Fiez, J. A., Corbetta, M., Buckner, R. L., Miezin, F. M., Raichle, M. E., & Petersen, S. E. (1997). Common blood flow changes across visual tasks: II. Decreases in cerebral cortex. Retrieved from http://www.nil.wustl.edu/labs/corbetta/PDFs%20for%20Web/Common%20blood%20flow%20changes%20across%20visual%20tasks-%20II.%20Decreases%20in%20 cerebral%20cortex.pdf

Sowman, P. F., Kuusik, A.,& Johnson, B. W. (2012). Self-initiation and temporal cueing of monaural tones reduce the auditory N1 and P2. *Experimental Brain Research, 222*, 149-157. doi:10.1007/s00221-012-3204-7

Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd. ed.). Cambridge, UK: Cambridge University Press.

Stekelenburg, J. J., Maes, J. P., Van Gool, A. R., Sitskoorn, M., & Vroomen, J. (2013). Deficient multisensory integration in schizophrenia: An event-related potential study. *Schizophrenia Research, 147*, 253-261. doi.org/10.1016/j.schres.2013.04.038

Stephane, K. E., Friston, K. J., & Frith, C. D. (2009). Dysconnection in schizophrenia: From abnormal synaptic plasticity to failures of self-monitoring. *Schizophrenia Bulletin, 35*, 509-527. doi:10.1093/schbul/sbn176

Swanson, N., Eichele, T., Pearlson, G., Kiehl, K., Yu, Q., & Calhoun, V. D. (2010). Lateral differences in the default mode network in healthy controls and patients with schizophrenia. *Human Brain Mapping, 32,* 654-664. doi:10.1002/hbm.21055

Sugimori, E., Asai, T., & Tanno, Y. (2009). Article in Japanese [Reliability and validity of the Auditory Hallucination-like Experience Scale]. *Shinrigaku Kenkyu [Japanese Journal of Psychology], 80*(5), 389-396.

Sylvester, C. M., Corbetta, M., Raichle, M. E., Rodebaugh, T., Schlaggar, B. L., Sheline, Y. I., . . . & Lenze, E. J. (2012). Functional network dysfunction in anxiety and anxiety disorders. *Trends in Neurosciences, 35*, 527-535. doi:10.1016/j.tins.2012.04.012

Tabachnik, B. G., & Fidell, L. S. (2013). *Using multivariate statistics* (6th. ed.). Boston, MA: Pearson.

Towle, V. L., Bolaños, J., Suarez, D., Tan, K., Grzeszczuk, R., Levin, D. N.,...Spire, J.-P. (2003). The spatial location of EEG electrodes: Locating the best-fitting sphere relative to cortical anatomy [Abstract]. *Electroencephalography & Clinical Neurophysiology, 86*, 1-6. doi.org/10.1016/0013-4694(93)90061-Y

Tremblay, K., Kraus, N., McGee, T., Ponton, C., & Otis, B. (2001). Central auditory plasticity: Changes in the N1-P2 complex after speech-sound training. *Ear & Hearing, 22*, 79-90.

Veale, J. F. (2014). Edinburgh Handedness Inventory - Short Form: A revised version based on confirmatory factor analysis. *Laterality, 19*, 164-177. doi.org/10.1080/1357650X.2013.783045

Wang, L., Metzak, P. D., & Woodward, T. S. (2011). Aberrant connectivity during self–other source monitoring in schizophrenia. *Schizophrenia Research, 125*, 136–142. doi:10.1016/j.schres.2010.11.012

Waters, F., Allen, P., Aleman, A., Fernyhough, C., Woodward, T. S., Badcock, J. C., . . . Larøi, F. (2012). Auditory hallucinations in schizophrenia and nonschizophrenia populations: A review and integrated model of cognitive mechanisms. *Schizophrenia Bulletin, 38*, 683-692. doi:10.1093/schbul/sbs045

Whitfield-Gabrieli, S., & Ford, J. M. (2012). Default mode network activity and connectivity in psychopathology. *Annual Review of Clinical Psychology, 8*, 49-76. doi:10.1146/annurev-clinpsy-032511-143049

Whitfield-Gabrieli, S., Thermenos, H. W., Milanovic, S., Tsuang, M. T., Faraone, S. V., McCarley, R. W., . . . Seidman, L. J. (2009). Hyperactivity and hyperconnectivity of the default mode network in schizophrenia and in first-degree relatives of persons with schizophrenia. *Proceedings of the National Academy of Sciences, 106*, 1279-1284. doi:10.1073/pnas.0809141106

Whitford, T. J., Ford, J. M., Mathalon, D. H., Kubicki, M., & Shenton, M. E. (2012). Schizophrenia, myelination, and delayed corollary discharges: A hypothesis. *Schizophrenia Bulletin, 38*, 486-494. doi:10.1093/schbul/sbq105

Whitford, T. J., Kubicki, M., & Shenton, M. E. (2011). Diffusion tensor imaging, structural connectivity, and schizophrenia. *Schizophrenia Research & Treatment,* Article ID 709523, 7 pages. doi:10.1155/2011/709523

Whitford, T. J., Mathalon, D. H., Shenton, M. E., Roach, B. J., Bammer, R., Adcock, R. A., . . . Ford, J. M. (2011). Electrophysiological and diffusion tensor imaging evidence of delayed corollary discharges in patients with schizophrenia. *Psychological Medicine, 41*, 959-969. doi:10.1017/S0033291710001376

Williams, S. M. (2010). *A major revision of the Edinburgh handedness inventory.* Retrieved from http://homepage.ntlworld.com/steve.williams7/page9.html

Wolf, N. D., Sambataro, F., Vasic, N., Frasch, K., Schmid, M., Schönfeldt-Lecuona, C., . . . Wolf, R. C. (2011). Dysconnectivity of multiple resting-state networks in patients with schizophrenia who have persistent auditory verbal hallucinations. *Journal of Psychiatry & Neuroscience, 36*, 366-374. doi: 10.1503/jpn.110008

Appendices

A Letter of approval from the Human Research Ethics Committee.

B Participant information sheet.

C Participant consent form.

D Schizotypal Personality Questionnaire - Brief Revised version (SPQ-BR).

E Demographics questionnaire.

F Depression Anxiety Stress Scales - 21 item version (DASS-21).

G Auditory Hallucination-like Experiences Scale (AHES).

H G*power 3 analysis.

I Regression tables for electrode sites FZ and CZ.

J CD: Raw data spreadsheets and SPSS analyses.

Appendix A

Letter of approval from the Human Research Ethics Committee.

Social Science Ethics Officer
Private Bag 01 Hobart
Tasmania 7001 Australia
Tel: (03) 6226 2763
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Katherine.Shaw@utas.edu.au

HUMAN RESEARCH ETHICS COMMITTEE (TASMANIA) NETWORK

4 July 2012

Dr Kim Felmingham
School of Psychology
University of Tasmania
Private Bag 30
Hobart Tasmania

Student Researcher: Valerie Ranson

Dear Dr Felmingham

Re: FULL ETHICS APPLICATION APPROVAL
Ethics Ref: H0012495 - The impact of the default mode network on misattribution of external and internal auditory signals in schizophrenia

We are pleased to advise that the Tasmania Social Sciences Human Research Ethics Committee approved the above project on 3 July 2012.

This approval constitutes ethical clearance by the Tasmania Social Sciences Human Research Ethics Committee. The decision and authority to commence the associated research may be dependent on factors beyond the remit of the ethics review process. For example, your research may need ethics clearance from other organisations or review by your research governance coordinator or Head of Department. It is your responsibility to find out if the approval of other bodies or authorities is required. It is recommended that the proposed research should not commence until you have satisfied these requirements.

Please note that this approval is for four years and is conditional upon receipt of an annual Progress Report. Ethics approval for this project will lapse if a Progress Report is not submitted.

The following conditions apply to this approval. Failure to abide by these conditions may result in suspension or discontinuation of approval.

1. It is the responsibility of the Chief Investigator to ensure that all investigators are aware of the terms of approval, to ensure the project is conducted as approved by the Ethics Committee, and to notify the Committee if any investigators are added to, or cease involvement with, the project.

2. Complaints: If any complaints are received or ethical issues arise during the course of the project, investigators should advise the Executive Officer of the Ethics Committee on 03 6226 7479 or human.ethics@utas.edu.au.

3. Incidents or adverse effects: Investigators should notify the Ethics Committee immediately of any serious or unexpected adverse effects on participants or unforeseen events affecting the ethical acceptability of the project.

4. Amendments to Project: Modifications to the project must not proceed until approval is obtained from the Ethics Committee. Please submit an Amendment Form (available on our website) to notify the Ethics Committee of the proposed modifications.

5. Annual Report: Continued approval for this project is dependent on the submission of a Progress Report by the anniversary date of your approval. You will be sent a courtesy reminder closer to this date. Failure to submit a Progress Report will mean that ethics approval for this project will lapse.

6. Final Report: A Final Report and a copy of any published material arising from the project, either in full or abstract, must be provided at the end of the project.

Yours sincerely

Katherine Shaw
Ethics Officer
Tasmania Social Sciences HREC

Appendix B

Participant information sheet.

UTAS

Research project

The impact of the default mode network on misattribution of external and internal auditory signals in schizophrenia.

Participant Information Sheet

Group C/D: High/Low Schizotypy Participants

1. Invitation

My name is Val Ranson. I would like to invite you to participate in research into the processing of auditory signals in schizophrenia. The details of the study follow and I hope you will consider being involved. I am a postgraduate student at the University of Tasmania and am conducting this research as part of the Masters in Clinical Psychology degree. My research supervisors are Dr. Kim Felmingham and Dr Andrea Carr, in the School of Psychology, University of Tasmania.

2. What is the purpose of this study?

The aim of this study is to examine how the brain processes auditory signals in people with schizophrenia compared to those who do not have a mental illness. In particular, we are interested in studying how the brain processes auditory signals that have been generated by a person themselves, compared to those that have been generated by other people. That is, how your brain responds to your own voice, compared to how it responds to someone else's.

3. Why have I been invited to participate?

The study will be comparing the brain activity of individuals with schizophrenia with the brain activity of individuals who don't have a mental illness. You have been given this Information Sheet because you have responded to the Schizotypal Personality Questionnaire and expressed interest in participating in the study.

I would like to recruit people who are:

- Over 18 years of age.
- Of normal hearing (hearing impairment may change how the brain processes sound).
- Who score either high or low on a measure of schizotypal personality.

Participation in the research is completely voluntary and there will be no consequences to your involvement with the University of Tasmania, if you decide not to proceed or if you decide to withdraw at any point.

4. What will I be asked to do?

If you agree to participate in this study, you will be asked to attend a two-hour testing session in the School of Psychology, University of Tasmania. Prior to commencement of the study, you will be asked to sign a consent form which will document your agreement to participate. You will be asked to fill in a few questionnaires about your mood, perceptual experiences (for example, whether you have ever heard voices when there was no one there), and a demographic questionnaire.

You will then be fitted with an EEG cap. This has electrodes resting against your hair that measure the electrical activity running across your scalp. EEG is a painless, non-invasive, and safe procedure. You will be trained to produce an auditory sound (saying the word "ah") and this will be recorded and played back to you whilst your brain activity is recorded. We will also play you the same auditory syllable that has been recorded by another person and measure your brain activity. We will also ask you to speak the same syllable while we record your brain's activity. We will also ask you to imagine the same auditory syllable whilst we record your brain activity. We will then remove the electrode cap and answer any questions that you have about the study.

5. Are there any possible benefits from participation in this study?

If you decide to participate in this research you will be helping us develop our knowledge about how the brain functions in schizophrenia. This research will provide a foundation upon which we can have a better understanding of the mechanisms associated with schizophrenia.

If you are a psychology first year student, you will be eligible to receive two hours of credit for your research participation. Other participants will be reimbursed $50 for their time and travel costs.

6. Are there any possible risks from participation in this study?

This study involves minimal risk to participants. You may feel some mild discomfort in wearing the EEG electrode cap. While the equipment used to measure brain activity may feel a little uncomfortable, it is not painful and cannot affect your brain in any way. However if you have sensitive skin, you should inform the researcher.

It is possible that you may get fatigued and, to alleviate this, rests will be given during the experimental session.

This study involves minimal risk, but if you become distressed at any time, you will be offered the option of accessing external support at the University Psychology Clinic at the University of Tasmania. This service is free of charge. Appointments can be made by contacting the clinic on (03) 6226 2805.

7. What if I change my mind during or after the study?

You are free to withdraw from the study at any time without penalty. That is, your decision to withdraw from the study will not affect your involvement with

community and support services, or with the University of Tasmania. You can withdraw from the study without explanation. If you decide to withdraw from the study, the data collected from you during the study will be destroyed immediately.

8. What will happen to the information when this study is over?

Your individual data will be treated confidentially. Your name will not be recorded with any data, except the Informed Consent form. Instead, your data will be given an ID number. The data will be kept in locked cabinets or on password secured computers at the School of Psychology at the University of Tasmania for a period of at least five years (with the exception of the medical questionnaires which will be destroyed on completion of the study).

9. How will the results of the study be published?

Following completion of the research, the data will be published in peer-reviewed journal articles and in conference presentations. No participant will be personally identifiable in these publications as only group data will be published. A summary of the results of these experiments will be available on the University of Tasmania School of Psychology Web page at www.scieng.utas.edu.au/psychol or will be available by contacting the researchers.

10. What if I have questions about this study?

If you have any questions about the study, you can contact the researcher, Val Ranson, by email at val.ranson@utas.edu.au or phone 04 ....., or Dr Kim Felmingham by email on Kim.Felmingham@utas.edu.au, or Dr Andrea Carr by email on A.R.Carr@utas.edu.au

This study has been approved by the Tasmanian Social Sciences Human Research Ethics Committee (Approval No. H0012495 valid to 02/07/2016).

Should you have any concerns or complaints about the conduct of this study, please contact the Executive Officer of the HREC (Tasmania) Network on (03) 6226 7479 or email human.ethics@utas.edu.au. The Executive Officer is the person nominated to receive complaints from research participants. Please quote ethics reference number H0012495.

Please keep this Information Sheet in case you wish to refer back to it later.

If you would like more information or if you wish to participate in the study, do please contact me.

Before carrying out the EEG procedure, I will answer any further questions you may have and will ask you to sign an Informed Consent form. The Informed Consent form specifies the information that must be fully explained to you.

Appendix C

Participant consent form.

UTAS

Participant Consent Form

The impact of the default mode network on misattribution of external and internal auditory signals in schizophrenia.

Participant Consent Statement:

1. I agree to take part in the research study named above.
2. I have read and understood the Information Sheet for this study.
3. The nature and possible effects of the study have been explained to me.
4. Any questions that I have asked have been answered to my satisfaction.
5. I understand that the study requires me to attend the School of Psychology where my brain activity will be recorded while I listen to auditory signals that have been recorded by myself or other people I also understand that my involvement in this study is expected to take no longer than two hours.
6. I understand that I will be asked about recreational drug habits, use of prescription medication and any psychiatric and neurological conditions. I also understand that I should indicate to the experimenter if I have sensitive skin and that I should request a rest if I become fatigued.
7. I understand that all research data will be treated as confidential. I agree that research data gathered for the study may be published provided that I cannot be identified as a participant.
8. I understand that my participation is voluntary and that I may withdraw from participation and/or withdraw my data at any time without prejudice to my academic standing or involvement in community services.

Participant's name: _____

Participant's signature: _____Date: _____

Investigator Statement

I have explained this research and the implications of participation in it to this volunteer and I believe that the consent is informed and that he or she understands the implications of participation

Investigator's name: _____

Investigator's signature: _____Date: _____

Appendix D

Schizotypal Personality Questionnaire - Brief Revised version (SPQ-BR).

Project: The impact of the default mode network on misattribution of external and internal auditory signals in schizophrenia

Approved by the University of Tasmania Human Research Ethics Committee
H0012495, valid to 03/07/2016

Schizotypal Personality Questionnaire, Brief, Revised version

For each item, please tick the box that applies to you

Item	Strongly Disagree	Disagree	Neutral	Agree	Strongly Agree
1. Are your thoughts sometimes so strong that you can almost hear them?					
2. I rarely laugh and smile.					
3. Do you feel that you cannot get "close" to people?					
4. Do you sometimes feel that people are talking about you?					
5. I sometimes avoid going to places where there will be many people because I will get anxious					
6. Do you tend to wander off the topic when having a conversation?					
7. I sometimes jump quickly from one topic to another when speaking.					
8. I sometimes forget what I am trying to say.					
9. I feel very uncomfortable in social situations involving unfamiliar people.					
10. I am an odd, unusual person.					
11. you often have to keep an eye out to stop people from taking advantage of you?					
12. Other people see me as slightly eccentric (odd).					
13. Have you had experiences with astrology, seeing the future, UFO's, ESP, or a sixth sense?					
14. I often ramble on too much when speaking.					
15. Do you believe in clairvoyance (psychic forces, fortune telling)?					
16. Do you sometimes get concerned that friends or co-workers are not really loyal or trustworthy?					
17. I have some eccentric (odd) habits.					
18. I tend to keep my feelings to myself.					
19. Do everyday things seem unusually large or small?					
20. Do you believe in telepathy (mind-reading)?					

21. When shopping do you get the feeling that other people are taking notice of you?					
22. Do you feel that there is no one you are really close to outside of your immediate family, or people you can confide in or talk to about personal problems?					
23. Do you often feel nervous when you are in a group of unfamiliar people?					
24. Do you sometimes feel that other people are watching you?					
25. I often feel that others have it in for me.					
26. When you look at a person or yourself in a mirror, have you ever seen the face change right before your eyes?					
27. I am not good at expressing my true feelings by the way I talk and look.					
28. Have you ever felt that you are communicating with another person telepathically (by mind-reading)?					
29. I find it hard to be emotionally close to other people.					
30. I often hear a voice speaking my thoughts aloud.					
31. People sometimes comment on my unusual mannerisms and habits.					
32. I get anxious when meeting people for the first time.					

Cohen, A. S, Russell, R. A., Najolia, G. N., & Brown, L. A. (2010). Towards a more psychometrically sound brief measure of schizotypal traits: Introducing the SPQ-Brief Revised. *Journal of Personality Disorders, 24*, 516-537.

Are you male? ☐ **or female?** ☐

If you are prepared to participate in the full study, please complete the following: -

Name:

Phone number or email address:

Thank you!
Your assistance with this study is greatly appreciated.

Appendix E

Demographics questionnaire.

UTAS
University of Tasmania

Dr Kim Felmingham (Chief Investigator, Senior Lecturer, School of Psychology)
Dr Andrea Carr (Investigator, Associate Lecturer, School of Psychology)
Ms Val Ranson (Student Investigator, School of Psychology)

Research project

The impact of the default mode network on misattribution of external and internal auditory signals in schizophrenia

Participant number: _____

Personal details form

On the form overleaf, please enter the information requested or

tick whichever options apply to you.

Please let me know if you feel unable to answer any of these questions.

Please note

This form is anonymous in order to protect your personal information.

It will only be accessed by the project researchers and will be securely destroyed after five years.

You may withdraw at any point during the research procedure.

1. What sex are you?	Male _____ Female _____
1. How old are you?	Years _____ Months _____

2. What relationship do you have to a person with a mental illness?	
A. I have a diagnosis of schizophrenia myself.	A. _____
B. My mother/father, son/daughter, or brother/sister has a mental illness.	B. _____
C. I have no family relationship with a person with a mental illness.	C. _____

3. If you have schizophrenia	
i. How old were you when first diagnosed with a mental illness?	Years _____
ii. How many years have you been receiving antipsychotic medication?	Years _____
iii. Are you currently taking an antipsychotic?	Yes ____ No _____

4. Have you ever had any of the following?	
i. A diagnosis of mental illness other than schizophrenia.	Yes ____ No _____
ii. A serious head injury that might still affect you in some way.	Yes ____ No _____
iii. Any other neurological disorder, e.g. epilepsy, stroke, brain tumour.	Yes ____ No _____
iv. A developmental disorder, e.g. learning difficulty, autism.	Yes ____ No _____
v. Heavy use of drugs and/or alcohol in the past 12 months.	Yes ____ No _____
vi. A need to use a hearing aid.	Yes ____ No _____
vii. Other medication that might influence your brain in some way.	Yes ____ No _____
If you ticked 'yes' to any of these, please briefly describe below:	

5. Effects of medication

Do you have tardive dyskinesia as a result of antipsychotic medication? Yes ____

If yes, is the effect Slight _____ Moderate _____ Severe _____

6. If you are currently taking medication for schizophrenia Is your response to it (*please tick*) Stable _____ Unstable _____
7. Do you smoke tobacco?
8. Are you currently employed? If so, in what position?
9. What is your highest level of education?
10. Are you living at home or independently in the community?

Approved by the University of Tasmania Human Research Ethics Committee
H0012495, valid to 03/07/2016

Appendix F

Depression Anxiety Stress Scales - 21 item version (DASS-21).

DASS21

Participant number:

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all

1 Applied to me to some degree, or some of the time

2 Applied to me to a considerable degree, or a good part of time

3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

(Lovibond & Lovibond. 1995)

Approved by the University of Tasmania Human Research Ethics Committee
H0012495, valid to 03/07/2016

Appendix G

Auditory Hallucination-like Experiences Scale (AHES).

UTAS

Research project

Misattribution of auditory signals in schizophrenia

Auditory Hallucination-like Experience Scale

Please rate each of the statements below according to your experience throughout your life.

1 = I have never experienced this.

2 = I have hardly ever experienced this.

3 = I have sometimes experienced this.

4 = I have quite often experienced this.

5 = I have very often experienced this.

Statement	Score
When on my own, I have laughed aloud in response to thoughts that I was having.	
I have experienced positive voices in my head.	
Random voices or noises around me have seemed significant to me.	
I have heard, as if in my ear, things commonly said by people important to me or often seen by me.	
I have thought that I had been absent-minded when I hadn't (e.g. forgot to lock a door, turn off the stove, or sign my name).	
When I have recalled conversations with others, I have felt that I could actually hear them speak.	
I have heard people talking and thought they were speaking about me or speaking badly of me.	
I have mistaken sounds for voices.	
On the point of going to sleep or on the point of waking up, I have felt paralysed and could not move.	
When buying a product in a shop, I have felt that I could hear the TV commercial for that product.	
When in a crowd, I have turned round because I thought that I heard my name called.	
Although voices were close by, it has seemed as if they were coming from a distance.	
I have imagined a conversation with another person.	
When a problem arose, I was able to clearly express my thoughts and feelings in words.	
I have heard jingles from TV commercials repeating in my head.	
I have had random thoughts that were hard to get rid of.	

When I have been alone, I have thought I heard someone call my name.	
I have heard music repeating in my head.	
My thoughts were so strong such that I felt I could actually hear them.	
I have felt like putting other people down or have felt like breaking things, and could not get rid of those thoughts.	
On the point of going to sleep or on the point of waking up, people or events that had occurred during the day have appeared again in front of me.	
When I have drunk alcohol, or the morning after I have drunk alcohol, I have felt like I was in a dream or I saw or heard things that weren't there, even though I was awake.	
On the point of going to sleep or on the point of waking up, I have heard my name being called or have heard someone else speaking.	
I have felt that I have been good at memorising music or tunes and that I have seldom forgotten what I have heard once.	
I have heard music in my head that was difficult to get rid of.	
I could actually hear sounds that I imagined.	
My ears have been very sensitive, when even the slightest sound has bothered me.	
I was convinced that I heard something that I could not actually have heard.	
I have thought things out loud and talked to myself when I did this.	
I have thought that I could hear the voice of something like God or a good spirit.	
Even though awake, I have felt like I was in a dream and saw or heard things that weren't real or had other unusual experiences.	
In times of trouble, I have spoken firmly to myself, in order to support and encourage myself.	
I have experienced voices in my head tempting me towards the negative side of my nature.	
I have felt that my family or I was in danger, and I could not get rid of the thought.	
I have seen people laughing and felt that they were laughing at me.	
I have thought that I could hear the voice of something like the devil or a bad spirit.	
I have imagined a conversation with another person, in order to clarify my own thoughts.	
I have heard sounds that were not really there (e.g. of vehicles or of birds chirping).	
I have heard a voice when nobody was there.	
I have felt that my voice was remote from me.	

Translated from Sugimori, E., Asai, T., & Tanno, Y. (2009). Reliability and validity of the auditory hallucination-like experience scale. [Article in Japanese.] *Shinrigaku Kenkyu, 80*(5), 389-396.

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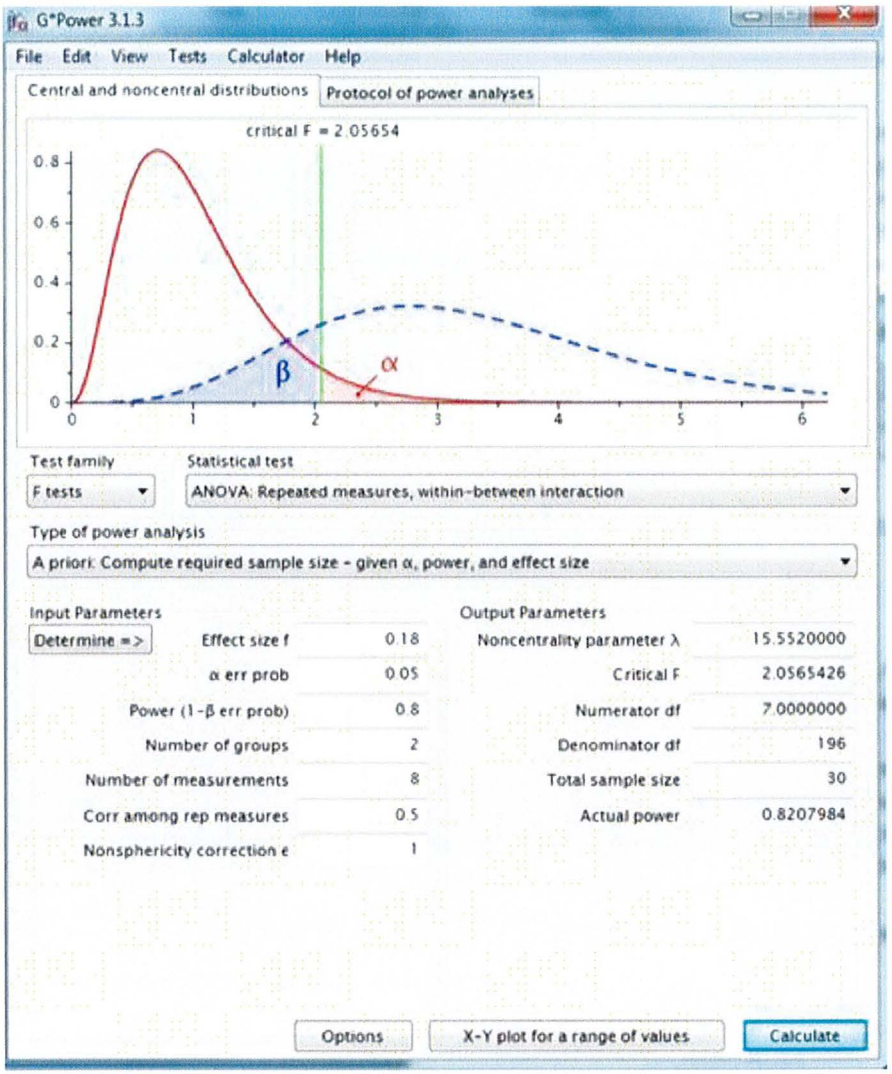
Appendix H

G3-power analysis


G*Power 3.1.3
File Edit View Tests Calculator Help
Central and noncentral distributions
Protocol of power analyses
critical F = 2.05654
0.8
0.6
0.4
0.2
0
0
1
2
3
4
5
6
β
α
Test family
F tests
Statistical test
ANOVA: Repeated measures, within-between interaction
Type of power analysis
A priori: Compute required sample size – given α, power, and effect size
Input Parameters
Determine =>
Effect size f 0.18
α err prob 0.05
Power (1-β err prob) 0.8
Number of groups 2
Number of measurements 8
Corr among rep measures 0.5
Nonsphericity correction ε 1
Output Parameters
Noncentrality parameter λ 15.5520000
Critical F 2.0565426
Numerator df 7.0000000
Denominator df 196
Total sample size 30
Actual power 0.8207984
Options
X-Y plot for a range of values
Calculate

Appendix I

Regression tables for electrode sites FZ and CZ.

Table I1

Hierarchical Regression Analyses Predicting N1 Amplitude Difference From Schizotypy, Controlling Separately for Depression, Anxiety, and Stress, at Electrode FZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.88	0.47		-1.87	.07	[-1.84, 0.09]
	Depression	0.08	0.04	.39	2.15	.04	[0.00, 0.15]
	$\overline{R}^2$ = .11, $F(1, 26) = 4.61$, $p = .04$						
Step 2	Constant	-1.70	1.06		1.61	.12	[-3.88, 0.47]
	Depression	0.05	0.05	.24	0.99	.33	[-0.05, 0.15]
	Schizotypy	0.01	0.02	.22	0.87	.39	[-0.02, 0.05]
	$\overline{R}^2$ = .11; $F(2, 25) = 2.66$, $p = .09$			$\Delta\overline{R}^2$ = .03, $\Delta F(1, 25) = 0.76$, $p = .39$			

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.58	0.46		-1.28	.21	[-1.52, 0.35]
	Anxiety	0.05	0.04	.27	1.43	.16	[-0.02, 0.13]
	$\overline{R}^2$ = .04, $F(1, 26) = 2.06$, $p = .16$						
Step 2	Constant	-2.03	1.10		-1.84	.08	[-4.29, 0.24]
	Anxiety	0.01	0.05	.03	-0.11	.91	[-0.10, 0.11]
	Schizotypy	0.02	0.02	.36	1.44	.16	[-0.01, 0.06]
	$\overline{R}^2$ = .08; $F(2, 25) = 2.10$, $p = .14$			$\Delta\overline{R}^2$ = .07, $\Delta F(1, 25) = 2.07$, $p = .16$			

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.44	0.59		-0.76	.46	[-1.65, 0.76]
	Stress	0.02	0.04	.13	0.66	.52	[-0.05, 0.09]
	$\overline{R}^2$ =0*, $F(1, 26) = 0.43$, $p = .52$						
Step 2	Constant	-2.33	1.02		-2.29	.03	[-4.43, -0.23]
	Stress	-0.04	0.04	-.24	-0.98	.34	[-0.14, 0.05]
	Schizotypy	0.04	0.02	.54	2.20	.04	[0.00, 0.07]
	$\overline{R}^2$ = .11; $F(2, 25) = 2.66$, $p = .09$			$\Delta\overline{R}^2$ = .16, $\Delta F(1, 25) = 4.82$, $p = .09$			

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety).

Table I2

Hierarchical Regression Analyses Predicting N1 Amplitude Difference From Schizotypy, Controlling Separately for Depression, Anxiety, and Stress, at Electrode CZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.40	0.43		-0.93	.36	[-1.28, 0.48]
	Depression	0.85	0.03	.46	2.67	.01	[0.02, 0.15]
	$\overline{R}^2$ = .19, $F(1, 26) = 7.14$, $p = .01$						
Step 2	Constant	-0.75	0.98		-0.77	.45	[-2.77, 1.27]
	Depression	0.07	0.04	.40	1.67	.11	[-0.02, 0.16]
	Schizotypy	0.01	0.02	.10	0.40	.69	[-0.03, 0.04]
	$\overline{R}^2$ = .16; $F(2, 25) = 3.53$, $p = .04$			$\Delta\overline{R}^2$ = .01, $\Delta F(1, 25) = 0.16$, $p = .69$			

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.13	0.4		0.28	.78	[-0.78, 1.03]
	Anxiety	0.04	0.04	.21	1.07	.29	[-0.04, 0.11]
	$\overline{R}^2$ = .01, $F(1, 26) = 1.15$, $p = .29$						
Step 2	Constant	-1.47	1.05		-1.39	.18	[-3.63, 0.70]
	Anxiety	-0.01	0.05	-.08	-0.30	.77	[-0.11, 0.08]
	Schizotypy	0.03	0.02	.42	1.66	.11	[-0.01, 0.06]
	$\overline{R}^2$ = .07; $F(2, 25) = 1.98$, $p = .16$			$\Delta\overline{R}^2$ = .10, $\Delta F(1, 74) = 2.28$, $p = .11$			

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.18	0.56		0.32	.75	[-0.97, 1.33]
	Stress	0.02	0.03	.12	0.60	.55	[-0.05, 0.09]
	$\overline{R}^2$ =0*, $F(1, 26) = 0.36$, $p = .55$						
Step 2	Constant	-2.00	1.06		-1.89	.07	[-3.59, 0.43]
	Stress	-0.04	0.05	-.20	-0.82	.42	[-0.13, 0.05]
	Schizotypy	0.03	0.02	.51	2.02	.05	[0.00, 0.07]
	$\overline{R}^2$ = .10; $F(2, 25) = 2.49$, $p = .10$			$\Delta\overline{R}^2$ = .15, $\Delta F(1, 25) = 4.57$, $p = .04$			

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety).

Table I3

Hierarchical Regression Analyses Predicting P2 Amplitude Difference From Schizotypy, Controlling Separately for Depression, Anxiety, and Stress, at Electrode FZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.64	0.58		1.11	.28	[-0.55, 1.84]
	Depression	0.00	0.04	-.01	-0.06	.96	[-0.09, 0.09]
	$\overline{R}^2$ = 0*, $F(1, 26) < 0.01$, $p = .96$						
Step 2	Constant	-1.23	2.28		-0.54	.60	[-2.57, 2.90]
	Depression	0.00	0.10	.01	0.04	.97	[-0.14, 0.10]
	Schizotypy	0.02	0.03	.16	0.61	.55	[-0.03, 0.05]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.08$, $p = .92$				$\Delta\overline{R}^2 = .01$, $\Delta F(1, 25) = 0.16$, $p = .69$		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.91	0.54		1.69	.10	[-0.20, 2.00]
	Anxiety	-0.03	0.04	-.15	-0.77	.45	[-0.12, 0.06]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.59$, $p = .45$						
Step 2	Constant	-0.38	1.32		-0.29	.78	[-3.08, 2.33]
	Anxiety	-0.08	0.06	-.34	-1.29	.21	[-0.20, 0.05]
	Schizotypy	0.02	0.02	.28	1.07	.30	[-0.02, 0.06]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.86$, $p = .43$				$\Delta\overline{R}^2 = .04$, $\Delta F(1, 25) = 1.13$, $p = .30$		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	1.13	0.66		1.70	.10	[-0.23, 2.50]
	Stress	-0.04	0.04	-.18	-0.94	.35	[-0.12, 0.04]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.89$, $p = .35$						
Step 2	Constant	-2.29	2.04		-1.12	.27	[-2.69, 2.35]
	Stress	-0.18	0.09	-.50	-1.99	.06	[-0.19, 0.03]
	Schizotypy	0.07	0.03	.51	2.04	.05	[-0.02, 0.06]
	$\overline{R}^2$ = .02; $F(2, 25) = 1.25$, $p = .31$				$\Delta\overline{R}^2 = .06$, $\Delta F(1, 25) = 1.58$, $p = .22$		

Note. n = 28 in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety).

Table I4

Hierarchical Regression Analyses Predicting P2 Amplitude Difference From Schizotypy, Controlling Separately for Depression, Anxiety, and Stress, at Electrode CZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.60	1.07		-0.56	.58	[-2.80, 1.60]
	Depression	0.07	0.08	.16	0.82	.42	[-0.10, 0.23]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.68$, $p = .42$						
Step 2	Constant	-2.27	2.42		-0.94	.36	[-7.26, 2.72]
	Depression	0.01	0.11	.02	0.08	.94	[-0.22, 0.23]
	Schizotypy	0.03	0.04	.20	0.77	.45	[-0.05, 0.10]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.63$, $p = .54$				$\Delta\overline{R}^2 = .02$, $\Delta F(1, 25) = 0.59$, $p = .45$		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.15	1.01		0.15	.88	[-1.92, 2.22]
	Anxiety	-0.01	0.08	-.03	-0.14	.89	[-0.18, 0.16]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.02$, $p = .89$						
Step 2	Constant	-3.55	2.40		-1.48	.15	[-8.50, 1.40]
	Anxiety	-0.13	0.11	-.32	-1.24	.23	[-0.36, 0.09]
	Schizotypy	0.06	0.04	.43	1.49	.10	[-0.01, 0.13]
	$\overline{R}^2$ = .03; $F(2, 25) = 1.43$, $p = .26$				$\Delta\overline{R}^2 = .10$, $\Delta F(1, 25) = 2.84$, $p = .10$		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.68	1.25		0.54	.59	[-1.89, 3.25]
	Stress	-0.05	0.07	-.12	-0.61	.55	[-0.20, 0.11]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.38$, $p = .55$						
Step 2	Constant	-3.45	2.17		-1.59	.12	[-7.91, 1.01]
	Stress	-0.19	0.09	-.50	-2.02	.05	[-0.38, 0.00]
	Schizotypy	0.08	0.03	.56	2.26	.03	[0.01, 0.15]
	$\overline{R}^2$ = .18; $F(2, 25) = 2.76$, $p = .08$				$\Delta\overline{R}^2 = .17$, $\Delta F(1, 25) = 5.09$, $p = .03$		

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Schizotypy = degree of schizotypy, assessed by the Schizotypal Personality Questionnaire (excluding the factor for Social Anxiety).

Table I5

Hierarchical Regression Analyses Predicting N1 Amplitude Difference From Experience of Auditory Hallucination, Controlling Separately for Depression, Anxiety, and Stress, at Electrode FZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.88	0.47		-1.87	.07	[-1.84, 0.09]
	Depression	0.08	0.04	.39	2.15	.04	[0.00, 0.15]
	$\overline{R}^2$ = .12, $F(1, 26) = 4.06$, $p = .04$						
Step 2	Constant	-0.67	1.01		-0.67	.51	[-2.75, 1.41]
	Depression	0.08	0.05	.42	1.79	.09	[-0.01, 0.18]
	Hallucination	-0.01	0.04	-.05	-0.23	.82	[-0.09, 0.07]
	$\overline{R}^2$ = .08; $F(2, 25) = 2.25$, $p = .13$				$\Delta\overline{R}^2 = 0$, $\Delta F(1, 25) = 0.05$, $p = .82$		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.58	0.46		-1.28	.21	[-1.52, 0.35]
	Anxiety	0.05	0.04	.27	1.43	.16	[-0.02, 0.13]
	$\overline{R}^2$ = .04, $F(1, 26) = 2.06$, $p = .16$						
Step 2	Constant	-0.50	1.26		-0.40	.69	[-3.09, 2.09]
	Anxiety	0.06	0.06	.29	0.90	.38	[-0.07, 0.19]
	Hallucination	-0.03	0.06	-.02	-0.07	.94	[-0.12, 0.11]
	$\overline{R}^2$ = .0*; $F(2, 25) = 0.99$, $p = .39$				$\Delta\overline{R}^2 = 0$, $\Delta F(1, 25) = 0.01$, $p = .94$		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.44	0.59		-0.76	.46	[-1.65, 0.76]
	Stress	0.02	0.04	.13	0.66	.52	[-0.05, 0.09]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.43$, $p = .52$						
Step 2	Constant	-1.30	1.13		-1.15	.26	[-3.62, 1.02]
	Stress	-0.02	0.06	-.09	0.29	.77	[-0.13, 0.10]
	Hallucination	0.05	0.05	.28	0.89	.38	[-0.06, 0.16]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.61$, $p = .55$				$\Delta\overline{R}^2 = .03$, $\Delta F(1, 25) = 0.80$, $p = .38$		

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).

Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Hallucination = lifetime extent of auditory hallucination experience, assessed by Factor 1 of the Auditory Hallucination-like Experience Scale.

Table I6

Hierarchical Regression Analyses Predicting N1 Amplitude Difference From Experience of Auditory Hallucination, Controlling Separately for Depression, Anxiety, and Stress, at Electrode CZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.40	0.43		-0.93	.36	[-1.28, 0.48]
	Depression	0.85	0.03	.46	2.67	.01	[0.02, 0.15]
	$\overline{R}^2$ = .19, $F(1, 26) = 7.14$, $p = .01$						
Step 2	Constant	0.70	0.89		0.78	.44	[-1.14, 2.53]
	Depression	0.12	0.04	.65	3.00	.01	[0.04, 0.20]
	Hallucination	-0.05	0.04	-.30	-1.40	.18	[-0.12, 0.02]
	$\overline{R}^2$ = .21; $F(2, 25) = 4.68$, $p = .02$				$\Delta\overline{R}^2$ = .06, $\Delta F(1, 25) = 1.95$, $p = .18$		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.13	0.44		0.28	.78	[-0.78, 1.03]
	Anxiety	0.04	0.04	.21	1.07	.29	[-0.04, 0.11]
	$\overline{R}^2$ = .01, $F(1, 26) = 1.15$, $p = .60$						
Step 2	Constant	0.72	1.21		0.60	.56	[-1.77, 3.22]
	Anxiety	0.07	0.06	.34	1.06	.30	[-0.06, 0.19]
	Hallucination	-0.03	0.05	-.17	-0.53	.60	[-0.14, 0.08]
	$\overline{R}^2$ = .0*; $F(2, 25) = 0.70$, $p = .51$				$\Delta\overline{R}^2$ = .01, $\Delta F(1, 25) = 0.28$, $p = .60$		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.18	0.56		0.32	.75	[-0.97, 1.33]
	Stress	0.02	0.03	.12	0.60	.55	[-0.05, 0.09]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.36$, $p = .55$						
Step 2	Constant	0.10	1.09		0.09	.09	[-2.15, 2.35]
	Stress	0.02	0.05	.10	0.30	.77	[-0.10, 0.13]
	Hallucination	0.01	0.05	.03	0.09	.93	[-0.10, 0.11]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.18$, $p = .84$				$\Delta\overline{R}^2$ = 0, $\Delta F(1, 25) = 0.01$, $p = .93$		

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).

Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Hallucination = lifetime extent of auditory hallucination experience, assessed by Factor 1 of the Auditory Hallucination-like Experience Scale.

Table I7

Hierarchical Regression Analyses Predicting P2 Amplitude Difference From Experience of Auditory Hallucination, Controlling Separately for Depression, Anxiety, and Stress, at Electrode FZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.64	0.58		1.11	.28	[-0.55, 1.84]
	Depression	0.00	0.04	-.01	-0.06	.96	[-0.09, 0.09]
	$\overline{R}^2$ = 0*, $F(1, 26) < 0.01$, $p = .96$						
Step 2	Constant	-1.36	2.14		-0.63	.53	[-5.77, 3.06]
	Depression	0.00	0.10	.01	0.03	.98	[-0.20, 0.20]
	Hallucination	0.06	0.09	.18	0.73	.47	[-0.11, 0.24]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.76$, $p = .48$			$\Delta\overline{R}^2 = .06$, $\Delta F(1, 25) = 1.51$, $p = .23$			

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.91	0.54		1.69	.10	[-0.20, 2.00]
	Anxiety	-0.03	0.04	-.15	-0.77	.45	[-0.12, 0.06]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.59$, $p = .45$						
Step 2	Constant	-2.55	1.28		-2.00	.06	[-5.18, 0.73]
	Anxiety	-0.18	0.06	-.81	-2.86	<.01	[-0.32, 0.05]
	Hallucination	0.16	0.06	.83	2.92	.05	[0.05, 0.28]
	$\overline{R}^2$ = .21; $F(2, 25) = 4.64$, $p = .02$			$\Delta\overline{R}^2 = .25$, $\Delta F(1, 25) = 8.51$, $p = <.01$			

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	1.13	0.66		1.70	.10	[-0.23, 2.50]
	Stress	-0.04	0.04	-.18	-0.94	.35	[-0.12, 0.04]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.89$, $p = .35$						
Step 2	Constant	-1.79	1.10		-1.62	.12	[-4.06, 0.48]
	Stress	-0.17	0.06	-.84	-3.11	<.01	[-0.28, -0.06]
	Hallucination	0.16	0.05	.83	3.10	<.01	[0.06, 0.27]
	$\overline{R}^2$ = .25; $F(2, 25) = 5.41$, $p = .01$			$\Delta\overline{R}^2 = .27$, $\Delta F(1, 25) = 9.64$, $p = <.01$			

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Hallucination = lifetime extent of auditory hallucination experience, assessed by Factor 1 of the Auditory Hallucination-like Experience Scale.

Table I8

Hierarchical Regression Analyses Predicting P2 Amplitude Difference From Experience of Auditory Hallucination, Controlling Separately for Depression, Anxiety, and Stress, at Electrode CZ

Controlling for Depression

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	-0.60	1.07		0.56	.58	[-2.80, 1.60]
	Depression	0.07	0.08	.16	0.82	.42	[-0.10, 0.23]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.68$, $p = .42$						
Step 2	Constant	-1.81	2.30		-0.79	.44	[-7.26, 2.72]
	Depression	0.03	0.10	.07	0.27	.79	[-0.22, 0.23]
	Hallucination	0.06	0.09	.15	0.60	.56	[-0.05, 0.10]
	$\overline{R}^2$ = 0*; $F(2, 25) = 0.51$, $p = .61$				$\Delta\overline{R}^2 = .01$, $\Delta F(1, 25) = 0.35$, $p = .47$		

Controlling for Anxiety

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.15	1.01		0.15	.88	[-1.92, 2.22]
	Anxiety	-0.01	0.08	-.03	-0.14	.89	[-0.18, 0.16]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.02$, $p = .89$						
Step 2	Constant	-4.41	2.61		-1.69	.10	[-8.50, 1.40]
	Anxiety	-0.21	0.13	-.50	-1.59	.12	[-0.36, 0.09]
	Hallucination	0.22	0.11	.59	1.88	.07	[-0.01, 0.13]
	$\overline{R}^2$ = .06; $F(2, 25) = 1.78$, $p = .19$				$\Delta\overline{R}^2 = .12$, $\Delta F(1, 25) = 3.55$, $p = .07$		

Controlling for Stress

Model	Predictor	*B*	SE	β	*t*	*p*	95% CI for *B*
Step 1	Constant	0.68	1.25		0.54	.59	[-1.89, 3.25]
	Stress	-0.05	0.07	-.12	-0.61	.55	[-0.20, 0.11]
	$\overline{R}^2$ = 0*, $F(1, 26) = 0.38$, $p = .55$						
Step 2	Constant	-4.11	2.17		-1.89	.07	[-8.58, 0.36]
	Stress	-0.26	0.11	-.69	-2.45	.02	[-0.49, -0.04]
	Hallucination	0.27	0.10	.73	2.59	.02	[0.06, 0.48]
	$\overline{R}^2$ = .16; $F(2, 25) = 3.57$, $p = .04$				$\Delta\overline{R}^2 = .21$, $\Delta F(1, 25) = 6.69$, $p = .02$		

Note. $n = 28$ in all analyses. CI = confidence interval. $\overline{R}^2$ = Adjusted R^2. *$\overline{R}^2$ = 0 if the value is spuriously negative (Tabachnick & Fidell, 2013).
Depression, Anxiety, and Stress = level of depression, anxiety, or stress during the week prior to testing, assessed by the Depression Anxiety Stress Scales short version. Hallucination = lifetime extent of auditory hallucination experience, assessed by Factor 1 of the Auditory Hallucination-like Experience Scale.

Appendix I

CD inside pocket attached to back cover

Raw data spreadsheets

- Clinical data.
- N1 amplitude data.
- P2 amplitude data.

SPSS analysis for electrode sites FZ, FCZ, and CZ

- Correlation analysis for main study variables.
- N1 and P2 ANOVAs.
- N1 amplitude difference (LS minus LO) regression analyses.
- P2 amplitude difference (LS minus LO) regression analyses.