

# The role of prediction and attention in phantom voice perception

Citation for published version (APA):

Duggirala, S. X. (2024). *The role of prediction and attention in phantom voice perception*. [Doctoral Thesis, Maastricht University]. Maastricht University. <https://doi.org/10.26481/dis.20240423sd>

## Document status and date:

Published: 01/01/2024

## DOI:

[10.26481/dis.20240423sd](https://doi.org/10.26481/dis.20240423sd)

## Document Version:

Publisher's PDF, also known as Version of record

## Please check the document version of this publication:

- A submitted manuscript is the version of the article upon submission and before peer-review. There can be important differences between the submitted version and the official published version of record. People interested in the research are advised to contact the author for the final version of the publication, or visit the DOI to the publisher's website.
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# THE ROLE OF PREDICTION AND ATTENTION IN PHANTOM VOICE PERCEPTION

Suvarnalata Xanthate Duggirala



# **The role of prediction and attention in phantom voice perception**

**Suvarnalata Xanthate Duggirala**

ISBN: 978-94-6483-961-6

Cover illustration: Dr. Sumiti Saharan

Printing: Ridderprint | [www.ridderprint.nl](http://www.ridderprint.nl)

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# The role of prediction and attention in phantom voice perception

DISSERTATION

to obtain the degree of Doctor at Maastricht University,  
on the authority of the Rector Magnificus, Prof. dr. Pamela Habibović  
in accordance with the decision of the Board of Deans,  
to be defended in public on  
Tuesday 23rd April 2024 at 16:00 hours

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to my parents,  
whom I owe everything





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# **Chapter 01**

## **General Introduction**

*“If hallucination is kind of an uncontrolled perception, then a perception is a controlled hallucination. [...] We have hallucinations all the time, except when we agree on them, then we call it reality (Seth, 2017)”*

- Anil Seth,

Professor in Cognitive and Computational Neuroscience

University of Sussex, United Kingdom



Perception is a dynamic process that extends beyond the notion of ‘seeing is believing’. It involves intricate and active mechanisms that rely on prior knowledge, current contexts, and it is shaped by the limitations of our sensory systems and cognitive capacities. Consequently, it yields a reconstructed representation of reality, akin to assembling pieces of a puzzle to understand the environment. This is illustrated by the fact that we can at first glance make sense of the following letter string with omissions.

Y\_U C\_N R\_AD TH\_S E\_S\_LY D\_SP\_TE M\_SS\_NG L\_TT\_RS.

In the auditory domain, consider a situation where we are listening to a friend speaking on the phone, and due to a momentary loss in signal, parts of the conversation are unintelligible or missing. Despite fragmented and noisy/missing sensory input, we usually seamlessly integrate such input, supported by previous knowledge and showing our ability to construct a coherent perception. However, this raises the question of how much ‘normal’ perceptual experiences overlap in this regard with ‘phantom’ perceptions such as hallucinations where something absent is perceived. Both types of perceptual experiences rely to a certain extent on predictions. We use past experiences to make predictions about what we should expect to perceive. These expectations are then compared with the incoming sensory input to generate a subjective perception of reality. We rely on predictions to make sense of noisy and at times ambiguous sensory input to streamline perception and make it efficient. One perspective is that the parallel between hallucinations and subjective perception likely lies in the extent of reliance on these predictions. Hallucinations might occur when we excessively trust our predictions, disregarding contrary sensory information (Corlett et al., 2019; Friston, 2005b; Powers, Kelley, & Corlett, 2016). For instance, if we strongly predict encountering a wild boar (whether seeing or hearing its growl) during a hike in the forest when it is dark, we may perceive one even if absent (no corresponding input). Here, prior knowledge about wild boars being typically found in forests and foraging during darkness influence perception, overriding any contradictory sensory information one might receive. However, if a companion also perceives a wild boar, this collective agreement might become reality (yet, there are exceptions, like collective hallucinations (Clarke, 2002)). This observation emphasizes that (i) ultimately, we actively participate as co-creators of perceptual experiences,

whether they manifest as controlled hallucinations or mutually agreed-upon realities (Seth, 2017), and (ii) there is likely a continuum between subjective perception and phantom perceptions such as hallucinations, both relying on predictions but differing in the degree of reliance. The workings of this hypothetical continuum, spanning from normal to phantom perceptions such as hallucinations, remain unknown, necessitating further investigation into the underlying mechanisms guiding the brain's reliance on predictions and prior experiences. Therefore, this dissertation centers on understanding the underlying mechanisms of phantom voice perceptions – auditory verbal hallucinations and voice hearing (the terms voice hearing and auditory verbal hallucinations will be used interchangeably throughout this dissertation; Aleman & Larøi, 2008; Baumeister, Sedgwick, Howes, & Peters, 2017; Larøi et al., 2012; Toh, Moseley, & Fernyhough, 2022). Specifically, it focusses on potential associations of change in predictive processing, control of attention allocation, and proneness to experiencing auditory verbal hallucinations.

This first chapter introduces how prediction and attention together contribute to subjective and phantom perceptions in audition. Thereafter, different theories are presented that are relevant within the context of the current dissertation and explain the phantom voice perception – auditory verbal hallucinations. This discussion of theoretical accounts is followed by a brief introduction into different neural indices that are used to explore the roles of prediction and attention in phantom voice perception in the subsequent chapters. Lastly, a roadmap of the chapters is provided.

## **1. Prediction and attention in perception**

Prediction refers to a cognitive process, which the brain relies on to generate expectations about upcoming sensory input based on prior knowledge and experience, and internal models or representations (Schroger, Kotz, & SanMiguel, 2015; Schröger, Marzecová, & SanMiguel, 2015). This allows it to efficiently process and interpret the environment and adjust behavior. Attention, on the other hand, refers to the mechanism of monitoring, selecting, and controlling relevant sensory input in perception to, and concurrently disregarding irrelevant sensory input (Näätänen, Alho, & Schröger, 2002; Schroger et al., 2015; Schröger et al., 2015). Attention can be

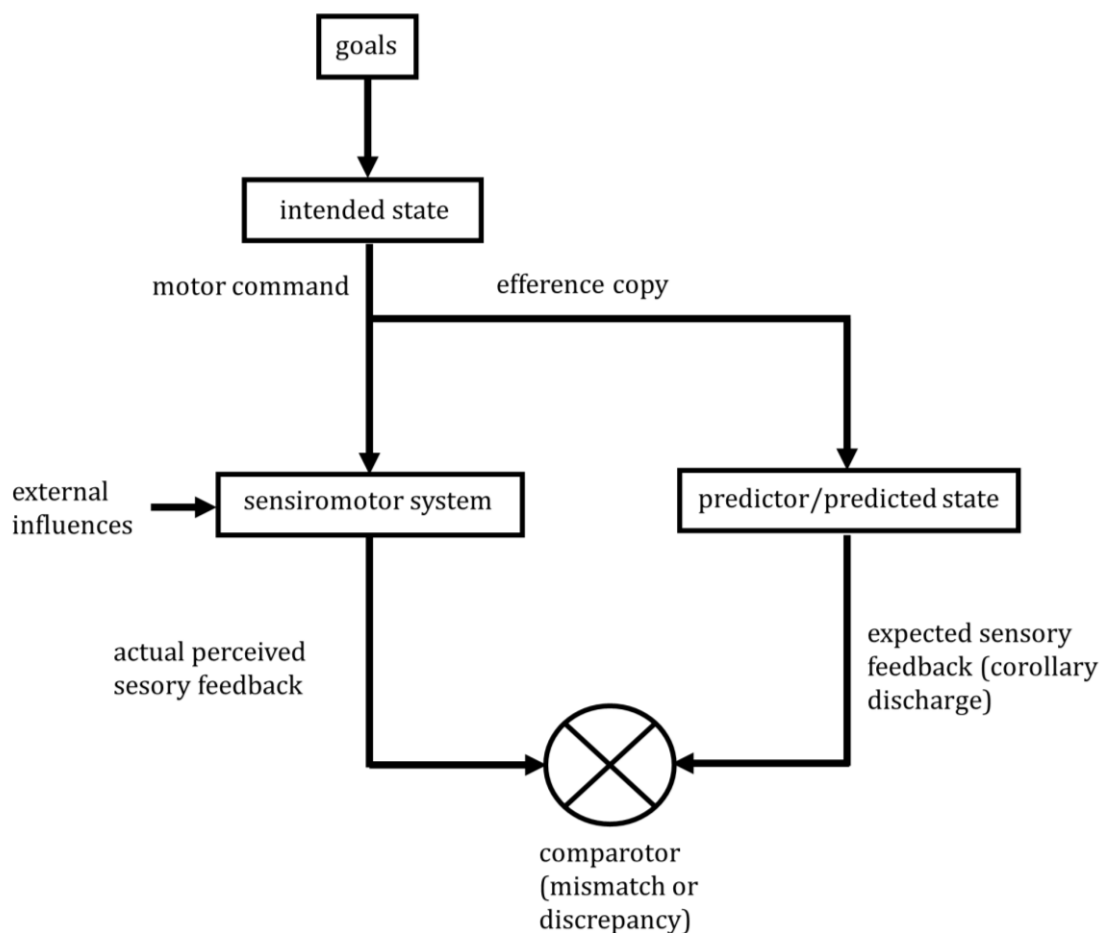
controlled voluntarily (e.g., focusing on a lecture while ignoring noise from fellow students) or captured involuntarily (e.g., during an intense conversation with a friend, your attention slips to hear the sound of a birdsong) in certain contexts.

Prediction has been classically studied in oddball paradigms, where unexpected 'deviant' stimuli are interspersed within a sequence of expected 'standard' stimuli (Kujala, Tervaniemi, & Schröger, 2007; Näätänen, Kujala, & Winkler, 2011). Sensory responses to unexpected stimuli reflect a regularity violation evoking a mismatched response (e.g., mismatch negativity (Kujala et al., 2007; Näätänen et al., 2011; Winkler, 2007) or a P3a of the auditory evoked potential associated with involuntary attention (Escera, Alho, Schroger, & Winkler, 2000; Friedman, Cycowicz, & Gaeta, 2001)), which can be interpreted as an error in predictive processes. These unexpected stimuli not only interrupt the operations of sensory processing and prediction but also invoke involuntary attentional orienting towards unexpected stimuli, exemplifying the possible synergistic relationship of attention and predictive processes (Parmentier, Elsley, Andrés, & Barceló, 2011; Schroger et al., 2015; Schröger et al., 2015; Wetzel & Schröger, 2014).

The interrelatedness of prediction and attention has been studied by focusing on the processing of non-violating, expected stimuli that match the predictions of internal predictive models using visual cues to indicate an upcoming sound or generating a sound through a motor act, i.e., button press (see review Hughes, Desantis, & Waszak, 2013; Schafer & Marcus, 1973). This approach is central to these studies, and I will focus on exploring the predictive processing and attentional control mechanisms. These studies used paradigms and tasks (e.g., motor-auditory task) (Schafer & Marcus, 1973) based on the internal forward model framework (figure 1) of sensorimotor control (Blakemore, Wolpert, & Frith, 2000; Wolpert, Ghahramani, & Jordan, 1995). This framework postulates that when an action is performed, a motor command of the movement is sent to the motor system. A copy of this motor command, referred to as 'efference copy', is sent to the sensory cortex, which generates a 'corollary discharge' – the expected sensory consequence or the outcome of the action (von Holst & Mittelstaedt, 1971; Wolpert et al., 1995). The expected sensation is measured against the actual sensation. The mismatch or discrepancy between the two sensations is used

to update predictions and refine the forward model. When the mismatch is minimal, signifying a closer match between the expected and the actual perceived sensation, perception is likely unaffected and cortical activity is suppressed (also electrophysiologically evidenced as suppressed N100) relative to when the mismatch is large (Blakemore, Rees, & Frith, 1998; Shergill et al., 2013). Under these circumstances of minimal mismatch, an action is perceived as internally generated and less salient as the sensory consequences match the expectation and consequently attract less attentional resources.

**Figure 1:** The forward model (adapted from Blakemore et al., 2000).



Another perspective in prediction research – the predictive coding framework (PC), derives from the concept of active inference as an explanation for perception (Lee & Mumford, 2003; Rao & Ballard, 1999). According to this perspective, perception emerges from an interplay of sensory input and internal models or representations. Of



note is that, unlike the internal forward model, there are multiple internal models at each level of the hierarchy in PC. These models are guided by likelihood and prior probabilities, shaped by past experiences, influencing the interpretation of sensory input. These models also infer potential causes of the sensory input and generate predictions, which when compared to actual sensory input, can lead to prediction errors in case of a mismatch. These errors drive model refinement through a hierarchical Bayesian process, integrating bottom-up input and top-down predictions via recurrent loops within a cortical hierarchy (Lee & Mumford, 2003; Rao & Ballard, 1999). Attention also plays a crucial role in assessing the precision and reliability of the prediction error and represents the degree of confidence assigned to the sensory input, thereby also modulating the responses to attended sensory input (Feldman & Friston, 2010; Hohwy, 2013). For example, when engaging in a sport like table tennis, the brain predicts the opponent's moves based on cues – the opponent's body positioning, racket angle, and previous patterns of play. Based on these predictions, one expects the ball's trajectory and plans return shots accordingly. If the actual outcome matches with one's own predictions, one executes a planned shot. However, if there is a mismatch between the expected and actual serve, the brain detects a mismatch – prediction error, prompting a quick adjustment of internal predictive models, quickly adapting self-actions to return the ball more effectively during the next serve. Attention is used to adjust the strength or precision of a prediction error, thereby playing a role in updating predictions (Feldman & Friston, 2010; Hohwy, 2013). Attentional resources may likely be required to minimize the prediction error (Feldman & Friston, 2010; Hohwy, 2013). In sum, cognitive constructs of prediction and attention likely work synergistically in these contexts, enabling adaptive behavior and facilitating efficient perception.

Alterations associated with predictive processing, such as the inability to predict the consequences of a self-generated action, and the control of attention allocation, like the inability to inhibit attention to an irrelevant stimulus, have been implicated in voice hearing (Ford, Gray, Faustman, Roach, & Mathalon, 2007; Heinks-Maldonado et al., 2007; Hugdahl et al., 2008). However, it is still not entirely clear as to how alterations in the interplay between prediction and attention would contribute to phantom auditory perception such as hearing voices in the absence of an external

source/stimulation. To address this question, it is imperative to understand the interplay between prediction and attention in (self-) voice production and perception. Even though one's own voice is the voice we encounter most often in daily communication, it typically receives minimal attention, and we are less aware of it while we are speaking. On the other hand, we become more conscious of any unexpected changes in voice quality, for example, when we have a cold. Under such circumstances, we are not only more aware of our voice, but it also garners a higher level of attention. The framework of the internal forward model offers a plausible explanation regarding these differences (Feinberg, 1978; Ford, Mathalon, et al., 2001b; Ford, Roach, & Mathalon, 2010; Pinheiro, Schwartz, & Kotz, 2018; Wolpert et al., 1995). Before speaking, a copy of the motor command also known as the efference copy, is transmitted to the forward model to anticipate the expected sensory outcome (self-generated own voice) of the action (speaking). When predictions match with actual sensations, activation in the auditory cortex reduces, distinguishing internally-generated sensations from external ones. In the event of a mismatch, where expected and actually perceived sensations diverge, the auditory cortex activity increases, leading to an enhanced attention allocation. There is compelling evidence from neuroimaging (Ford, Mathalon, Whitfield, Faustman, & Roth, 2002) and neurophysiological (Ford & Mathalon, 2004; Ford, Mathalon, et al., 2001a, 2001b; Ford et al., 2013) research that auditory verbal hallucinations result from the inability to differentiate sensations produced by the self and those arising from the external environment due to alterations in the internal forward model. However, several open questions remain: Are alterations of prediction and attention reciprocal in voice hearing? How are these processes modulated by proneness to hallucinate? Are these alterations also present in non-clinical individuals who are highly prone to hallucinate? This dissertation aims to understand and address these questions.

## **2. Phantom voice perceptions - Auditory verbal hallucinations**

Auditory hallucinations are primarily experienced as voices (Larøi, 2012). These voices may involve verbal speech, closely resembling the act of hearing other people speak, or they can display distinctions from real voices. The experience of hearing voices in the absence of an external source is referred to as auditory verbal

hallucinations (AVH). AVH are the most commonly reported type of hallucinations in individuals with a psychiatric diagnosis such as schizophrenia, bipolar disorder, and post-traumatic stress disorder (American Psychiatric Association & Association, 1994). These experiences are also reported in individuals from the general population (Daalman, Boks, et al., 2011; Johns, Hemsley, & Kuipers, 2002; Johns et al., 2014; Larøi, 2012; Larøi & Van Der Linden, 2005b). To understand the underlying mechanisms of voice hearing, researchers have explored this experience at cognitive, neurological, sociological as well as phenomenological levels. Here I briefly introduce the theories relevant in the context of the current dissertation.

### **a. Theories**

Several theoretical frameworks have been proposed to understand the experience of AVH (Braver, Barch, & Cohen, 1999; Ditman & Kuperberg, 2005; Kapur, 2003; Lesh, Niendam, Minzenberg, & Carter, 2011). This section provides a succinct overview of theories that hold significance for the present dissertation.

#### *Self-monitoring and inner speech*

The self-monitoring and inner speech theory, originally proposed by Frith and colleagues (Frith, Friston, Liddle, & Frackowiak, 1992; Frith, Blakemore, & Wolpert, 2000; Frith & Done, 1988), offers an explanatory framework for understanding AVH as a consequence of the distorted sense of agency or altered source monitoring. Self-monitoring can be understood as the ability to identify and distinguish self-generated sensations from those that arise from external sources. Based on the forward model framework (Blakemore et al., 2000; Wolpert et al., 1995), this theory suggests that deficits in self-monitoring, leading to an inability to differentiate self-generated and externally-generated actions, can account for positive symptoms in psychosis (see section 1 for the details on the forward model).

Specifically, when there is a mismatch between the expected and actual perceived sensations, for example, due to a malfunctioning prediction mechanism, the cancellation of expected and actual sensory feedback signals does not occur. This, in turn, might lead to the feeling that voluntary actions are 'externally controlled' or

‘unintentional’ in patients with a psychotic disorder (Blakemore & Frith, 2003; Frith et al., 2000; Haggard & Eimer, 1999; Libet, Wright Jr, & Gleason, 1983; Seal, Aleman, & McGuire, 2004; Spence et al., 1997). As the generation of speech is a form of motor action, monitoring of speech generation might be linked to the conscious awareness of facilitating and differentiating between self-generated and externally-generated input (Feinberg, 1978). Any alterations within this system would lead to a diminished ability to recognize self-generated content. Therefore, inner speech would not be recognized as originating from oneself and would be mistakenly attributed to an external source. Empirical studies have supported this theory by reporting abnormal workings of internal models during the self-generation of speech in people experiencing psychosis (Ford & Mathalon, 2004; Ford, Mathalon, et al., 2001a, 2001b; Ford et al., 2013; Ford, Roach, Faustman, & Mathalon, 2007).

#### *Aberrant salience hypothesis*

Early electrophysiological animal research showed that dopaminergic activity in the mesolimbic pathway increased momentarily when encountering unexpected rewards or cues to reward, but decreased when reward was omitted (Berridge, 2019; Bromberg-Martin, Matsumoto, & Hikosaka, 2010; Howes & Nour, 2016). This neural activity reflects incentive salience stemming from motivated action selection. However, midbrain dopamine neurons are not uniform and exhibit functional heterogeneity. On the one hand, some neurons encode the motivational value of positive outcomes like food, which drives seeking behavior and value learning. On the other hand, other neurons respond to salient non-rewarding aversive stimuli, triggering orienting and exploration behavior.

The aberrant salience hypothesis in schizophrenia postulates that alterations in dopamine release lead to the over-attribution of meaning and motivational value to irrelevant environmental events (Fletcher & Frith, 2009; Howes & Nour, 2016; Kapur, 2003, 2005). This misattribution of salience may involve both rewarding as well as aversive signaling, contributing to positive symptoms such as delusions and hallucinations in psychosis. Cognitive theories of psychosis provide a link between socio-developmental risk factors (e.g., childhood trauma, social adversity),



neurobiological substrate (e.g., sensitive dopaminergic system), and subjective experiences (e.g., pessimistic - expecting and perceiving negativity and 'externalizing' thinking style) of psychosis (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001a). This implies that salience can be assigned to actions generated by oneself, resulting in the perception of these experiences as originating externally and giving rise to misconceptions and phantom perceptions - AVH. Similarly, computational accounts of brain function have linked salience misattribution by integrating sensory input with internal models of the world, highlighting the role of cortical-subcortical interactions modulated by subcortical dopamine transmission (Fletcher & Frith, 2009). In this regard, sensory input becomes salient when it contradicts the brain's predicted representation of the world, which is encoded in sensory brain regions. Continuous discrepancies between expected and actual perceived sensory input prompt the brain to adapt its internal representation of the world. Subtle disturbances in dopamine signaling have also contributed to radical maladaptive changes in the internal representation of the world, resulting in experiencing AVH (Fletcher & Frith, 2009). Neuroimaging studies have shown reduced activity in the mesolimbic pathway for reward-predicting stimuli whereas increased activity was observed for neutral stimuli in unmedicated and first episode psychosis patients compared to healthy controls (Howes & Nour, 2016; Murray et al., 2008). However, methodological challenges exist in directly demonstrating aberrant dopaminergic activity in humans. The full spectrum of symptoms in psychosis and the specificity of aberrant salience processing to schizophrenia remain unclear.

#### *Linking alterations of self-monitoring to salience misattribution*

These two accounts of self/source-monitoring and salience misattribution may not be independent of each other and can be linked with alterations in the predictive processing. Both theories postulate that flawed predictive processing can lead to a discrepancy between top-down predictions and bottom-up actual sensory input (Davies, Teufel, & Fletcher, 2018). This, in turn, may lead to the inability to recognize self-generated actions or attribution of self-generated actions to an external source. Similarly, altered expectation based on top-down predictions may result in attributing meaning to an irrelevant incoming stimulus, leading to dysfunctional salience

attribution (Galdos et al., 2011). This account is supported by empirical studies wherein aberrant salience is manifested as attributing negative emotional qualities to a neutral stimuli (Allott et al., 2015), recognizing meaningful speech in noise, or the inability to inhibit attention to an irrelevant stimuli (Alba-Ferrara, de Erausquin, Hirnstein, Weis, & Hausmann, 2013).

## **b. The continuum hypothesis**

While it may seem logical to view health and illness as separate and exclusive, it is essential to acknowledge that not all illnesses neatly fit into such a binary classification. In the context of psychiatric disorders, it is critical to understand that the underlying causes are often multifaceted and complex, stemming from a combination of environmental, genetic, and cognitive factors that interact and result in a biological alteration (David, 2010; Johns et al., 2002; Laroi et al., 2012; Lawrie, Hall, McIntosh, Owens, & Johnstone, 2010; Myin-Germeys et al., 2003; van Os, Hanssen, Bak, Bijl, & Vollebergh, 2003; van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009; Verdoux, Liraud, Assens, Abalan, & van Os, 2002; Verdoux & van Os, 2002). The traditional categorical approach to understanding psychosis fails to distinguish between the mere presence of symptoms and the existence of a clinically diagnosable disorder. In contrast, a more nuanced continuum model posits that observable traits associated with psychosis manifest themselves well before developing into clinically significant impairment or disorder (Krabbendam, Myin-Germeys, Hanssen, & van Os, 2005; Lawrie et al., 2010; van Os et al., 2003; van Os et al., 2009; Verdoux & van Os, 2002). These traits, often referred to as ‘psychotic-like experiences’, exist on a continuum with other subjective experiences, potentially progressing from a state of wellness to illness, whereby the expression of these traits tend to increase. Evidence supporting this continuum has been studied and reviewed in several publications in the past decade (Baumeister et al., 2017; Binbay et al., 2012; Garrison et al., 2017; van Os et al., 2009). Considering the existing evidence supporting a continuum of psychotic experiences among the general population, it is thus important to understand the underlying mechanisms, facilitating the transition from experiencing a symptom to developing clinically diagnosable psychiatric disorders.

### *Similarities and differences*

Psychotic and non-clinical AVH share similarities in perceptual phenomenology in terms of loudness, location – internal or external, number of voices, personification, and identity of the voice – attributing the voices to a person (Daalman, Boks, et al., 2011; Johns et al., 2002; Johns et al., 2014). Similarly, both psychotic and non-clinical voice hearers share underlying brain activity during voice hearing (Diederen et al., 2013; Diederen, Daalman, et al., 2012). Regardless of their clinical diagnosis, individuals experiencing AVH show similar difficulties with attentional control – controlling allocation or inhibition of attention and memory processing (Badcock, Chhabra, Maybery, & Paulik, 2008; Brookwell, Bentall, & Varese, 2013; Chhabra, Badcock, Maybery, & Leung, 2011; McKague, McAnally, Puccio, Bendall, & Jackson, 2012; Waters, Allen, et al., 2012; Waters & Badcock, 2009; Waters, Maybery, Badcock, & Michie, 2004). Differences between non-clinical voice hearers and voice hearers with a psychotic disorder pertain to age of onset – non-clinical voice hearers reportedly showcase an early onset ( $\pm 12$  years of age) as well as frequency and duration with which the voices are experienced (Daalman, Boks, et al., 2011; Johns et al., 2002; Johns et al., 2014). The most significant differentiating factor is the emotional quality of the voices and the associated distress and lack of perceived control (Daalman, Boks, et al., 2011; Hill, Varese, Jackson, & Linden, 2012; Jenner, Rutten, Beuckens, Boonstra, & Sytma, 2008; Johns et al., 2002; Johns et al., 2014; Larøi, 2012). Voice hearers with a psychotic disorder more often hear more negative and derogatory voice content, which causes distress resulting in the need for care due to impaired functioning in everyday life (Beavan & Read, 2010; Krabbendam, Myin-Germeys, Hanssen, de Graaf, et al., 2005). On the other hand, non-clinical voice hearers more often hear neutral or positive voice content, have more insight and control over their voices, and experience less distress as a consequence of voice hearing (Daalman, Boks, et al., 2011; Johns et al., 2002; Larøi, 2012).

Delving into a distinguishing element such as the emotional quality of voices within a common framework such as the neural foundation of voice processing holds the potential to yield valuable insights into grasping the fundamental mechanisms

underlying AVH. Additionally, this approach could shed light on the progression from non-clinical to clinical transitions in voice hearing.

### *Measure of hallucination proneness*

Several assessment methods have been employed to examine the prevalence and phenomenology of hallucinatory experiences in non-clinical individuals from the general population, including self-report questionnaires and structured- or semi-structured interviews (Johns et al., 2002). One of the most frequently used self-report questionnaires is the Launay Slade Hallucination Scale (LSHS) that measures the predisposition to hallucinatory experiences in individuals from the general population (Aleman, Nieuwenstein, Böcker, & De Haan, 2001; Launay & Slade, 1981; Waters, Badcock, & Maybery, 2003). It comprises 16 items, encompassing various domains of hallucinations (tactile, olfactory, visual, hypnagogic, and hypnopompic), with participants providing responses on a five-point Likert scale (Bentall & Slade, 1985). A revised version was validated across different groups of individuals sampled from the general population (Aleman et al., 2001; Castiajo & Pinheiro, 2017; Sahu et al., 2020). Thus, empirical chapters 4-6 use LSHS scores as a measure of hallucination proneness (HP).

Based on the theories of self-monitoring, salience misattribution and the HP continuum, the current dissertation aims to systematically explore how changes in predictive processing and attentional control may be associated with phantom voice perception as a function of HP.

## **3. Neural markers**

A number of neural correlates have been employed to study the physiological/biological basis of the behavioral manifestations linked to a particular condition. This empirical research in this dissertation incorporated the two most commonly used non-invasive (neuro-)imaging methods to study human brain function – functional magnetic resonance imaging (fMRI; chapter 2 and 3) and electroencephalography (EEG; chapter 4-6).

### **a. Functional magnetic resonance imaging**

fMRI is a non-invasive neuroimaging method utilized to measure and map brain activity indirectly by detecting changes in blood oxygenation and flow, thus reflecting local neural activity (Glover, 2011; Poldrack, Mumford, & Nichols, 2011). fMRI capitalizes on the principle that active brain regions experience enhanced blood supply to meet their oxygen and nutrient demands, resulting in a higher concentration of oxygenated hemoglobin compared to deoxygenated hemoglobin. Through the utilization of a powerful magnetic field and radio waves, the MRI scanner captures the discrepancy in hemoglobin levels, referred to as the hemodynamic response function, generating detailed images of the brain's activity using complex signal processing algorithms. This method enables the investigation of the interplay between specific cognitive or behavioral tasks and corresponding neural changes in distinct brain regions (Poldrack et al., 2011).

Using fMRI (chapter 2), I examined whether attention is controlled and directed by positive and negative emotional information in a conflict between task-relevant and irrelevant aspects. Although both positive and negative emotions exert influence over attention allocation, they elicit distinct behavioral reactions, with negative emotions being linked to avoidance tendencies, and positive emotions being associated with approach-oriented behaviors (Fredrickson, 1998, 2001; Fredrickson & Branigan, 2005). For example, one might avoid walking alone in a dark alley at night as it seems threatening/dangerous. However, if the same street is decorated with flowers and lights in the evening, it could prompt an approach behavior. Next to assessing the behavioral response by means of reaction times and accuracy, this chapter also investigated neural activity within the dorsal and ventral parts of anterior cingulate cortex (ACC) as well as at the whole brain level. As changes in the processing of emotions and attentional control as well as their interaction are often reported in both psychotic and non-clinical voice hearers, in chapter 3, fMRI studies examining this interaction were reviewed along the psychosis continuum. Specifically, sensitivity towards negative or positive emotion in tasks exploring emotion-attentional control and its association with positive and negative symptoms were discussed. In addition to examining the neuroimaging evidence associated with emotion-attentional control interactions, contributions of functional changes in subcortical and cortical brain

regions that facilitate emotion-cognitive control coupling (e.g., thalamus, basal ganglia, and angular gyrus) along the psychosis continuum were addressed.

### **b. Electrophysiological signals**

EEG is a neurophysiological technique used to capture spontaneous electrical activity produced by the brain (Elul, 1972; Kirschstein & Köhling, 2009; Teplan, 2002). It involves placing small metal electrodes or sensors on the scalp to detect and amplify the electrical signals generated by the brain's neurons. This signal reflects amalgamation of postsynaptic potentials originating from a parallel-oriented group of neurons firing in synchrony. Modified forms of the EEG method encompass evoked potentials or event-related potentials (ERP), which entail averaging the EEG activity synchronized with the introduction of a specific sensory, motor or cognitive event, and help studying psychophysiological correlates of cerebral processes (Cohen, 2014; Luck, 2014). Scalp-recorded ERP components can be distinguished based on their polarity – positive or negative, amplitude, latency, and topographical distribution (Luck, 2014). Due to its high temporal resolution, this method offers valuable advantages, enabling the examination of temporal dynamics of neural activity in the fields of cognitive science, cognitive psychology, and psychophysiological research. Using EEG (chapter 4-6), self-voice processing was investigated while participants self-generated their own pre-recorded voices via a button-press compared to passively listening to the same voices generated by the computer (motor-auditory paradigm; (Schafer & Marcus, 1973)). This paradigm typically generates a series of ERPs - P50, N100, P200 and N200, reflecting different information processing stages in predictive processing. A brief introduction of each ERP is provided below.

The P50 is an early positive component of the evoked potential recorded at around 50 ms after the presentation of an auditory stimulus. Typically, the P50 response is studied using a paired click paradigm (Shen et al., 2020; Smith, Boutors, & Schwarzopf, 1994). When two clicks are heard within a time interval of 500 ms, the second click is usually filtered out by the individual's perceptual system due to its perceived redundancy. A reduced P50 response for the second click is expected as an indicator of normal filtering process involving selective attention, also termed sensory gating.

Many studies have considered the P50 ERP as a promising candidate endophenotype of schizophrenia (for a review of studies De Wilde, Bour, Dingemans, Koelman, & Linszen, 2007; Patterson, Hetrick, Boutros, Jin, Sandman, Stern, Potkin, & Bunney Jr, 2008). In the motor-auditory paradigm, the P50 response is sensitive to predictability of stimulus occurrence and attention allocation (Pinheiro, Schwartz, Gutierrez, & Kotz, 2019; White & Yee, 2006). In ERP studies, researchers have observed a suppression in amplitude of the N100 component as a result of self-generated sensations compared to those generated externally (Heinks-Maldonado, Nagarajan, & Houde, 2006; Rosburg, Boutros, & Ford, 2008). This attenuation has been documented in studies investigating speech production using talk-listen (Ford et al., 2010; Heinks-Maldonado, Mathalon, Gray, & Ford, 2005) and button press motor-auditory tasks (Knolle, Schroger, Baess, & Kotz, 2012; Knolle, Schroger, & Kotz, 2013a, 2013b; Knolle, Schwartz, Schroger, & Kotz, 2019; Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018). The N100 component, which typically peaks around 100 ms after stimulus onset, is generated in primary and secondary auditory cortices, with potential contributions from frontal brain regions (Godey, Schwartz, De Graaf, Chauvel, & Liegeois-Chauvel, 2001; Näätänen & Michie, 1979; Näätänen & Picton, 1987; Zouridakis, Simos, & Papanicolaou, 1998). The modulation of N100 amplitude reflects changes in the prediction error i.e., the mismatch between the expected and the actual perceived sensory feedback. The P200 component in ERP studies is localized to a number of neural sources, such as the planum temporale and auditory association areas (Crowley & Colrain, 2004; Godey et al., 2001). Historically, the P200, with its peak occurring approximately 200 ms after the stimulus onset, has been associated with attention allocation and categorization processes (Knolle et al., 2012; Knolle et al., 2013a; Knolle et al., 2019). Nevertheless, its specific functional significance remains incompletely elucidated. The N100 suppression for self-generated auditory stimuli is often accompanied by the P200 response suppression. However, the N100 and P200 suppression may indicate different mechanisms in predictive processing (Chen, Chen, Liu, Huang, & Liu, 2012; Knolle et al., 2012; Knolle et al., 2013a; Sowman, Kuusik, & Johnson, 2012). The P200 response is more sensitive to temporal perturbation to predictability (Chen et al., 2012) and may be indicative of the conscious perception of self-generated auditory stimuli (Knolle et al., 2012; Knolle

et al., 2013a). The auditory N200 component, originating from the frontal and medial brain regions including the anterior cingulate cortex (ACC), typically follows a prominent negative peak with a frontocentral topography around 200-400 ms (Knolle et al., 2019). Specifically, the anterior N200 component has received considerable attention in studies related to attentional/cognitive control, encompassing activities such as strategy and error monitoring, novelty detection and attention orienting, feedback processing, and immediate action control, including response inhibition (Folstein & Van Petten, 2008; Knolle et al., 2019).

By employing the motor-auditory task (Bass, Jacobsen, & Schroger, 2008; Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018; Schafer & Marcus, 1973), chapter 4-6), participants varying in HP self-generated and passively listened to their own voices changing from fully neutral to fully emotional (angry/pleasure) - 100% neutral; 60-40% neutral-emotional; 50-50% neutral-emotional; 40-60% neutral-emotional and 100% emotional. This approach was designed to influence the certainty of sensory feedback processing related to one's own voice and changes in attentional control. Within this context, I hypothesized changes in the aforementioned ERPs. Specifically, I expected modulations in the P50 component linked to attention allocation and filtering processes, the N100 component associated with the disparity between expected and perceived sensory feedback (i.e., prediction error), along with attention allocation, the P200 component reflecting conscious differentiation between self- and externally generated stimuli, and the N200 component related to error awareness and attentional control.

#### **4. Thesis overview**

This doctoral dissertation aimed at understanding how predictive and attentional processes interact synergistically and contribute to phantom voice perceptions, i.e., AVH, as a function of HP. Using fMRI, chapter 2 investigated the influence of valence-specific emotions on attentional control by employing an adapted verbal flanker task with neutral, negative, and positive stimuli. In chapter 3, an overview of the neuroimaging evidence supporting a potential interaction of emotion and attentional control along the hypothesized psychosis continuum is provided. Different types of



paradigms, particularly focusing on factors such as emotional valence of the stimulus, symptom severity, illness onset and medication were discussed to closely understand the alterations in this interaction. To further explore the underlying mechanisms of voice hearing, chapter 4, 5, and 6 relied on the principles of internal forward modeling in combination with theories that explain AVH such as self-monitoring and salience misattribution using self-voice production and perception. Auditory stimuli were created by manipulating the levels of negative (chapter 4 and 7) and positive (chapter 5) emotional quality thereby altering the certainty of recognizing one's own voice. Using these stimuli in the auditory-motor paradigm coupled with EEG in two different task-designs, alterations in sensory feedback processing and attentional control to unexpected changes in self-voice quality as a function of HP were examined. These examinations were conducted within a non-clinical sample of individuals from the general population who varied in their HP but did not hear voices (chapter 4 and 5). This sample was extended to clinical voice hearers in chapter 6, aiming to examine these processes across the spectrum of HP. Lastly, in chapter 7, a summary of all chapters is provided, followed by an elaboration on broader implications of the empirical results in the context of sensory feedback processing, attentional control, and (emotional) self-voice and HP.



# **Chapter 02**

## **Emotional salience but not valence impacts anterior cingulate cortex conflict processing**

*Published as:* Duggirala, S. X., Belyk, M., Schwartze, M., Kanske, P., & Kotz, S. A. (2022). Emotional salience but not valence impacts anterior cingulate cortex conflict processing. *Cognitive, Affective, & Behavioral Neuroscience*, 22(6), 1250-1263. <https://doi.org/10.3758/s13415-022-01025-9>

## **Abstract**

Stimuli that evoke emotions are salient, draw attentional resources, and facilitate situationally appropriate behavior in complex or conflicting environments. However, negative and positive emotions may motivate different response strategies. For example, a threatening stimulus might evoke avoidant behavior, whereas a positive stimulus may prompt approaching behavior. Therefore, emotional stimuli might either elicit differential behavioral responses when a conflict arises or simply mark salience. The present study used functional magnetic resonance imaging to investigate valence-specific emotion effects on attentional control in conflict processing by employing an adapted flanker task with neutral, negative, and positive stimuli. Slower responses were observed for incongruent than congruent trials. Neural activity in the dorsal anterior cingulate cortex was associated with conflict processing regardless of emotional stimulus quality. These findings confirm that both negative and positive emotional stimuli mark salience in both low (congruent) and high (incongruent) conflict scenarios. Regardless of the conflict level, emotional stimuli deployed greater attentional resources in goal directed behavior.

**Keywords:** Emotion · Salience · Valence · Conflict processing · fMRI

## **1. Introduction**

Navigating a complex environment requires the selection of appropriate responses while ignoring conflicting information. This implies that efficient attentional control is required to resolve conflict that may arise from opposite action tendencies triggered by both relevant and irrelevant stimuli (Norman & Shallice, 1986; Posner & Fan, 2008). Such situations are modeled in experimental settings using conflict paradigms where irrelevant distractors surround task-relevant targets, thereby creating either matching (congruent) or mismatching (incongruent) action tendencies (Eriksen & Eriksen, 1974; Simon & Rudell, 1967; Stroop, 1992). Prolonged response times (RT), increased error rates, and elevated neural activity in the anterior cingulate cortex (ACC) are typically associated with incongruent relative to congruent trials (Barch et al., 2001; Fan, Flombaum, McCandliss, Thomas, & Posner, 2003; Fan, Hof, Guise, Fossella, & Posner, 2008; Kerns, 2005, 2006; van Veen, Holroyd, Cohen, Stenger, & Carter, 2004). These factors suggest high attentional control in conflict processing. Additional attentional control processes and systems are activated by salient situations often signaled by emotional stimuli (Norman & Shallice, 1986, 2000). For example, a potentially threatening situation, such as encountering a snake, may pose a challenge to well-being and invoke avoidance behavior in the form of freeze, flight, or fight responses. Emotionally evocative stimuli can modulate attentional control in conflict processing by slowing down or speeding up cognitive and behavioral responses. However, negative and positive emotional stimuli motivate different response strategies, because they are associated with avoidance and approach behavior, respectively (Fredrickson, 1998, 2001; Fredrickson & Branigan, 2005). These stimuli therefore may either stimulate different approaches to attentional control in conflict processing or merely act as salience markers by triggering attention. Research has focused more on negative emotions and interpreted them as evolutionary considerations as a threatening stimulus can affect survival and well-being. However, there is little evidence whether valence influences attentional control in a specific way in conflict processing both in behavioral and neural terms.

Task-irrelevant emotional information can either be presented before a conflict trial or concurrent with the task relevant stimulus dimension such that it is present

continuously throughout the conflict trial (for details on emotional manipulation in the cognitive control tasks see review Duggirala, Schwartz, Pinheiro, & Kotz, 2020). Prior evidence on emotional priming reports disrupted continuous maintenance of contextual information and challenges ideas of attentional reorientation. Negative and positive emotional priming have either resulted in slower responses (Blair et al., 2007; Hart, Green, Casp, & Belger, 2010; Melcher, Born, & Gruber, 2011; Padmala, Bauer, & Pessoa, 2011; Straub, Kiesel, & Dignath, 2020) or have shown no emotional interference compared with neutral priming in conflict processing tasks (Cohen & Henik, 2012; Cohen, Henik, & Mor, 2011; Cohen, Henik, & Moyal, 2012). On the other hand, continued passive exposure to emotional information competes with task-relevant processes for attentional resources in conflict processing and ultimately influences task-performance. Studies from this latter category where (task-irrelevant) emotion is part of the stimulus dimension have shown more varied results.

Studies using modified versions of flanker or Simon tasks with stimuli connoting a negative emotion have reported facilitated conflict processing, with faster reaction times for negative incongruent than neutral trials (Kanske & Kotz, 2011c, 2012b; Zinchenko, Kanske, Obermeier, Schroger, & Kotz, 2015). Conversely, several studies using emotional Stroop tasks with negative stimuli described inhibition of conflict processing and correspondingly longer color-naming latencies for negative than neutral trials (Ben-Haim, Mama, Icht, & Algom, 2014; Brennan et al., 2015; Frings, Englert, Wentura, & Bermeitinger, 2010; Frings & Wuhr, 2012; Malhi, Lagopoulos, Sachdev, Ivanovski, & Shnier, 2005; Mitterschiffthaler et al., 2008; Mohanty et al., 2005; Rahm, Liberg, Wiberg-Kristoffersen, Aspelin, & Msghina, 2013; Veroude, Jolles, Croiset, & Krabbendam, 2013; Wingenfeld et al., 2009). Although emotion is task-irrelevant per se, it is a behaviorally relevant stimulus dimension in these tasks. Inconsistent behavioral findings between these tasks might be attributed to context, the degree of interference created by stimuli, and the corresponding strength of the resulting conflict. For example, although in the emotional Stroop task, interference is produced by the emotional meaning of a word, an additional layer of interference is created by the flanker colors in the flanker task. A stimulus connoting a negative emotion may be distracting when conflict is low (e.g., color-word emotional Stroop trial) and results in slower responses (Ben-Haim et al., 2014; Brennan et al., 2015;

Malhi et al., 2005; Mitterschiffthaler et al., 2008; Mohanty et al., 2005; Rahm et al., 2013; Veroude et al., 2013; Wingenfeld et al., 2009). Conversely, the same stimulus in a high-conflict context (e.g., incongruent flanker or Simon trials) may recruit additional resources and activate neural networks to cope with the prospect of increased threat (Holtz, Pane-Farre, Wendt, Lotze, & Hamm, 2012), leading to faster responses.

Positive emotions have a different ethological role. Rather than a narrowed focus on immediate and pressing reactions, they motivate a broadening and expansion of attentional focus when circumstances are favorable (Fredrickson, 1998, 2001; Fredrickson & Branigan, 2005). However, the exact mechanisms underlying the effect of positive stimuli on attentional control in conflict processing remain unclear. Like negative emotions, positive emotions facilitate conflict processing in flanker and Simon tasks and lead to shorter reaction times in incongruent positive than neutral trials (Kanske & Kotz, 2011a, 2011d; Xue et al., 2013). Similarly and consistent with negative emotions, emotional Stroop tasks using positive words yield inhibition of conflict processing and longer reaction times (Dresler, Meriau, Heekeren, & van der Meer, 2009). However, some studies using modified versions of the flanker task with positive verbal or audio-visual stimuli also showed no difference in reaction times compared with negative (Li et al., 2014) or neutral trials (Wu & Zhang, 2019; Zinchenko et al., 2017). Studies using emotional Stroop tasks with positive words likewise reported no difference in reaction times over negative or neutral trials (Malhi et al., 2005; Richards, French, Johnson, Naparstek, & Williams, 1992). The influence of positive emotion on attentional control in conflict processing thus remains unclear.

Although different conflict processing tasks might engage distinct sub-processes of attentional control to regulate emotional interference, they might share a similar neural basis. Increased ACC activation is a typical finding in most conflict paradigms, including Stroop, flanker, and Simon tasks (Fan et al., 2003). However, studies using emotionally evocative stimuli in these tasks further report a bifurcation in the functionality of dorsal and ventral parts of the ACC (Kanske & Kotz, 2011b, 2011c; Kim, Kroger, & Kim, 2011; Milham & Banich, 2005; Weissman, Gopalakrishnan, Hazlett, & Woldorff, 2005). The dorsal portion of the ACC is associated with conflict processing

independent of emotional stimulus quality (Kanske & Kotz, 2011b, 2011c; Xu, Xu, & Yang, 2016). However, the ventral part of the ACC is sensitive to emotional conflict and to the resolution of conflict by emotional distractors (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Kanske & Kotz, 2011b, 2011c). These findings were obtained with negative emotional stimuli. The sensitivity of these regions to positive emotional stimuli in conflict processing is therefore still unknown. Studies that compared positive to negative emotional stimuli within a modified version of emotional Stroop task, did not report any significant brain activity in ACC (Arioli, Basso, Poggi, & Canessa, 2021). A similar picture emerges with regards to neural networks. Meta-analyses of neuroimaging studies as well as individual neuroimaging studies using irrelevant emotional stimuli in conflict tasks report increased activity in a fronto-parietal-temporal network consisting of the ACC, inferior, middle and medial/superior frontal gyrus, the dorsolateral prefrontal cortex, the inferior and superior parietal lobule, the angular and supramarginal gyrus, the orbitofrontal cortex, the insula, the inferior and superior temporal gyrus, the precuneus, the precentral and postcentral gyrus and amygdala (Cromheeke & Mueller, 2014; Malhi et al., 2005; Mohanty et al., 2005; Rahm et al., 2013; Song et al., 2017; Veroude et al., 2013; Wingenfeld et al., 2009). Furthermore, experiments reporting enhanced task-performance (faster RTs/low errors) observed increased activity in the inferior and superior frontal gyrus and the angular gyrus, whereas diminished performance was linked to increased activation in the medial/superior frontal gyrus, the precuneus, the inferior frontal gyrus, the amygdala and the fusiform gyrus (Cromheeke & Mueller, 2014). Most of the studies included in these meta-analyses (Cromheeke & Mueller, 2014; Song et al., 2017) compared negative to neutral stimuli, neglecting positive emotions. There is a clear void in the literature regarding the role of positive emotion on conflict processing.

The current study extends prior work (Kanske & Kotz, 2011c) to test whether (i) negative and positive emotions have similar or opposing effects on task performance in conflict processing, (ii) the dACC and vACC activate differently for negative and positive emotional stimuli in conflict processing, and (iii) negative and positive emotions engage distinct neural systems in conflict processing. To answer these questions, we employed a pre-validated (Kanske & Kotz, 2011c) verbal adaptation of



the Eriksen Flanker task with standardized neutral, negative, and positive German words during functional magnetic resonance imaging (fMRI). We hypothesized that stimuli connoting a negative emotion would activate the neural systems involved in goal-driven processes facilitating conflict processing whereas stimuli with a positive connotation might engage systems involved in reward and memory retrieval leading to distraction and inhibition of conflict processing.

## **2. Methods**

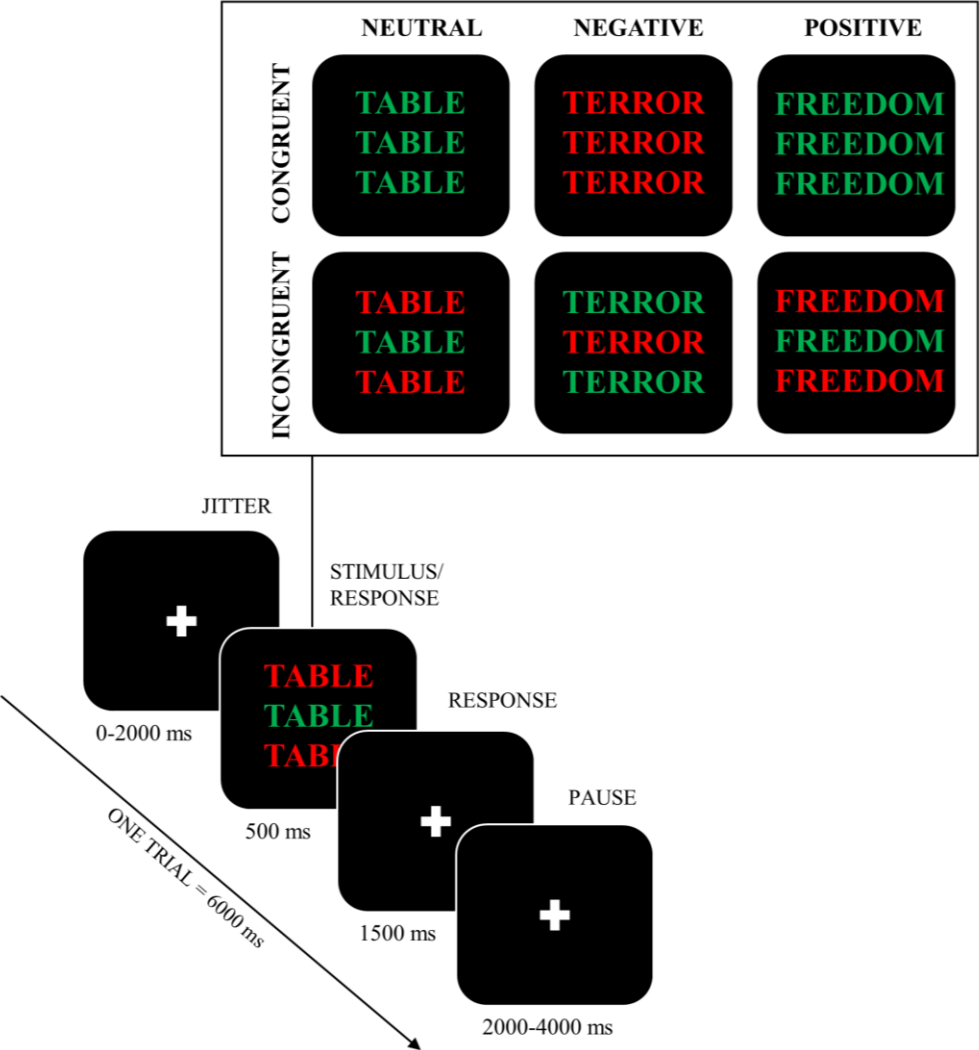
### *Participants*

Twenty-three healthy adults participated in the study. A priori power calculations using G-Power statistical software (Faul, Erdfelder, Lang, & Buchner, 2007) indicated that with  $\alpha = 0.05$  and power (1-error probability) = 0.85 and a medium effect size of 0.25, a sample of a minimum 21 participants would be required for the current task design. This sample size also is supported by previous publications using a similar task design (Kanske & Kotz, 2011b, 2011c, 2012b). Two participants were excluded from further analyses (one due to technical issues during data acquisition and the other for revealing the exclusion criteria [left-handedness] after the experiment), leaving a final sample of 21 right-handed healthy adults (9 females; age range: 19-26 years; mean age = 22.29, SD = 1.95 years). All participants were native German speakers and had normal or corrected-to-normal vision at the time of the experiment. Participants reported to be healthy and had no history of neurological or psychiatric disorders. The study was approved by the ethical review committee psychology and neuroscience, Maastricht, The Netherlands (ERCPN- 176\_01\_02\_2016\_A1). All participants provided their informed consent before the start of the study. They either received financial compensation or study credits for taking part in the study.

### *Experimental paradigm and stimuli*

A modified verbal version of the visual flanker task was employed in the fMRI scanner. Participants were asked to identify the display color of a centrally presented word using their right index finger and right middle finger, while ignoring the color of two flanker words positioned above and below the target word (figure 1) (Kanske & Kotz, 2011c). Flanker and target word colors could be identical or different, creating

congruent (C) and incongruent (IC) trials. Forty pre-standardized German nouns belonging to neutral, negative, and positive emotional categories, respectively, were selected from a corpus that had been validated for emotional valence (negative-neutral-positive), arousal (low-high), and concreteness (concrete-abstract) (Kanske & Kotz, 2010). These word groups significantly differed in valence and arousal (table 1; (Kanske & Kotz, 2010, 2012a)). Accordingly, emotion was task-irrelevant but part of the behaviorally relevant stimulus dimension.



**Figure 1:** Modified version of the flanker task (Kanske & Kotz, 2011c).

**Table 1:** Mean reaction times and percent correct responses.

Category	Congruent		Incongruent		Conflict Effect
	RT (ms)	% Correct responses	RT (ms)	% Correct responses	
Neutral	628.76 (83.71)	96.19 (5.22)	643.26 (88.91)	96.43 (4.22)	14.49
Negative	632.52 (88.29)	96.90 (4.32)	642.44 (88.41)	95.95 (5.15)	9.91
Positive	619.25 (80.70)	96.67 (5.08)	645.02 (84.93)	92.85 (8.74)	25.77

The stimuli were presented in a fully randomized event-related design comprising two identical runs consisting of 80 trials each. These trials were equally distributed among null, neutral, positive, and negative categories. Within each category (i.e., neutral, negative, and positive), there were ten congruent and incongruent trials, respectively (Kanske, Schonfelder, & Wessa, 2013). Each trial lasted for 6 seconds. Within each trial, a stimulus was displayed for 500 ms followed by a response time of 1,500 ms. Stimulus onset within a trial was jittered between 0 to 2,000 ms at 113-, 563-, 1,013-, 1,463-, 1,913-ms intervals to avoid temporal orienting or habituation effects (figure. 1). Each word stimulus was used once, and there were no repetitions during the task. The task therefore comprised six conditions in total (incongruent neutral, congruent neutral, incongruent negative, congruent negative, incongruent positive, congruent positive), each consisting of 20 trials. A fixation cross was displayed during the null trials. Response mapping and the order of the runs were counterbalanced across participants.

### *Procedure*

The study took place in a single session, comprising two parts. In the first part, participants filled in an online questionnaire that recorded demographic information. Participants were then familiarized with the task outside the scanner to avoid training effects inside the scanner. The task was programmed and presented using Presentation software (Neurobehavioral Systems, Inc., Version 18). In the second part, participants underwent anatomical and functional scanning. Stimuli were rear-projected onto a screen with black background (Eiki LCD projector, 60 Hz refresh rate,

1,024 × 768 display resolution), which was visible for the participants through a mirror attached to the head-coil. Participants gave their responses via an MRI-compatible response keypad.

#### *MRI data acquisition*

Participants were scanned at a Siemens 3-T MRI scanner as they lay in headfirst supine position with their head movement restricted by foam cushions. Following a localizer sequence, high resolution anatomical images were acquired via a T1-weighted MPRAGE sequence (TR = 2,250 ms, TE = 2.21 ms, FoV = 256 mm, flip angle = 9°, slice thickness = 1 mm, number of slices = 192, orientation = sagittal, voxel size = 1 mm<sup>3</sup> isotropic). Anatomical scans were followed by a short reversed phased gradient echo-planar imaging (EPI) scan, after which normal phased functional scans were taken during which participants performed the modified version of the Ericksen flanker task. A T2\*-weighted EPI sequence was used to acquire blood oxygen level dependent sensitive functional images (TR = 2,000 ms, TE = 30 ms, flip angle = 70°, number of slices = 60 axial slices, slice thickness = 2 mm, interslice gap = 1 mm, FoV = 208 mm, in-plane resolution = 2 mm × 2 mm, acquisition = interleaved ascending).

#### *Analyses*

##### *Behavioral data*

Performance accuracy and mean RTs were calculated as % hits and mean response times to a stimulus, respectively. Error trials and trials with RT longer than 1500 ms were excluded from further data analysis. Conflict scores were calculated by subtracting mean RT for congruent trials from mean RT for incongruent trials. A 2 × 3 repeated measures ANOVA was performed on mean RT and performance accuracy scores to reveal the main effect of congruency and emotion, and their interaction. Statistical package for social sciences (SPSS, version 18.0, Chicago, IL) was used to analyze the behavioral data.

##### *Neuroimaging data*

The functional images were preprocessed and analyzed using SPM12 (Wellcome Department of Neurology, Institute of Neurology, London, UK), implemented in

MATLAB 2016 (Mathworks Inc., Sherborn, MA). Before preprocessing, distortion correction was performed using the TOPUP algorithm, which estimates image distortions by comparing EPI data collected with normal and reversed phase encoding directions to remove artifacts caused by magnetic susceptibility (Jenkinson, Beckmann, Behrens, Woolrich, & Smith, 2012). Preprocessing steps involved correcting for differences in slice time using the middle slice as the reference. A mean image of all slice time corrected functional scans of each participant was created, to which individual volumes were spatially realigned using rigid body transformation. Head movements in all three dimensions were within the 2-mm threshold. Structural images of each participant were co-registered with their mean functional image and all functional images were normalized to the Montreal Neurological Institute (Montreal, Quebec, Canada) T1 template. Then, the images were spatially smoothed using an 8-mm, full-width at half maximum Gaussian filter. Further statistical analyses were performed on each participant's data using the general linear model (GLM). The design matrix consisted of two sessions corresponding to each run. In each run, 7 regressors corresponding to baseline (null) and active conditions (congruent and incongruent regressors for neutral, positive, and negative emotion, respectively) were defined. For these regressors, the onset of the stimulus represented the event onset. The jittered fixation cross presented before or after were not included in the modeling. This was done because we already have a baseline (null) condition in the design, which was modeled in the GLM. Trials corresponding to wrong response (errors) also were excluded from the modeling to keep the fMRI analysis comparable to the behavioral analysis. In addition, six motion regressors derived from the rigid body realignment were included to model linear residual movement effects.

At the participant level, contrasts corresponding to incongruent neutral, incongruent negative, incongruent positive, congruent neutral, congruent negative, and congruent positive conditions were defined. At group level, these contrasts from each participant were transferred to random effects analysis. A  $2 \times 3$  within subjects repeated measures ANOVA with congruency (2 levels) and emotion (3 levels) as main factors was performed using a simple flexible factorial model in SPM.

### *ROI analysis*

Functional search volumes were defined by drawing spheres of 10-mm radius around the peak MNI coordinates drawn from an independent sample (Kanske & Kotz, 2011c) defining the dorsal and ventral ACC. Parameter estimates were extracted from the beta images by defining a sphere of 6 mm around a single participant peak within the functional search volume. Effect sizes were calculated as percent signal change ( $PSC = [\text{beta}(\text{task}) * \text{max}(\text{HRF}) * 100] / [\text{beta}(\text{constant})]$ ) where  $\text{beta}(\text{task})$  refers to the parameter estimate of the effect of interest,  $\text{max}(\text{HRF})$  is the maximum of the a single event of the current duration convolved with the current basis function, and  $\text{beta}(\text{constant})$  the parameter estimate of the current session constant) using the rfxplot toolbox (<http://rfxplot.sourceforge.net/>) with task (all regressors correspond to active conditions, i.e., incongruent neutral, congruent neutral, incongruent negative, congruent negative, incongruent positive, congruent positive) versus null contrast as the unbiased effect of interest (Gläscher, 2009). PSC estimates the evoked change in BOLD response for a condition between two conditions. For more detailed information on this procedure, please refer to <http://rfxplot.sourceforge.net/documentation/manual.pdf>.

### *Whole brain analysis*

Whole brain activations corresponding to the following contrasts were assessed: (i) main effect of congruency (incongruent > congruent), (ii) main effect of emotion ([negative + positive] > neutral), (iii) interaction of congruence and emotion, (iv) main effect of negative emotion (negative > neutral), and (v) main effect of positive emotion (positive > neutral). Final whole brain activations are reported at  $p < 0.001$  and a minimum cluster size of 17 contiguous voxels. We applied a well-validated Monte-Carlo simulations approach to correct for multiple comparisons (cluster\_threshold\_beta.m; The Mathworks, Natick, MA; 2015a; Slotnick, 2017a, 2017b; Slotnick, Moo, Segal, & Hart, 2003; Slotnick & Schacter, 2006). After running 10,000 simulations, it was determined that for an individual voxel threshold of  $p < 0.001$ , a cluster-extent threshold of 17 contiguous voxels (equivalent to a volume of 136 mm<sup>3</sup>) was necessary to correct for multiple comparisons to achieve a significance

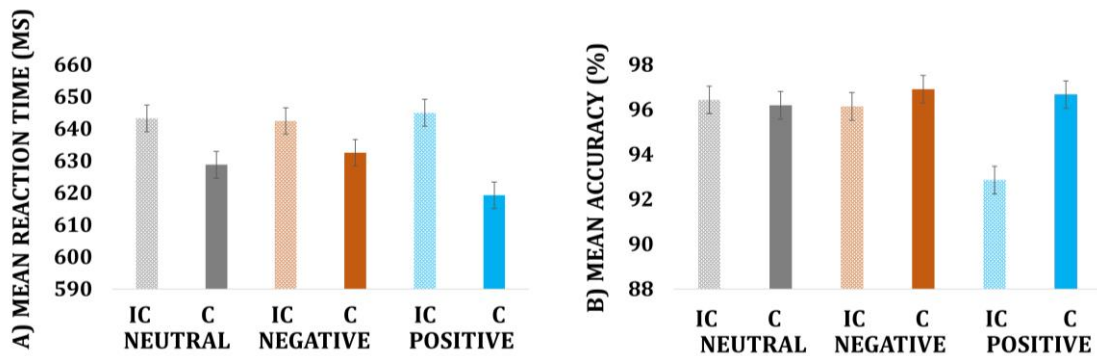
level of  $p < 0.05$ . Therefore, only clusters of activation equal or exceeding that size were considered significantly active.

### 3. Results

#### *Behavioral data*

The within-subjects ANOVA of mean RTs with the factors congruency (incongruent and congruent) and emotion (neutral, negative, and positive) yielded a significant main effect of congruency ( $F(1, 20) = 16.081$ ,  $p = 0.001$ ;  $\eta^2p = 0.446$ ), whereas there was no significant effect of emotion ( $F(2, 40) = 0.278$ ,  $p = 0.759$ ;  $\eta^2p = 0.014$ ) or a congruence-by-emotion interaction ( $F(2, 40) = 1.013$ ,  $p = 0.372$ ;  $\eta^2p = 0.048$ ) (figure. 2a; table 1). Analysis of mean percent accuracies yielded no significant main effects of congruency ( $F(1, 20) = 3.828$ ,  $p = 0.065$ ;  $\eta^2p = 0.161$ ), emotion ( $F(2, 40) = 2.068$ ,  $p = 0.14$ ;  $\eta^2p = 0.094$ ) or interaction of these factors ( $F(2, 40) = 2.270$ ,  $p = 0.117$ ;  $\eta^2p = 0.102$ ) (figure. 2b; table 1).

**Figure 2:** A) Mean reaction times for correct responses in milliseconds. B) Mean accuracy expressed as percent correct responses for each condition. Vertical bars indicate the standard error of mean.



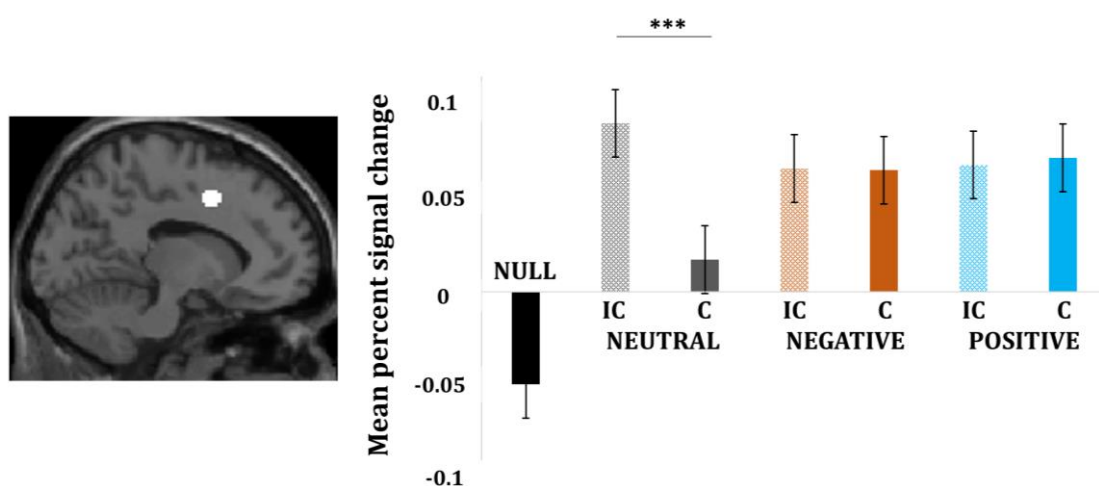
Note: IC = incongruent, C = congruent; \*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$ .

## Neuroimaging data

### ROI analyses

Repeated-measures ANOVA were performed on percent signal change (PSC) values with congruency and emotion as factors for each ROI (figure. 3). The dorsal ACC showed no significant main effect of congruency ( $F(1, 20) = 3.582, p = 0.07; \eta^2p = 0.152$ ), no significant effect of emotion ( $F(1, 20) = 0.844, p = 0.44; \eta^2p = 0.04$ ), but a significant congruence-by-emotion interaction ( $F(2, 40) = 8.458, p = 0.001; \eta^2p = 0.30$ ). Follow-up analyses revealed a significant difference between incongruent compared with congruent neutral ( $t(20) = 4.722, p < 0.001$ ) but not for incongruent vs. congruent negative ( $t(20) = -0.095, p = 0.92$ ) or incongruent vs. congruent positive trials ( $t(20) = -1.009, p = 0.32$ ). No significant neural activity was found in the ventral ACC.

**Figure 3:** Percent signal change and contrast estimates for dorsal ACC [-11 7 44]. Vertical bars in the graphs indicate SEM. Activations are reported at  $p < 0.001$ , cluster FDR  $< 0.05$  (in accordance with Monte Carlo simulations), cluster size  $> 17$  voxels; IC = incongruent, C = congruent



*Note:* Activations are reported at  $p < 0.001$ , cluster FDR  $< 0.05$  (in accordance with Monte Carlo simulations), cluster size  $> 17$  voxels; IC = incongruent, C = congruent; \*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$ .

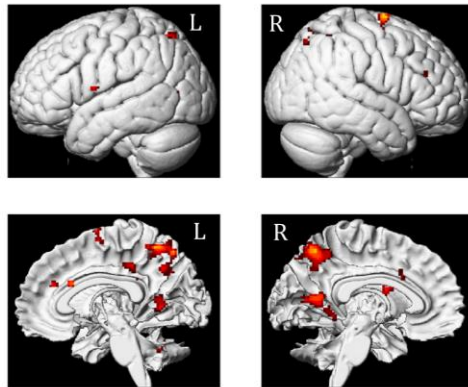


### *Whole brain analyses*

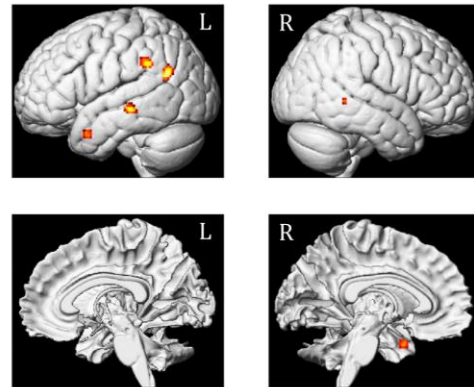
A  $2 \times 3$  within subjects ANOVA with congruency and emotion as main factors using a simple flexible factorial model in SPM yielded a significant main effect of congruency in a frontoparietal network consisting of the right superior frontal gyrus, the left and the right superior parietal lobule, the right precuneus, the right middle frontal gyrus, the left middle cingulate cortex, the left precentral gyrus, and the right cerebellum (figure. 4; table 2). The main effect of emotion was found in the left middle temporal pole, left middle temporal gyrus, left supramarginal gyrus, left angular gyrus and right middle temporal gyrus. The main effect of emotion was further broken down into the main effect of negative and positive emotion (table 2). Last, an interaction of congruence and emotion did not confirm significant whole brain activity.

**Figure 4:** Whole brain activations A) Main effect of congruency (*IC > C irrespective of stimulus type*); B) Main effect of emotions [(Positive + Negative) vs. Neutral]; C) Main effect of Negative (vs. neutral) emotion; D) Main effect of Positive (vs. neutral) emotion.

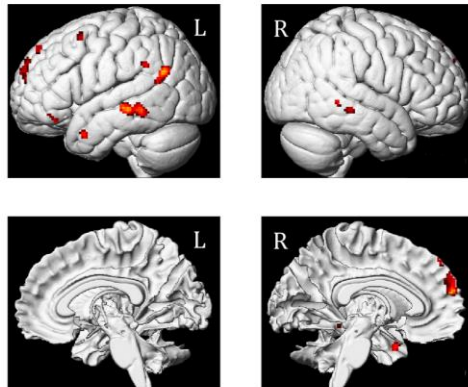
**A) MAIN EFFECT OF CONGRUENCY (IC > C IRRESPECTIVE OF THE STIMULUS TYPE )**



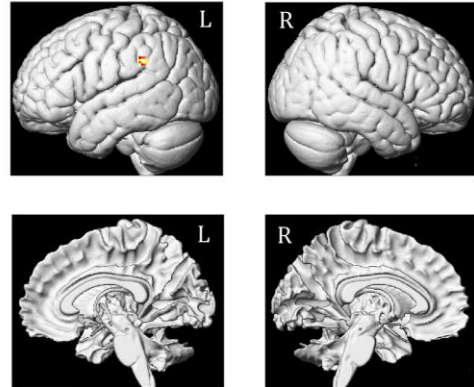
**B) MAIN EFFECT OF EMOTION (EMOTIONS > NEUTRAL TRIALS IRRESPECTIVE OF THE CONGRUENCY)**



**C) MAIN EFFECT OF NEGATIVE EMOTION (NEGATIVE > NEUTRAL )**



**D) MAIN EFFECT OF POSITIVE EMOTION (POSITIVE > NEUTRAL )**



*Note:* Activations are reported at  $p < 0.001$ , cluster FDR  $< 0.05$  (in accordance with Monte Carlo simulations), cluster size  $> 17$  voxels; IC = incongruent, C = congruent; \*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$ .

**Table 2:** Whole brain activations for within subjects ANOVA with congruency and emotions as factors. All activations are reported at  $p < 0.001$ , cluster FDR  $< 0.05$  (in accordance with Monte Carlo simulations), cluster size  $> 17$  voxels.

Brain region	BA	Peak MNI coordinate			Voxel level T score	Cluster size K (voxels)
		x (mm)	y (mm)	z (mm)		

<i>a) Main effect of congruency (IC &gt; C irrespective of the stimulus type)</i>						
R. Superior Frontal Gyrus	-	16	0	76	4.46	129
R. Superior Frontal Gyrus*	6	22	-6	62	3.44	
L. Superior Parietal Lobule	-	-20	-64	52	4.42	341
R. Precuneus	-	14	-56	52	4.00	331
R. Superior Parietal Lobule*	7	22	-70	50	3.71	
R. Middle Frontal Gyrus	-	32	34	24	3.64	25
L. Precentral Gyrus	-	-52	0	12	3.59	19
L. Middle/Anterior Cingulum	32	-8	14	36	3.47	22
R. Cerebellum_6	-	28	-60	-28	3.46	53
<i>b) Main effect of emotion (emotional &gt; neutral trials irrespective of the congruency)</i>						
L. Middle Temporal Pole	-	-44	8	-28	4.12	46
L. Middle Temporal Gyrus	-	-54	-28	-6	4.06	95
L. SupraMarginal Gyrus	40	-60	-44	32	3.91	92
L. Angular gyrus	39	-50	-62	26	3.88	114
R. Middle Temporal Gyrus	-	56	-42	0	3.42	17

Brain region	BA	Peak MNI coordinate			Voxel level	Cluster size
		x (mm)	y (mm)	z (mm)	T score	K (voxels)
c) Main effect of negative emotion (negative > neutral trials)						
L. Middle Temporal Gyrus	-	-56	-28	-6	4.39	250
L. Middle Temporal Gyrus*	21	-64	-38	-6	3.71	
L. Middle Temporal Gyrus*	-	-58	-44	-10	3.48	
L. Middle Temporal Pole	38	-42	8	-30	4.05	36
L. Superior Frontal (Medial)	10	-6	60	22	3.80	200
L. Superior Frontal (Medial)*	9	-4	54	28	3.58	
R. Middle Temporal Gyrus	-	52	-32	-8	3.79	38
L. Angular Gyrus	39	-52	-62	26	3.76	132
L. Superior Temporal Gyrus *	22	-62	-58	18	3.48	

L. Middle Frontal Gyrus	-	-40	12	58	3.64	22
L. Superior Frontal (Medial)	6	-10	48	46	3.58	26
L. SupraMarginal Gyrus	40	-60	-46	32	3.49	28
R. Middle Temporal Gyrus	-	56	-42	-2	3.44	17
L. Inferior Frontal (Orbital) Gyrus	-	-40	34	-18	3.36	21
L. Inferior Frontal (Orbital) Gyrus*	11	-44	38	-12	3.24	
d) <i>Main effect of positive emotion</i> (positive > neutral trials)						
L. SupraMarginal Gyrus	40	-60	-42	32	3.47	34

Note: \* denotes a subpeak within a cluster. L = left, R = Right.

#### 4. Discussion

The current study examined whether negative and positive emotions differentially modulate attentional control in conflict processing or act as global indicators of salience in conflict processing. In particular, the study sought to examine the valence specific influence of emotion on dorsal and ventral ACC in conflict processing. The behavioral results replicated the classic conflict effect, i.e., slower responses for incongruent than congruent trials (Eriksen & Eriksen, 1974). However, the task did not produce a significant effect of emotional valence on conflict processing. Region of interest analysis revealed a general role of the dorsal ACC in monitoring conflict and appropriate response selection irrespective of emotional valence or the level of conflict (figure 3). Furthermore, whole brain analyses showed that both negative and positive emotion elicited activity in an extensive network of brain regions associated with controlling the response to interference caused by emotion in conflict processing. This might indicate that emotion marks salience and engages attentional control in order to maintain task-performance.

##### *Behavioral response conflict*

We replicated the behavioral main effect of conflict (Eriksen & Eriksen, 1974) such that slower responses for incongruent as compared to congruent trials were observed. However, we did not find any significant effect of emotional valence on conflict

processing. In this respect, our findings differ from previous behavioral results obtained with a similar version of the flanker task with negative emotional stimuli (Kanske & Kotz, 2011c) or positive emotional stimuli (Kanske & Kotz, 2011a). These earlier studies reported shorter RTs for incongruent emotional than neutral trials, suggesting that negative and positive emotion both facilitate conflict processing. The divergent findings likely reflect differences in the task-context. While previous studies examined the effects of negative and positive emotional stimuli in separate sessions (Kanske & Kotz, 2011a, 2011c), we presented them in close temporal proximity in a fully randomized order within a run. In the present study, neutral, negative, and positive trials were presented in equal numbers within each experimental run to balance contrasts for positive and negative emotional stimuli. However, this resulted in greater potential for switching costs between trials of different congruence, arousal, and valence. This might have created an experimental context wherein higher cognitive effort was required to sustain attentional control. Furthermore, the switching between negative and positive emotional trials may have diluted their individual effect on conflict processing. Previous studies using neutral, negative, and positive emotional stimuli in a flanker task primarily focused on a carry-over effect of enhanced cognitive control that originated in the previous trial (Alguacil, Tudela, & Ruz, 2013; Gratton, Coles, & Donchin, 1992; Landman & van Steenbergen, 2020; Zeng et al., 2016). More specifically, this means that the reaction time in the current trial is reduced if it is preceded by an incongruent compared with a congruent trial. While these studies (Landman & van Steenbergen, 2020; Zeng et al., 2016) reported increased engagement of cognitive control if the previous trial was incongruent for both negative and positive compared with neutral trials, they did not report any reduced conflict/interference effect for emotional compared to neutral conditions. This missing conflict effect for emotional stimuli is inconsistent with some previous studies (Kanske & Kotz, 2011a, 2011b, 2011c, 2011d, 2012b). This may be due to differences in experimental design (blocked or mixed) or the difficulty of the task (two-response vs. four response paradigms or two vs. four flankers). Considering that the influence of emotion on conflict processing may depend on the broader experimental context, future studies need to validate these interpretations by looking into the response switching costs and conflict adaptation, analyzing the effect of the

previous (emotional/nonemotional or congruent/incongruent) trial on the reaction time or accuracy of the current trial (Chechko, Kellermann, Schneider, & Habel, 2014; Chen, Li, He, & Chen, 2009).

#### *Region of interest analysis: dorsal and ventral ACC*

We replicated the expected increase in dACC activation for incongruent compared to congruent trials for neutral stimuli. However, with emotional stimuli the dACC also displayed increased activation for congruent trials (figure 3). Hence, while we confirm the expected role of the dACC in detecting a response conflict, we observed an unexpected increase in activation linked to emotion alone. This dACC response in emotional congruent trials may nonetheless be attributed to the presence of conflict or interference in these trials. While during the incongruent emotional trials, both the color of the top and bottom flanker words and the emotional meaning of the word interferes with the judgment of, and response to the task-relevant ink color of the centrally presented target word (figure 1), interference/conflict during congruent emotional trials is created only by the emotional meaning of the word, much like in the emotional Stroop trials (Feroz, Leicht, Rauh, & Mulert, 2019; Song et al., 2017). Accordingly, recruiting the dACC in both congruent and incongruent emotional trials can be attributed to its increased responsiveness to the occurrence of conflicts to the information processing (Botvinick, Cohen, & Carter, 2004; Mayer et al., 2012; Spunt, Lieberman, Cohen, & Eisenberger, 2012; Xu et al., 2016). This interpretation is in line with previous conflict studies that report consistent dACC activity in the presence of conflict irrespective of the presence of emotional stimuli (Egner, Etkin, Gale, & Hirsch, 2008; Feroz et al., 2019; Kanske & Kotz, 2011c; Song et al., 2017; Spunt et al., 2012). Thus, the recruitment of dACC regardless of the level of conflict or emotionality of the stimulus, points toward a more general role of this brain region in assessing and monitoring incompatible information during parallel cognitive demands and appropriate response selection during conflict processing (Aarts, Roelofs, & van Turennout, 2009; Brockett & Roesch, 2021; Brockett, Tennyson, deBettencourt, Gaye, & Roesch, 2020; Goldfarb & Henik, 2007; Mayer et al., 2012; Xu et al., 2016).

According to previous studies (Kanske & Kotz, 2011c; Mohanty et al., 2007), activity in the vACC is associated with inhibition of emotional distractors and successful conflict

resolution. This is depicted by faster responses during incongruent emotional trials or smaller conflict effect as compared to neutral trials or neutral conflict, respectively. Unlike these studies (Kanske & Kotz, 2011c; Mohanty et al., 2007), the constant high demand in attentional control that was likely introduced by all high-arousal emotional trials and/or the switching costs between trials may be the reason for the lack of a significant response in the emotional subdivision of ACC (Song et al., 2017).

#### *Whole brain neural activity*

A fronto-parieto-cerebellar network of brain regions was more active during incongruent than congruent trials irrespective of stimulus type (table 2). These results are in line with an extant literature that indicates the involvement of these regions in maintaining attentional/cognitive control in conflict processing irrespective of stimulus quality (e.g., emotionality). The right superior frontal gyrus is associated with conflict anticipation and inhibition of impulsive responses during conflict processing (Aarts et al., 2009; Hu, Ide, Zhang, & Li, 2016; Ovaysikia, Tahir, Chan, & DeSouza, 2011), the middle frontal gyrus with inhibitory control and conflict processing in the presence of both emotional and nonemotional stimuli (Berron, Fruhholz, & Herrmann, 2015; Cservedka, Stroup, Etkin, & Nagel, 2015; Fan et al., 2007; Sebastian, McCrory, De Brito, & Viding, 2017) and the middle/anterior cingulate cortex with conflict monitoring and detection regardless of the stimulus quality (e.g., emotionality) (Botvinick, 2007; Braem et al., 2017; Carter & van Veen, 2007; Kanske & Kotz, 2011c; Kim, Chung, & Kim, 2013; Palermo, Stanziano, & Morese, 2018). Similarly, the superior parietal lobule has been associated with response conflict and anticipation, contextual interference and biasing of attention (Berron et al., 2015; Durston et al., 2003; Fan et al., 2007; Fruhholz, Fehr, & Herrmann, 2009; Fruhholz, Godde, Finke, & Herrmann, 2011) and the cerebellum with mediating conflict resolution by modulating response selection and biasing attention to detect change in the environment (Becerril & Barch, 2013; Kotz, Stockert, & Schwartz, 2014; Schweizer et al., 2007).

While both negative and positive emotion elicited a response in the left supramarginal gyrus, negative emotion activated a more extensive network of brain regions comprising the left and right middle temporal gyrus, the left middle temporal pole, the left superior/medial frontal gyrus, left angular gyrus/the left superior temporal gyrus,

the left middle frontal gyrus, and the left inferior (orbital) frontal gyrus (table 1). These brain regions have been suggested to play a role in controlling the interference caused by emotion in conflict processing. The supramarginal gyrus has been associated with emotion regulation and attention during conflict (Jiang et al., 2020; Olk, Peschke, & Hilgetag, 2015; Wadden et al., 2018), the inferior frontal gyrus with emotion regulation and suppression (Beauregard, Levesque, & Bourgouin, 2001; Berron et al., 2015; Egner, 2011; Kotz, Dengler, & Wittfoth, 2015; Ochsner et al., 2004; Wittfoth et al., 2010), and the middle temporal lobes with developing stimulus specific representations and flexible relational rules (Dougal, Phelps, & Davachi, 2007; Rose, Haider, Weiller, & Buchel, 2002). These brain regions are associated with processes related to top-down emotion regulation and attention modulation rather than bottom-up emotion perception. This might imply that emotional stimuli in the current paradigm were engaging these brain regions to sustain and regulate attentional control to focus on task-relevant aspects and maintain task performance. Similarly, the lack of activation in emotion-specific brain regions such as amygdala in emotional trials, usually reported in conflict processing tasks (Cromheeke & Mueller, 2014; Kanske & Kotz, 2011c), also points to a dampening of bottom-up emotional reactivity to sustain attentional control and maintain task-performance (McRae et al., 2010).

These results indicate that emotional contexts are salient and influence appropriate response selection even when this selection is relatively straightforward (congruent emotional trials). Further research is needed to test whether the context produced by the temporal succession of emotional valences within either a fully randomized or blocked design systematically modulates response conflict. This mixing of opposite valence dimensions may be the reason why our findings did not disentangle how valenced stimuli influence conflict processing.

## **5. Limitations**

Some limitations and caveats should be noted. A potential limitation of the current study is the low number of trials per condition. This might have influenced that prior results could not be replicated (Kanske & Kotz, 2011a, 2011b, 2011c, 2011d, 2012b), in particular the significant interaction of emotion and congruence. Furthermore, in the view of the current sample size ( $N = 21$ ), the current study might be underpowered



to estimate valence-specific effects of emotion on conflict processing. However, previous studies with similar sample size (N = 20 to 26 (Kanske & Kotz, 2011a, 2011b, 2011c, 2011d)) have reported significant interactions of emotion and congruence.

## **6. Conclusions**

This fMRI study sought to elucidate the influence of negative and positive emotion on conflict processing using a modified version of the Eriksen flanker task. Slower responses were observed for incongruent than congruent trials. However, no significant differences between negative and positive stimuli on conflict processing were observed. Functional MRI results pointed to a general role of dorsal ACC in monitoring and assessing conflict, as well as in selecting appropriate responses. Furthermore, the fMRI results showed that emotion enhances salience and drives appropriate response selection, even during low conflict, to accomplish task goals. Switching between trials of different congruence, arousal and valence may have created an experimental context that required higher cognitive effort to sustain attentional control. This also may have diluted the valence specific effects on conflict processing. Overall, our findings demonstrate that attentional control may help reduce the influence of emotional contexts in both high and low conflict situations to achieve overall task goals.

## **Acknowledgments**

The authors thank Joseph Johnson for support with data collection, David Linden for feedback on the manuscript, and Laura Verga, Radhika Rajan, and Mithun James for discussions on data analyses.



# **Chapter 03**

## **Interaction of emotion and cognitive control along the psychosis continuum: A critical review**

*Published as:* Duggirala, S. X., Schwartze, M., Pinheiro, A. P., & Kotz, S. A. (2020). Interaction of emotion and cognitive control along the psychosis continuum: A critical review. *International Journal of Psychophysiology*, 147, 156-175. <https://doi.org/10.1016/j.ijpsycho.2019.11.004>.

Note: to maintain consistency in the terminology used throughout this dissertation, the term "psychotic patients" has been revised to "patients with a psychotic disorder".

## **Abstract**


To better understand how emotion impacts cognitive control is important as both influence adaptive behavior in complex real-life situations. Performance changes in emotion and cognitive control as well as in their interaction are often described in patients with a psychotic disorder as well as in non-clinical participants who experience psychosis-like symptoms. These changes are linked to low motivation and limited social interaction. However, it is unclear whether these changes are driven by emotion, cognitive control, or an interaction of both. This review provides an overview of neuroimaging evidence on the potential interaction of emotion and cognitive control along the psychosis continuum. The literature confirms that over-sensitivity towards negative and lowered sensitivity towards positive emotional stimuli in tasks exploring emotion-cognitive control interaction are associated with the severity of positive and negative symptoms in psychosis. Changes in the dynamic interplay between emotion and context-sensitive cognitive control, mediated by arousal, motivation, and reward processing may underlie poor interpersonal communication and real-life skills in psychosis. In addition, structural and functional changes in subcortical and cortical associative brain regions (e.g., thalamus, basal ganglia, and angular gyrus) may contribute to alterations in emotion and cognitive control interaction along the psychosis continuum. There is limited evidence on how antipsychotic medication and age at illness-onset affect this interaction.

## **1. Introduction**

Next to positive (e.g., hallucinations, delusions) and negative (e.g., poverty of speech, apathy) symptoms, impairments of emotion processing, cognitive control, and their interaction are key features of psychosis (Becerril & Barch, 2011; Benes, 2010; Dichter, Bellion, Casp, & Belger, 2010; Minzenberg, Laird, Thelen, Carter, & Glahn, 2009; Ruocco et al., 2014). Emotion and cognitive control processes conjointly contribute to socially appropriate and goal-directed behavior (Drevets & Raichle, 1998; Gray, Braver, & Raichle, 2002; Pessoa, 2008). Changes in how emotion impacts cognitive control have been linked to reduced motivation and poor daily-life functioning in patients with a psychotic disorder (PP; (Anticevic, Repovs, & Barch, 2012; Becerril & Barch, 2011; Bertocci et al., 2012; Rey et al., 2014)). Neural changes observed in psychosis, such as hypoactivation of the cognitive control system (e.g., lateral prefrontal cortex [PFC], anterior cingulate cortex) or hyperactivation of the emotion processing system (e.g., amygdala, orbito-frontal cortex, hippocampus, insula), have been associated with the impaired ability to use emotional cues to guide cognitive control processes required for efficient social interactions (Anticevic, Repovs, & Barch, 2012; Becerril & Barch, 2011; Dichter, Bellion, et al., 2010). However, it is still unclear if such impairment arises from inefficient cognitive control, affected emotion processing, or from dysfunctional neural processing in brain regions that engage in converging and transferring sensory information to emotion processing and cognitive control systems (e.g., thalamus, basal ganglia, angular gyrus (Eack et al., 2016; Ettinger, Meyhofer, Steffens, Wagner, & Koutsouleris, 2014; Ettinger et al., 2015; Pauly et al., 2008; Pauly et al., 2010)). Behavioral and neuroimaging studies have shown that these changes also extend to clinically high-risk psychosis (CHR-P) individuals and non-clinical individuals with psychosis-like experiences (table 1, figure 1), suggesting that the respective impairments manifest early on and potentially deteriorate following illness onset (Addington, Penn, Woods, Addington, & Perkins, 2008; Modinos et al., 2015; Mohanty et al., 2008; Mohanty et al., 2005; Nenadic et al., 2015; Pauly et al., 2010; van 't Wout, Aleman, Kessels, Laroi, & Kahn, 2004).

**Figure 1:** The psychosis continuum. This continuum implies that patients with a psychotic disorder and non-clinical populations experience the same symptoms of psychosis, however, the quality (affect content, frequency, effect of symptoms on daily-life functioning) is different. Definitions of the groups depicted on the continuum are provided in table 1.

	Healthy control	Psychosis-prone	Clinically high-risk psychosis	Patients with a Psychotic disorder
<b>Symptoms:</b>	absent	hallucinations, delusions	hallucinations, delusions	hallucinations, delusions
<b>Distress:</b>	absent	no	may or may not be present	yes
<b>Insight:</b>	absent	yes	may or may not be present	no
<b>Reality:</b>	absent	not impaired	may or may not be impaired	impaired
<b>Affect :</b>	absent	positive	negative	negative
<b>Frequency:</b>	absent	low	medium	very high
<b>Medication:</b>	absent	no	may or may not be present	yes
<b>Example:</b>	healthy individuals who score <b>zero/low</b> on questionnaires measuring psychosis symptoms	healthy individuals who score <b>high</b> on questionnaires measuring psychosis symptoms, schizotypal personality	clinically high-risk psychosis, family history of psychosis	first-episode schizophrenia, chronic schizophrenia, schizo-affective disorder, bipolar disorder with psychosis


**Psychosis continuum**

**Table 1:** Glossary of terms.

S.No	Term	Definition
1	Affect	An abstract concept that represents the experience of an emotion. Valence, arousal, and motivation are three dimensions of affect.
2	Cognitive control	An adaptive mechanism that uses attentional processes (e.g., sustained attention, selective attention, inhibition, updating and maintenance of contextual information) to select relevant responses and to inhibit inappropriate responses while maintaining contextual information to guide goal-directed behavior (Botvinick et al., 2001; Niendam et al., 2012).
3	Clinically high-risk psychosis	Sub-clinical individuals experiencing attenuated (positive) psychotic symptoms (e.g., hallucinations, delusions) for at least 3 months that are clinically relevant but below the required threshold for a DSM-IV axis I psychotic disorder diagnosis. In this prodromal phase, individuals display decline in daily functioning for at least 12 months; however, they understand and have insight into their symptoms (for details see Woods et al., 2010; McGlashan et al., 2010; Addington et al., 2011). Depending upon the level of distress caused, these individuals may or may not receive antipsychotic medication.
4	Emotion-cognitive control interaction	The term emotion-cognitive control interaction refers to the dynamic reciprocal communication of emotion processing and cognitive control networks either directly or via intermediary associative brain regions (Eack et al., 2016; Ettinger et al., 2014, 2015; Pauly et al., 2008, 2010). This interaction is often influenced by internal factors such as motivation, arousal and self-regulation.

5	Hyperarousability	A personality trait where external or internal stress/stressful events cause a strong increase in arousal making an individual over-sensitive towards environmental stimuli. This state of hyperarousal or over-sensitivity may cause anomalous perceptual experiences such as hallucinations in highly vulnerable individuals (Clamor et al., 2015).
6	Patients with a psychotic disorder	Patients suffering from schizophrenia and related disorders such as schizoaffective disorder and bipolar disorder with psychosis.
7	Psychosis	Transdiagnostic range of conditions symbolized by severe distortions in thoughts and emotions. Positive symptoms include false beliefs (delusions) and anomalous perceptual experiences (hallucinations), whereas negative symptoms include depression, apathy, and anhedonia.
8	Psychosis-prone individuals	Non-clinical healthy individuals with psychotic-like experiences (e.g., hallucinations). These symptoms are not clinically relevant and therefore have no negative effect on daily life functioning and nor cause distress to the individual (Allen et al., 2006). As the symptoms cause no distress, these individuals are not under antipsychotic medication. These individuals usually score high on questionnaires measuring predisposition to symptoms of psychosis (e.g., Launay-Slade Hallucinations Scale).

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The current review has two main goals: (i) to identify and discuss the factors affecting the interaction of emotion and cognitive control, and (ii) to distinguish whether altered emotion processing is a defining feature of psychosis independent of cognitive control or altered cognitive control results in impaired emotion processing. A systematic review of the functional magnetic resonance imaging (fMRI) literature on the interaction of emotion and cognitive control along the psychosis continuum (figure 1), published until January 2019, was conducted using the PubMed database (<https://www.ncbi.nlm.nih.gov/pubmed>). In line with the definition of cognitive control used in table 1, the review focuses on experimental studies employing typically used “cognitive control” tasks (e.g., Stroop, Flanker or Simon task) as well as an n-back working memory task, a continuous performance task, an oddball paradigm or Go/No-Go and emotion regulation tasks where attentional processes involved in cognitive control are used to examine the impact of emotion on cognitive control. Advanced search terms included the keywords (“schizophrenia” or “psychosis” or “bipolar disorder” or “schizotypy” or “schizotypal personality disorder” or “at-risk psychosis” or “high-risk psychosis” or “psychosis-prone”) AND (“functional magnetic resonance imaging” or “fMRI” or “functional neuroimaging”) AND (“cognitive control” or “cognitive conflict” or “conflict resolution” or “executive control” or “attentional control” or “executive attention” or “executive functioning” or “attention” or “Stroop” or “Flanker” or “Simon”) AND (“affect” or “emotion perception” or “emotion dysregulation” or “emotion regulation” or “emotional conflict” or “emotional control” or “emotion”). Titles and abstracts of eligible studies were first screened for these keywords before the full text was analyzed (see table 2). A total of 26 studies met the above search criteria and were included in the current review.

**Table 2:** Summary of fMRI studies examining the interaction of emotion and cognitive control in psychosis.

Study	Paradigm (stimuli)	Emotion	Patient Information						Patient group as compared to control group	Instructions and performance	and
		Valence (sub-type); modality	Task-relevance	Number	Age [patient (SD), HC(SD)]	Illness duration (SD)	Medication (typical/atypical antipsychotics)	Neuropsychological assessment			
Schizophrenia											
(Pauly et al., 2008)	2-back WM task (letters)	Negative (disgust/unpleasant), neutral (olfactory)	Irrelevant, non-target	12 Adolescent-onset SZ; 12 HC	17.5 (0.55); 17.5 (1.76)	1.4*	Yes (atypical)	PANSS, GAF, CPT-IP, LN span, TMT, MWT-B, verbal fluency, Sniffin’ sticks	• Interaction of WM and negative emotion: ↑ activity in EP regions (OFC, mFC) ↓ activity in WM regions (dlPFC, ACC)	I: 2-back task instructions, respond to target accurately and quickly; emotion manipulation through olfactory simulation every 5 seconds P: decreased RT for emotional trials in both groups but no significant main effect of group or interaction of group and emotion	
(Dichter, Bellion, et al., 2010)	Forced-choice visual oddball task (circles,	Negative (aversive), neutral (pictures)	Irrelevant, non-target	12 SZ/schizoaffective disorder, 13 HC	29.4 (10.2); 31.6 (10.7)	No info	Yes (atypical and other)	SANS, SAPS, IQ test, NART	• Inhibition of aversive stimuli: ↓ activity in executive and limbic regions (dlPFC, ACC)	I: left index finger response to circles and right to all others P: slower response and poor accuracy in SZ but	

	squares, aversive and neutral pictures)								<ul style="list-style-type: none"> <li>• Inhibition of aversive stimuli: ↑ activity in ventral fronto-limbic areas for aversive stimuli</li> <li>• Target stimuli: ↑ activity in executive and limbic regions (dlPFC, ACC)</li> <li>• Negative &gt; positive and neutral: ↑ Amygdala, hippocampus, middle frontal (dlPFC) activity</li> <li>• No effect of medication.</li> </ul>	no significant main effect of group
(Becer ril & Barch, 2011)	N-back task with neutral, happy and fearful faces	Positive (happy), negative (fearful) and neutral faces	Irrelevant, target dimension	38 SZ; 32 HC	36.66 (9.12); 36.19 (10.86)	17.4 (11.2)	Yes (atypical)	SANS, SAPS	<ul style="list-style-type: none"> <li>• Negative &gt; positive and neutral: ↑ Amygdala, hippocampus, middle frontal (dlPFC) activity</li> <li>• No effect of medication.</li> </ul>	I: n-back task instructions P: RT: Neutral > Negative (significant): SZ Positive > Neutral: SZ Neutral= Negative: HC Positive = Neutral: HC <i>Accuracy:</i> Negative > Neutral: SZ (significant) Neutral > positive: SZ Negative> neutral: HC Neutral > positive: HC
(Diaz et al., 2011)	Short term memory task: encode, maintain (while	Negative (no info) and neutral	Irrelevant, Non-target	11 SZ; 17 HC	32.57 (12.7); 24.01 (3.89)	No info	Yes (mixed psychotropics)	PANSS	<ul style="list-style-type: none"> <li>• No effect of negative (vs. neutral) distractors in SZ; ↓ activity in pre-frontal and amygdala regions in SZ</li> </ul>	I: encode and retrieve P: no significant interaction of group and trial type/emotion

	looking at negative and neutral distractor pictures) and recognize through forced choice probe									Same performance for negative and neutral trials in SZ
(Anticevic, Repovs, Corlett, & Barch, 2011)	Visual WM task (encode, delay fixation, distractor, delay fixation, retrieve)	Negative (threat: on the basis of picture) and neutral	Irrelevant	28 SZ; 24 HC	36.39 (9.54); 37.18 (7.59)	No info	Yes (no info)	SAPS, SANS	<ul style="list-style-type: none"> <li>Negative: ↓ activity in dlPFC, vlPFC, amygdala as compared to neutral and HC</li> </ul>	I: encode and retrieve P: significant effect of diagnosis x distraction however, no difference in interference between negative and neutral for SZ
(Anticevic, Repovs, & Barch, 2012)	Simple perceptual decision task (fixation→emotionally aversive/neutral distractor)	Negative (threat) and neutral pictures	Irrelevant; Shown before and during the trial, non-target	28 SZ; 24 HC	36.79 (7.72); 36.73 (8.85)	No info	Yes (no info)	SANS, SAPS	<ul style="list-style-type: none"> <li>Negative &gt; neutral: negative connectivity between amygdala and prefrontal cortex in SZ</li> </ul>	I: indicate the location of blue circle P: SZ were slower for negative distraction but no significant interaction of group x distraction effect

	→ flanked by blue and green circle→fix ation)									
(Verca mmen et al., 2012)	Verbal emotional go/no-go task (words)	Positive (no info), negative (no info) and neutral	Relevant (attend) and irrelevant (inhibit) blocks	20 SZ; 23 HC	34.4 (7.8); 33.3 (7.1)	No info	Yes (atypical)	PANSS, SQLS, WAIS- III	<ul style="list-style-type: none"> <li>• Negative inhibition: No activity in dorsal prefrontal-parietal network for SZ</li> <li>• Positive inhibition: ↑ activity in middle frontal cortex</li> </ul>	I: inhibit or attend instructions before the block P: SZ were significantly slower and made more errors in emotional trials (harder to inhibit negative than positive distractors)
(Tully, Lincol n, & Hooke r, 2014)	Multi- source interferenc e task with negative and neutral pictures in the backgroun d (MSIT- Emotion)	Negative (sad) and neutral	Irrelevant, non-target	23 SZ; 24 HC	39.3 (9.60); 35.54 (12.23)	No info	Yes (both typical and atypical)	PANSS; SAS-SR	<ul style="list-style-type: none"> <li>• Negative incongruent: ↓ activity in dorsal LPFC in SZ</li> </ul>	I: identity of number different from the other two by pressing a button (task is a combination of Flanker and Simon) P: no significant main effect of group; SZ responded significantly slower and made more errors during negative as compared to neutral trials
(Kim, Yang, &	Verbal memory retrieval (encode,	Negative (mixed- threat) and neutral	Irrelevant, target dimension	15 SZ; 15 HC	28.4 (8.0); 29.7 (5.3)	6.8 (4.5)	Yes (psychotropic, no info)	PANSS	<ul style="list-style-type: none"> <li>• ↓ activity in OFG and insula for negative words</li> </ul>	I: Memorize and retrieve P: Performance decreased with negative emotional words more

Jeong, 2015)	delay, retrieve, words)									for SZ than HC; no significant group difference on accuracy during retrieval trials
(Eack et al., 2016)	n-back task (letters, random flanking of emotional faces to create emotional interference)	Positive (happy), negative (fear) and neutral	Irrelevant, non-target, present in some trials	20 SZ; 20 HC	27.80 (6.61); 26.50 (5.82)	4.85 (3.18)	Yes (psychotropic, no info)	BPRS, GAS	<ul style="list-style-type: none"> <li>• Positive &gt; negative emotion stimuli: ↑ activity in bilateral striatum, OFG, vmPFC</li> </ul>	I: n-back task instructions P: no significant effect of group or condition or emotion or group-condition interaction.
(Mukherjee et al., 2016)	Modified version of the emotional face assessment paradigm (face match)	Negative (mixed: anger, fear) and neutral	Irrelevant, target	22 SZ, 29 HC	45.82 (7.55); 46.38 (7.27)	No info	No info	Social functioning, deficit syndrome severity, SES	<ul style="list-style-type: none"> <li>• Emotional distractors: ↓ connectivity between amygdala and mPFC; ↓ activity in dACC</li> </ul>	I: face match P: accuracy decrease for emotional trials in SZ as compared to both neutral and HC
(Comte et al., 2017)	Variable attention and congruency task (VAAT,	Positive (joy) and negative (mixed: fear, disgust, anger)	Relevant, target	26 SZ; 33 HC	32.31 (8.87);	No info	Yes (no info)	PANSS	<ul style="list-style-type: none"> <li>• ↓ amygdala and ACC activity for bottom up processes; ↑ connectivity between</li> </ul>	I: Determine the emotional content of center face or background picture

	visual/emotional pictures)								vACC and dACC and vACC and dlPFC	P: SZ significantly slower but no main effect of valence
									• Negative correlation between dlPFC and antipsychotic dosage	
(Park, Chun, Park, Kim, & Kim, 2018)	Visual Simon task	Positive (mixed) and negative (mixed)	Relevant, target	17 SZ; 20 HC	27.2 (7.3); 26.1 (5.1)	No info	Yes (typical)	PANSS	• Negative emotion: ↓ amygdala activity and ↓ connectivity with dlPFC • Controlled for medication effects	I: right or left button in response to positive or negative emotion P: significant slower RT for both negative and positive trials in SZ

### Bipolar Disorder

(Pavuluri, O'Connor, Harral, & Sweeney, 2008)	Pediatric color word matching task (words)	Positive (happy, excitement), negative (sad, depressed) and neutral	Target, irrelevant	10 PBD; 10 HC	15.0 (2.36); 16.2 (1.32);	4 months before testing	No	WASI; YMRS; CDRS-R; WRAT-3, Reading (SS)	• Negative: ↑ activity in rACC and left amygdala, ↓ activity in vlPFC, dlPFC  • Positive: ↓ posterior ACC, insula, vlPFC, OFC; ↑ activity in amygdala	I: match color of the word with one of two colored circles below P: overall PBD slower and less accurate than HC but no significant main effect of group; significant effect of valence. RT: Negative > Neutral > Positive
(Passarotti, Sweeney, & Passarotti, 2018)	2-back task (faces)	Positive (happy), negative	Target, relevant	17 PBD; 13 HC	14.29 (2.05); 14.38 (3.57)	No info	Yes (medication free 7 days before baseline scanning, second	YMRS; WASI-FSIQ; CDRS-R;	• Negative: ↓ activity in vlPFC, caudate	I: 2-back: match both face and emotion P: No significant difference between BD

Pavuluri, 2011)		(angry) and neutral					generation antipsychotics)		<ul style="list-style-type: none"> <li>• Positive: ↑ activity in amygdala, vIPFC, r-dIPFC, MTG, MFG</li> </ul>	and HC for RT; more errors in BD than HC in emotional conditions; overall PBD slower and less accurate than HC PBD → RT: Negative > positive > neutral Accuracy: positive > negative ≈ neutral HC → RT: negative > neutral > positive Accuracy: positive > negative ≈ neutral
(Bertucci et al., 2012)	2-back task (letters, flanked by emotional face distractors)	Positive (happy), negative (fear) and neutral	Non-target, irrelevant	18 BD; 16 HC	31.94 (8.54); 29.74 (8.22)	14.00 (6.63)	Yes (mixed psychotropic)	HAMD-25; YMRS; NART; STAI	<ul style="list-style-type: none"> <li>• ↓ activity in ACC and ↑ activity in striatum (putamen)</li> </ul>	I: n-back related P: RT was slower for face distractor trials, no significant group difference
(Townsend et al., 2013)	Emotion regulation task (pictures, passive viewing and emotion downregulation)	Negative (no info) and neutral	Target, relevant	30 BD; 26 HC	37.9 (12.6); 35.5 (12.4)	20.7 (13.6)	Yes (mixed psychotropic)	YMRS; HDRS	Emotion down-regulation: <ul style="list-style-type: none"> <li>• ↓ activation in bilateral vIPFC, bilateral ACC and posterior CC, medial frontal gyrus and bilateral dIPFC</li> </ul>	I: two conditions → passive view, decrease emotion P: NA



									<ul style="list-style-type: none"> <li>• Amygdala: ↓ activity for both groups for negative downregulation compared to observe condition</li> </ul>	
(Favre et al., 2013)	Modified version of the word-face emotional Stroop task (emotional words embedded on emotional faces)	Positive (happy), negative (fear) and neutral	Relevant, target	16 BD; 16 HC	40.47 (11.8); 40 (12.5)	13.9 (6.8)	Yes (mixed psychotropic)	MADRS; YMRS; STAI	<ul style="list-style-type: none"> <li>• Incongruent &gt; congruent: ↓ activity in bilateral inferior and left superior frontal gyri, right insula, right fusiform gyrus and bilateral occipital gyri</li> <li>• Negative &gt; positive incongruent: ↑ Bilateral hippocampus, parahippocampal gyri and the left middle temporal gyrus activity.</li> </ul>	<p>I: identify emotional expression of faces and ignore emotional words embedded on them; word and face could be congruent (both positive affect, “joy” embedded on happy face) or incongruent (one positive and other negative affect, “joy” embedded on an angry face)</p> <p>P: overall significant larger RT in BD, but similar accuracy, no significant group x valence or group x Stroop effect</p> <p>Significant effect of emotional valence: negative incongruent trials generated slower RT and more errors than</p>

(Brotman et al., 2014)	Constrained and unconstrained processing of emotional faces	Positive (happy), negative (angry and fear) and neutral	Target, relevant	36 PBD; 26 BD; 57 PHC; 62 HC	14.77(2.55); 41.70(10.30); 14.30(2.57); 34.24(9.54)	4.03*; 20.26*	Yes (mixed psychotropic)	WASI; SIGH-SAD; YMRS; CDRS	<ul style="list-style-type: none"> <li>• (PBD&gt;BD) &gt; HC explicit and implicit conditions: ↑ activity in Amygdala</li> <li>• Explicit and implicit conditions: ↓ in IFG (only positive), r-ACC (negative and positive), putamen (explicit, positive)</li> <li>• Passive view negative: ↑ r-ACC, putamen</li> <li>• ↓ activity in cognitive control network e.g., rACC, MFG</li> </ul>	<p>positive incongruent trials in both groups</p> <p>I: passive viewing, implicit attention: “How wide is the nose?”, two explicit attention: “how hostile is the face?”, “how afraid are you?”.</p> <p>P: PBD rated neutral faces as more hostile; overall patients responded significantly slower</p>
(Rey et al., 2014)	Modified version of the word-face Stroop task (emotional words embedded on	Positive (joy) and Negative (fear)	Relevant, target	11 BD; 12 HC	42.6(11.4); 41.3(12)	21.8(9.9)	Yes (psychotropic)	YMRS; MADRS-S	<ul style="list-style-type: none"> <li>• ↓ activity in cognitive control network e.g., rACC, MFG</li> </ul>	<p>I: identify emotional expression of faces and ignore emotional words embedded on them; word and face could be congruent (both positive affect, “joy” embedded on happy face) or incongruent (one positive and other negative affect,</p>

	emotional faces) with either low (same response button) or high attentional demands (response button depended upon stimulus gender)									“joy” embedded on an angry face) P: RT was significantly longer for BD for both congruent and incongruent trials; no effect of valence
(Favre, Polosa, n, Pichat, Bouge rol, & Baci, 2015)	Modified version of the word-face emotional Stroop task (emotional words embedded on emotional faces)	Positive (joy) and negative (fear)	Relevant, target	14 BD; 13 HC	44.07 (9.63); 44.08 (10.85)	16.08 (11.10)	Yes (psychotropic)	MADRS; YMRS	<ul style="list-style-type: none"> <li>• ↓ activity in dlPFC for conflict monitoring</li> <li>• ↑ activity in amygdala during emotional conflict</li> </ul>	I: identify emotional expression of faces and ignore emotional words embedded on them; word and face could be congruent (both positive affect, “joy” embedded on happy face) or incongruent (one positive and other negative affect, “joy” embedded on an angry face) P: BD were slower but the difference was not significant

(Corbala, Beaulieu, & Armony, 2015)	Emotion regulation paradigm (view or ate, pictures);	Negative (no info) and neutral	Relevant, target	19 BD; 17 HC	41.0 (12.5); 41.4 (13.3)	No info	Yes (no info)	HAMD-29; YMRS; SIGH-SAD; MADRS; CGI; STAI-S	<ul style="list-style-type: none"> <li>• Negative downregulation: ↑ amygdala activity</li> <li>• Negative passive view: ↑ vIPFC</li> <li>• Controlled for medication effects</li> </ul>	I: passive view, downregulate emotion P: not applicable
(Mukherjee et al., 2016)	Modified version of the emotional face assessment paradigm (face match)	Negative (anger, fear) and neutral	Irrelevant, target	15 BDP, 29 HC	42.93 (6.46); 46.38 (7.27)	No info	No info	Social functioning, deficit syndrome severity, SES	<ul style="list-style-type: none"> <li>• No difference between BD and HC</li> </ul>	I: face match P: no significant difference

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#### CHR psychosis or non-clinical psychosis-prone individuals

(Monty et al., 2005)	Emotional Stroop task (words)	Negative (no info), Positive (no info), neutral; verbal-visual	Irrelevant; Emotional target word	17 high PP; 17 HC	19.1 (1.9); 20.5 (3.9)	NA	No	CSPP, PSWQ	<ul style="list-style-type: none"> <li>• Negative vs Neutral: - ↑ activity in amygdala, hippocampus; right dlPFC, basal ganglia, cerebellum</li> <li>- ↓ left dlPFC, ITG, STG, MOG</li> </ul>	I: identify the color of the word, ignore the meaning P: Stroop interference of High PP was more than HC but not significantly different
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(Modinos, Ormel, & Alemana, 2010)	Passive viewing of pictures (viewing neutral, negative, and reappraising negative)	Negative, neutral; visual	Relevant, target	17 high PP; 17 low PP	High PP = 19.8 (1.8); Low PP = 21 (2.8)	NA	No	CAPE	<ul style="list-style-type: none"> <li>• No info about Positive vs. Neutral</li> </ul> <p>High PP:</p> <ul style="list-style-type: none"> <li>• ↓ prefrontal-amygdala coupling</li> <li>• ↑ prefrontal activity during reappraisal, amygdala response did not decrease during reappraisal</li> <li>• ↓ cognitive control of emotion</li> </ul>	<p>I:</p> <p>View: view the photo and experience the emotion naturally</p> <p>Attend: continue viewing</p> <p>Reinterpret: reappraise the content of negative picture so that it no longer elicited negative response</p> <p>Rate the negative affect after few seconds</p> <p>Relax: relax</p> <p>P: both groups reported successful reduction of experienced negative emotion</p>
(Pauly et al., 2010)	2-back WM task (words);	Negative (disgust), neutral; olfactory	Non-target, irrelevant	12 CHR psychosis; 12 HCs	24.22 (4.61); 24.46 (4.67)	No info	Yes (mixed bag, some received antipsychotics)	PANSS, HDRS, GAF, MWT-B, LN-span, CPT-IP, TMT, PERT, Sniffin' Sticks	<p>Interaction of emotion and WM:</p> <ul style="list-style-type: none"> <li>• ↓ activity in STG, ITG, caudate nucleus, posterior insula, supramarginal gyrus</li> <li>• ↑ activity in thalamus, cerebellum, posterior ITG</li> </ul>	<p>I: n-back WM task related</p> <p>P: no difference between groups</p>

van der Velde et al., 2015	Emotional regulation task (pictures)	Negative and neutral; visual	Target, relevant	15 CHR psychosis, 16 HC	23.1 (4.4); 22.1 (3.6)	No info	Yes (mixed, antipsychotics, antidepressants)	PANSS	• Negative appraisal: ↓ vIPFC	I: attend neutral, attend negative and reappraise P: no significant difference
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*Abbreviations:* SZ = Schizophrenia, BD = Bipolar disorder, HC = healthy controls, PP = psychosis proneness, CHR = clinically high risk, I = Instructions, P = Performance, EST = emotional Stroop task, EP = emotion processing, WM = working memory, RT = reaction time, dlPFC = dorsolateral prefrontal cortex, rACC = rostral anterior cingulate cortex, OFC = orbitofrontal cortex, mFC = middle frontal cortex, STG = superior temporal gyrus, ITG = inferior temporal gyrus, vmPFC = ventral medial prefrontal cortex, rs-fc= resting state functional connectivity, tb-fcMRI = task based functional connectivity, NA: not applicable

PANSS = positive and negative syndrome scale, SANS = scale for the assessment of the negative symptoms, SAPS= scale for the assessment of the positive symptoms, SCID – DSM = Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, SQLS = schizophrenia quality of life scale, MSCEIT = Mayer–Salovey–Caruso Emotional Intelligence Test, BPRS = brief psychiatric rating scale, VAAT = variable attention and congruency task; MADRS = Montgomery and Asberg Depression Rating Scale, YMRS = Young Mania Rating Scale, CAPE = Community Assessment of Psychic Experiences Questionnaire,; WRAT = Wide Range Achievement Test – Third Edition (Reading Subtest), KYMRS = Kiddie Young Mania Rating Scale, CDRS-R = Child Depression Rating Scale-Revised, PBD = Pediatric Bipolar Disorder, WASI IQ = Wechsler Abbreviated Scale of Intelligence Intelligent Quotient, SIGH-SAD = Structured Interview Guide for the Hamilton Depression Rating Scale; DIGS= the Diagnostic Interview for Genetic Studies; SAS-SR = social adjustment scale – self-report; CPZ= chlorpromazine; GAS = global assessment scale; PERT = Penn Emotion Recognition Test; TMT = Trail Making Test; CPT-IP = computerized identical pairs version of the Continuous Performance Test; LN span = working memory-letter–number span; MWT-B = Mehrfachwahl-Wortschatz-Intelligenztest-Version B; GAF = Global Scale of Functioning; HDRS = Hamilton Rating Scale for Depression; CSPP = Chapman scales for psychosis proneness, PSWQ = penn state worry questionnaire, NART = National adult reading test, SAS = Social adjustment scale , SES = Socio-economic status, HAMD = Hamilton depression rating scale, STAI = State-trait anxiety inventory, CGI = Clinical global impression, PERT = penn emotion recognition test

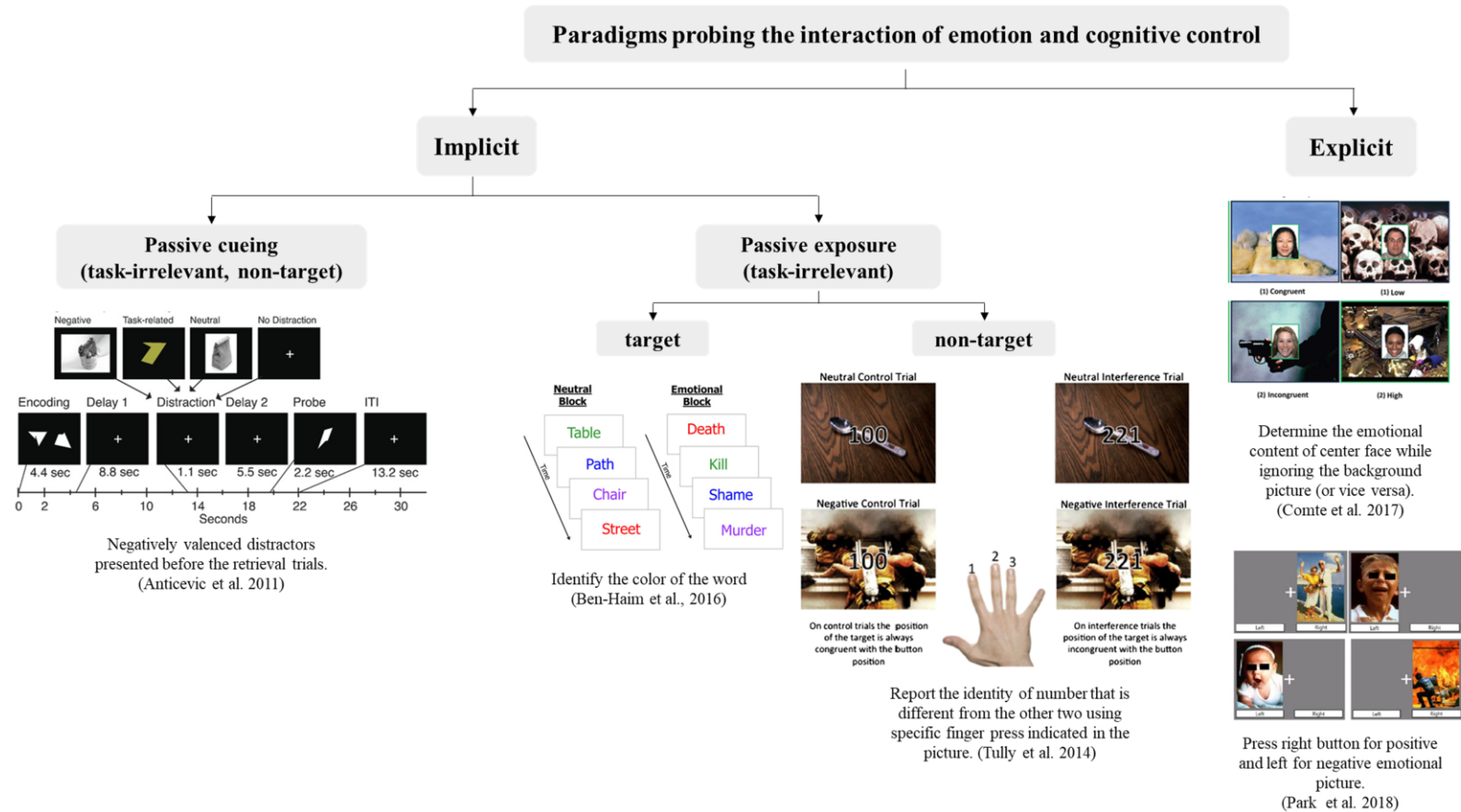
\*Illness duration was calculated by subtracting mean illness onset from mean age; ↑increased; ↓decreased.

To address our first aim, the identified studies were assessed in terms of how emotion was implemented in the respective task settings to identify a set of discernible categories influencing emotion-cognitive control interaction. These categories included valence-specificity, antipsychotic medication, age at illness onset, and illness chronicity. Each of these categories have shown to influence both cognitive control and emotion processing in psychosis (Eack et al., 2016; Pinkham, Gur, & Gur, 2007; Pino et al., 2014; Rajji, Ismail, & Mulsant, 2009). The review then discusses the mediating role of affect in the interaction of emotion and cognitive control in psychosis. Lastly, we address the likely neuroanatomical causes underlying significant changes in the interaction of emotion and cognitive control in psychosis based on the reviewed literature.

## **2. Experimental paradigms testing the interaction of emotion and cognitive control**

The interaction of emotion and cognitive control has been studied in multiple task settings. Tasks may engage conflict resolution and monitoring, selective and sustained attention, inhibition, working memory, emotion regulation, and require constant maintenance of the task context (Anticevic, Repovs, & Barch, 2012; Anticevic et al., 2011; Anticevic, Repovs, Krystal, & Barch, 2012; Becerril & Barch, 2011; Diaz et al., 2011; Dichter, Bellion, et al., 2010; Mukherjee et al., 2016; Tully et al., 2014; Vercammen et al., 2012). Distinct manipulations of emotion are introduced at different time points into these task settings. Examples include tasks in which emotional information precedes a trial, probing conflict to challenge reorienting of attention to resolve a conflict (Anticevic et al., 2011; Diaz et al., 2011). In other tasks, emotional information is presented throughout a trial to challenge the sustaining of attention and maintaining of contextual information (Anticevic, Repovs, Krystal, et al., 2012; Besnier et al., 2011; Dichter, Bellion, et al., 2010). Overall, emotion has been manipulated in cognitive control tasks in three ways (figure 2):

**Figure 2:** Paradigms testing the impact of emotion on cognitive control.





Prior passive cueing, i.e., an emotional cue is passively/implicitly presented, prior to a cognitively challenging task, without requiring an explicit behavioral response. In this case, emotion is not part of the target dimension established by the task instructions and, thereby, can be considered task-irrelevant. Although presenting emotional distractors prior to task-relevant stimuli only transiently captures attention, it disrupts the continuous maintenance of contextual information that is necessary to execute task-appropriate behavior during cognitive control tasks. This has immediate consequences for task performance, reflected in slower reaction times and higher error rates. Compared to healthy controls, PP tend to show larger interference effects when emotional distractors precede a cognitively challenging trial (such as the retrieval of information in a short-term memory task or the conflict resolution in a Stroop task) (Anticevic et al., 2011; Diaz et al., 2011). On the other hand, performance of CHR-P individuals was not significantly different from healthy controls in these tasks (Pauly et al., 2010).

Continued passive exposure, i.e., an emotional cue is passively presented in the background whilst performing an unrelated task. Here, emotion may or may not be part of the target dimension but is task-irrelevant. In this setting, task-irrelevant emotion draws attention away from task-relevant cognitive processes, resulting in deteriorated task performance. Examples include typically used conflict processing tasks such as the emotional Stroop or Flanker tasks, in which participants have to identify the ink color of emotional words while ignoring the stimulus meaning (Ben-Haim et al., 2016). Here, emotion is part of the target dimension although it remains task-irrelevant (Besnier et al., 2011). Healthy participants respond faster to negative emotion (as compared to neutral) when it is part of the target dimension that results in facilitation of conflict processing during typically used cognitive control tasks (Kanske & Kotz, 2011a, 2011b, 2011c). However, studies using cognitive control tasks such as n-back working memory task have yielded mixed results (Kessel et al., 2016; Luo et al., 2014). Compared to healthy controls, PP take longer to identify the color of negative than neutral words, indicating greater task-interference due to a heightened attentional bias towards task-irrelevant emotional information (Besnier et al., 2011). The background presentation of emotional distractors in a modified version of the Simon task is another variant of this setting. The Simon task requires participants to

respond to the location of a digit on a screen or entails a forced choice task in which participants judge the color of geometric figures embedded in the distractor (Anticevic, Repovs, & Barch, 2012; Anticevic, Repovs, Krystal, et al., 2012; Dichter, Felder, & Smoski, 2010). Here, emotion is not part of the target dimension, and it is task-irrelevant. PP take longer to react to emotional stimuli in visual and verbal n-back working memory tasks (Dichter, Bellion, et al., 2010; Dichter, Felder, et al., 2010; Pauly et al., 2008). Impaired task performance in an emotional Stroop task extends to CHR-P and non-clinical individuals who experience psychotic-like symptoms in some studies (Besnier et al., 2009; Van Strien & Van Kampen, 2009; Yaffe & Walder, 2016) whereas other studies report no significant differences between these populations and healthy controls (Mohanty et al., 2005; van 't Wout et al., 2004).

Explicit selective attention, i.e., an emotional cue is the target dimension and therefore task-relevant. Conflict or task-interference arises with competing attentional demands associated with two different emotional stimuli. These stimuli can be of the same (e.g., both visual (Comte et al., 2017) or different modalities (e.g., auditory vs. visual; (Zinchenko et al., 2017)). PP struggle to consciously and selectively monitor emotional stimuli that are of primary relevance for a task but incongruent (Comte et al., 2017; Park et al., 2018). They also experience difficulty (reflected in slower reaction times and low accuracy) in judging the emotion conveyed by a face embedded in a broader emotional background serving as an emotional distractor (Comte et al., 2017). Similarly, they showed impaired task performance (i.e., slower reaction times) in a modified version of the Simon task that uses emotional face stimuli, in which participants have to press a right-hand response button for a positive stimulus and a left-hand button for a negative emotional stimulus (Park et al., 2018). We did not find any studies with CHR-P individuals or non-clinical individuals with psychotic-like symptoms (table 2). In addition, emotion regulation tasks are also included in this category, as they require (explicit) attentional control of emotion processing regions to efficiently regulate the emotional experience (Brotman et al., 2014; Corbalan et al., 2015; Modinos et al., 2010; Townsend et al., 2013; van der Velde et al., 2015). Taken together, emotion seems to disrupt cognitive control processes in behavioral tasks and this disruption is exaggerated in PP (table 2).

### **3. Key factors contributing to the interaction of emotion and cognitive control**

Several key factors (e.g., emotional valence, medication effects, age at illness onset, illness chronicity) influencing the interaction of emotion and cognitive control can be differentiated on the basis of the current literature review. These factors are separately discussed in the ensuing sections.

#### *Emotional valence*

Although both positive and negative emotions attract attention, they evoke distinct behavioral responses (Fredrickson, 1998, 2001; Fredrickson & Branigan, 2005). Positive emotion is associated with the broadening of attentional scope, relaxation and well-being, whereas negative emotion is linked to attention constriction and decreased attentional flexibility (Eysenck, Derakshan, Santos, & Calvo, 2007; Fredrickson & Branigan, 2005; Johnson, Waugh, & Fredrickson, 2010). This suggests that they may also differentially influence task performance when emotion interacts with cognitive control (Fredrickson, 1998, 2001; Fredrickson & Branigan, 2005). Earlier studies testing healthy individuals in cognitive control tasks have shown that valence-specific influence on cognitive control depends on the context and task-relevance of the emotional stimulus (Berger, Richards, & Davelaar, 2017; Hart et al., 2010; Kanske & Kotz, 2011b, 2011c, 2011d; Kessel et al., 2016).

Most studies examining the impact of negative emotion on cognitive control in PP have reported impaired task performance (i.e., slower reaction time; (Bertocci et al., 2012; Brotman et al., 2014; Comte et al., 2017; Corbalan et al., 2015; Dichter, Bellion, et al., 2010; Favre et al., 2015; Kim, Yang, et al., 2015; Mukherjee et al., 2016; Park et al., 2018; Pauly et al., 2008; Pavuluri et al., 2008; Rey et al., 2014)), whereas others have shown an enhanced effect of negative emotion (i.e., faster reaction time) compared to neutral stimuli (Becerril & Barch, 2011), or even null effects (Anticevic et al., 2011; Diaz et al., 2011; Eack et al., 2016; Kim, Yang, et al., 2015). Impaired task performance in cognitive control tasks with negative emotional stimuli in PP could result from an enhanced attentional bias towards stimuli representing threat, fear, and paranoia. This potentially relates to specific illness symptoms (Kapur, 2003; Kinderman, Prince, Waller, & Peters, 2003). For instance, as hallucinations often elicit negative affect, PP

are likely to be more sensitive to negative cues, which may constitute more potent distractors than neutral stimuli (Kapur, 2003; Kapur, Arenovich, et al., 2005). Similarly, as negative emotions exert a lasting effect on attention (Strauss, Allen, Duke, Ross, & Schwartz, 2008), they might continue to influence cognitive processes even when the negative stimulus is absent (Anticevic et al., 2011; Diaz et al., 2011). Thereby, PP may assign salience to neutral stimuli (Kapur, 2003). As negative and neutral stimuli are presented in close temporal succession in cognitive control tasks, performance will not differ for both stimulus types. An interaction of negative emotion and cognitive control is associated with reduced neural activity in the cognitive control network and increased activity in emotion processing in PP (Bertocci et al., 2012; Brotman et al., 2014; Comte et al., 2017; Corbalan et al., 2015; Dichter, Bellion, et al., 2010; Favre et al., 2015; Kim, Yang, et al., 2015; Mukherjee et al., 2016; Pauly et al., 2008; Pavuluri et al., 2008; Rey et al., 2014). Behavioral results also extend this effect to CHR-P and non-clinical individuals with psychotic-like experiences (Besnier et al., 2009; Mohanty et al., 2005; Van Strien & Van Kampen, 2009; Yaffe & Walder, 2016). Reduced lateral PFC and increased amygdala activity has been observed in CHR-P individuals in an emotional Stroop task (Mohanty et al., 2005). Similarly, decreased functional connectivity between the PFC and amygdala was reported in an emotion regulation task in healthy individuals who are highly prone to psychotic symptoms (Modinos et al., 2010). These neuroimaging data may suggest failure of attentional control of emotional processing regions when negative emotional stimuli are used in cognitive control tasks in psychosis.

On the other hand, only a small number of studies investigated the impact of positive emotion on cognitive control and the results are inconsistent/inconclusive (table 1). Studies that examined the influence of both positive and negative emotions on cognitive control either showed a selective attentional bias towards negatively valenced stimuli (Becerril & Barch, 2011; Brotman et al., 2014; Favre et al., 2013; Pavuluri et al., 2008; Vercammen et al., 2012) or no difference between negative and positive stimuli (Bertocci et al., 2012; Comte et al., 2017; Eack et al., 2016; Park et al., 2018; Rey et al., 2014). A failure to observe a positive attentional bias may be associated with the inability to process reward-related cues due to increased levels of striatal dopamine in schizophrenia (Juckel et al., 2006). This is supported by altered

basal ganglia activity in n-back tasks with positive words in PP (Eack et al., 2016; Juckel et al., 2006). In summary, PP seem to be over-sensitive to negative emotion and under-sensitive to positive emotional stimuli, which may be related to the severity of positive and negative symptoms, respectively.

### *Medication effects*

Although antipsychotic medication has been reported to affect both cognitive control and emotion processing separately, its impact on tasks probing the interaction between emotion and cognitive control remains unclear. Studies investigating the effects of antipsychotics on emotion and cognitive control systems produced mixed findings (Cohen & Servan-Schreiber, 1992; Del-Ben et al., 2005; Harmer, Mackay, Reid, Cowen, & Goodwin, 2006; Meltzer & McGurk, 1999; Pinkham, Gur, et al., 2007; Sharma, 1999; Weinberger, Berman, & Illowsky, 1988; Weiss, Bilder, & Fleischhacker, 2002; Weiss et al., 2003). Effects of conventional antipsychotics acting on dopamine receptors (e.g., haloperidol) and atypical antipsychotics acting on both dopamine and serotonin receptors (e.g., risperidone) are distinguishable in schizophrenia and bipolar disorder with psychosis (Gaebel & Wölwer, 1992; Williams, Loughland, Green, Harris, & Gordon, 2003). While typical antipsychotic medication tends to show no favorable effect on emotion processing, atypical antipsychotics can lead to improvement in emotion perception and cognitive control that remain significant even after controlling for the effects of positive symptoms (Kee, Kern, Marshall, & Green, 1998; Williams et al., 2003). However, other studies could not replicate such beneficial effects of atypical antipsychotics on emotion processing and cognitive control, even though symptoms had been remitted (Herbener, Hill, Marvin, & Sweeney, 2005). Atypical antipsychotics have also been shown to alter the function of the amygdala and prefrontal cortex in the context of tasks tapping into emotion-cognitive control interaction (Cohen & Servan-Schreiber, 1992; Del-Ben et al., 2005; Harmer et al., 2006; Pinkham, Gur, et al., 2007; Sharma, 1999; Takahashi et al., 2005; Weinberger et al., 1988; Weiss et al., 2002; Weiss et al., 2003). However, again results are mixed. Some studies show a reduction whereas others show no effect of atypical antipsychotics on amygdala activity (Del-Ben et al., 2005; Harmer et al., 2006; Takahashi et al., 2005).

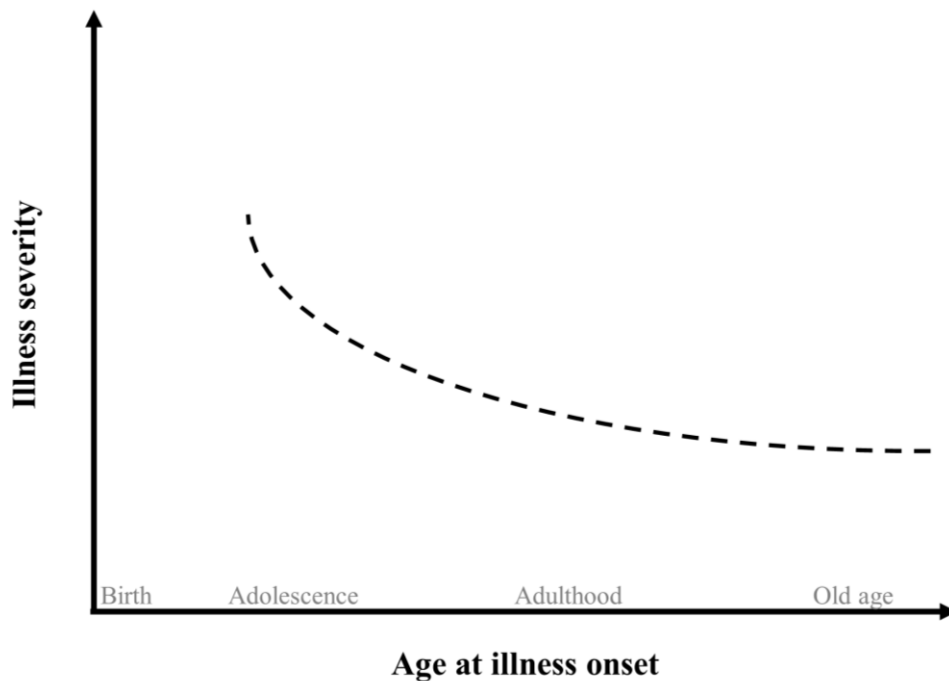
Likewise, several other pharmacological agents (e.g., selective serotonin reuptake inhibitors,  $\beta$ -adrenergic blockers; dopamine D2 receptor antagonists; anxiolytics, benzodiazepines; agonists and antagonists at the GABAA receptor) were also shown to modulate the amygdala and prefrontal activation in schizophrenia (Del-Ben et al., 2005; Harmer et al., 2006; Lewis & Gonzalez-Burgos, 2006; Paulus, Feinstein, Castillo, Simmons, & Stein, 2005; Takahashi et al., 2005; van Stegeren et al., 2007). However, the results have not been replicated (Pinkham, Gur, et al., 2007). Although the pharmacological treatment of schizophrenia involves atypical or typical antipsychotics or a combination of both, the treatment of bipolar disorder tends to rely on a combination of different types of psychotropic medications such as antipsychotics, anticonvulsants, antidepressants, anxiolytics, and mood stabilizers (table 2). As these drugs have differential effects on distinct neurotransmitter systems and brain regions, the effects of medication on behavior or neural systems in the context of emotion-cognitive control interaction remain unclear. Similarly, the studies included in the current literature review do not allow disentangling the effects of distinct antipsychotics on the interaction of emotion on cognitive control in psychosis as the evidence is mixed. However, there is some indication that antipsychotics may normalize but perhaps not completely eradicate psychosis-related changes in emotion-cognitive control interaction. This tentative conclusion is supported by studies with non-clinical individuals with psychotic-like experiences. Alterations in emotion processing, cognitive control, and their interaction have been observed in CHR-P and non-clinical individuals with psychotic-like experiences (Bourque et al., 2017; Modinos et al., 2010; Mohanty et al., 2008; Mohanty et al., 2005; Pauly et al., 2010; van 't Wout et al., 2004).

As alterations of emotion-cognitive control interaction are seen in both CHR-P and non-medicated psychosis-prone individuals, preceding the clinical diagnosis of psychosis and administration of antipsychotics, it is unlikely that the observed changes are entirely caused by medication. Future research should focus on longitudinal investigations of the psychosis continuum to explore whether and how medication affects the interactions between emotion and cognitive control in PP.

*Age at illness-onset*

Recent studies have suggested that the functional impact of psychosis is associated with age at illness-onset (Girard & Simard, 2008; Gogtay, Vyas, Testa, Wood, & Pantelis, 2011; Howard, Rabins, Seeman, & Jeste, 2000; Mason, Stott, & Sweeting, 2013). Although both early- and late-onset psychosis share the same core psychopathophysiology, symptoms tend to be more severe in early-onset psychosis (Ju, Liu, Zhang, & Zhou, 2019; Vahia et al., 2010). Accordingly, early-onset psychosis requires a higher daily dosage of antipsychotic medication (Vahia et al., 2010). Early onset of psychosis refers to experiencing psychotic symptoms either before (childhood-onset psychosis) or during the critical phase of adolescence, which is characterized by major morphological and functional changes in the brain and represent increased vulnerability to different types of psychopathology (Gogtay et al., 2011). This is particularly relevant for emotion-cognitive control interaction as structural and functional maturation of brain regions implicated in executive function and cognitive control (e.g., lateral PFC, inferior parietal lobe) occurs at the end of adolescence (Biffin et al., 2009; Frangou, 2010; Gogtay et al., 2011). This suggests that if the onset of psychosis occurs before or during adolescence, it will likely disrupt the normal maturation process in these brain regions, affecting associated cognitive functions such as executive and cognitive control (Biffin et al., 2009; Feinberg, 1982; Frangou, 2010; Gogtay et al., 2004; Gogtay et al., 2011; Vidal et al., 2006). On the other hand, studies show inconsistent findings with respect to emotion processing. While some studies reported reduced gray matter (GM) volume in insular cortex, hippocampus, and amygdala in CHR-P individuals compared to healthy controls (Velakoulis et al., 2006), others reported no significant differences (Gogtay et al., 2011). Overall, a reciprocal relationship is observed between age at illness-onset and severity of illness (figure 3).

**Figure 3:** Illness chronicity and age at illness onset. Psychotic symptoms (and prognosis) are most severe if the first-episode of psychosis occurs in early adolescence.



We did not identify any effect of age at illness-onset on emotion-cognitive control interaction in the current literature (table 2). However, extrapolating the discussed findings to the issue of how emotion and cognitive control interact, we would expect distinct changes in the dorsal cognitive control network, which, in turn, should have consequences for emotion-cognitive control interaction. Future longitudinal studies are required to specify how emotion and cognitive control mechanisms interact along the developmental trajectories of age at illness onset.

### *Illness chronicity*

Progressive decline in behavioral performance in tasks exploring emotion-cognitive control interaction has been observed along the psychosis continuum, from non-clinical individuals with psychotic-like experiences to patients (table 2). Whereas no significant behavioral differences were observed (e.g., ratings of emotional stimuli, affect downregulation, reaction time, errors) between healthy controls and non-clinical individuals with psychotic-like experiences (Modinos et al., 2010; Mohanty et al., 2005; Pauly et al., 2010; van der Velde et al., 2015), task performance was found to be significantly reduced in PP compared to healthy controls (Anticevic, Repovs, & Barch, 2012; Anticevic et al., 2011; Anticevic, Repovs, Krystal, et al., 2012; Becerril &



Barch, 2011; Comte et al., 2017; Diaz et al., 2011; Dichter, Bellion, et al., 2010; Mukherjee et al., 2016; Pauly et al., 2008; Tully et al., 2014; Vercammen et al., 2012). This is in line with several cross-sectional studies that compared CHR-P, first-episode psychosis, and chronic schizophrenia patients, where progressive deterioration in cognitive functions such as attention and executive function was observed post illness-onset in patients compared to healthy controls (Fusar-Poli, 2012; Mesholam-Gately, Giuliano, Goff, Faraone, & Seidman, 2009; Pino et al., 2014). Similarly, alterations in emotion processing were observed along the psychosis continuum from CHR-P individuals (Addington et al., 2008; Amminger, Schafer, Papageorgiou, et al., 2012; Dickson, Calkins, Kohler, Hodgins, & Laurens, 2014; Kohler et al., 2014; Roddy et al., 2012; van Rijn, Aleman, et al., 2011; van Rijn, Schothorst, et al., 2011; Wolwer et al., 2012) to chronic schizophrenia (Hooker et al., 2011; Pinheiro et al., 2013; Pinheiro et al., 2014; Pinkham, Brensinger, Kohler, Gur, & Gur, 2011; Thaler et al., 2013) in several cross-sectional studies. However, longitudinal studies revealed that alterations in cognitive function were not progressive after illness onset (Keefe et al., 2006; Reichenberg et al., 2010), which may be a consequence of medication.

Progressive neurodegeneration leads to changes in gray matter and cortical thickness in regions relevant for the interaction of emotion and cognitive control in CHR-P, first-episode psychosis, and chronic schizophrenia patients (Feinberg, 1982; Gogtay et al., 2004; Vidal et al., 2006). These regions include prefrontal cortices, hippocampus, amygdala, basal ganglia, and thalamus (Pino et al., 2014). However, the interpretation of progressive functional and structural neurodegeneration with increased illness severity is limited by inconsistent results within PP. This may be due to symptoms' heterogeneity and medication effects. Most studies investigating schizophrenia and bipolar disorder with psychosis were conducted with patients on antipsychotic medication. As antipsychotics have been shown to profoundly impact both brain structure and function, it is difficult to determine whether progressive neurodegeneration is exclusively due to illness (Cahn et al., 2002; Cohen & Servan-Schreiber, 1992; Del-Ben et al., 2005; Harmer et al., 2006; Meltzer & McGurk, 1999; Pinkham, Gur, et al., 2007; Pinkham, Penn, Perkins, Graham, & Siegel, 2007; Pino et al., 2014; Sharma, 1999; Takahashi et al., 2005; Weinberger et al., 1988; Weiss et al., 2002; Weiss et al., 2003). However, recent studies reported structural (cortical thickness)

and functional (connectivity in fronto-parietal and cingulate-opercular networks) changes also in non-clinical schizotypy (De & Cummings, 2002; DeRosse et al., 2015; Nenadic et al., 2015; Wiebels, Waldie, Roberts, & Park, 2016) and first-episode unmedicated schizophrenia patients (Wang et al., 2019; Weissman, Perkins, & Woldorff, 2008). This suggests that neurodegenerative processes occur at an early stage of illness and manifest irrespective of medication. These findings support the notion of progressive deterioration of the neural circuits underlying the interaction between emotion and cognitive control along the psychosis continuum.

#### **4. Affect, emotion-cognitive control interaction and psychosis**

Understanding the influence of emotion on cognitive control is crucial as emotion affects how cognitive control is regulated and the adaptive interaction between them leads to appropriate behavior in real-life situations (Pessoa, 2008). Likewise, the role of arousal, valence, self-regulation, motivation, and reward processing on emotion-cognitive control interaction should also be considered as these variables (see table 1) may mediate the dynamic reciprocal interactions between the dorsal cognitive control and the ventral emotion processing systems in psychosis (Barch, 2005, 2008; Barch, Yodkovik, Sypher-Locke, & Hanewinkel, 2008; Clamor et al., 2014; Gard, Fisher, Garrett, Genevsky, & Vinogradov, 2009; Mueller, 2011; Pessoa, 2008; Roseman & Elliot, 2008; Roseman & Smith, 2001; Vanlessen, De Raedt, Koster, & Pourtois, 2016; Vanlessen, De Raedt, Mueller, Rossi, & Pourtois, 2015).

The existing evidence suggests that an optimal arousal level is required for adaptive behavior (Clamor et al., 2014). Both hyperarousal and hypoarousal alter the way one perceives and interacts with environmental stimuli (Clamor et al., 2014; Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002). Altered states of arousal can contribute to perceptual anomalies such as hearing of thoughts as voices or exacerbate paranoid or threat belief (Clamor et al., 2014). Increased levels of arousal can have a negative impact on emotion regulation (e.g., oversensitivity or increased responsiveness towards emotional stimuli) and, consequently, affect their interactions with cognitive control mechanisms. Accordingly, high arousal levels may trigger psychotic symptoms in PP (Docherty, St-Hilaire, Aakre, & Seghers, 2009; Myin-Germeys et al., 2003), CHR-P (Palmier-Claus, Dunn, & Lewis, 2012; Trotman et al., 2014) and non-clinical

individuals with psychotic-like experiences (Clamor et al., 2014). Exposure to stress leading to increased arousal was found to exacerbate psychotic symptoms in schizophrenia patients and in CHR-P individuals (Dinzeo, Cohen, Nienow, & Docherty, 2004, 2008; Docherty et al., 2009; Myin-Germeys et al., 2003; Palmier-Claus et al., 2012; Trotman et al., 2014), whereas hyperarousability was positively correlated to higher levels of psychotic-like experiences in non-clinical individuals (Clamor et al., 2014). Similarly, the ability to self-regulate goal-directed behavior amidst conflict and errors, referred to as self-regulation or effortful control, has been shown to influence both emotion processing and cognitive control (Hofmann, Schmeichel, & Baddeley, 2012; Kanske & Kotz, 2012b; Rothbart & Ahadi, 1994). Studies have shown negative correlations between positive and negative symptoms and self-regulation measures in schizophrenia (Santosh, Roy, & Kundu, 2015). However, the reviewed literature did not take into account the effect of measures such as self-regulation on emotion-cognitive control interaction in psychosis (table 2). As self-regulation is negatively affected by an increase in cognitive load, threat and reduced motivation (Hofmann et al., 2012; Li et al., 2018; Ward & Mann, 2000) and as psychosis is associated with amotivation and increased sensitivity towards negative emotional stimuli, we would expect self-regulatory abilities to influence the impact of emotion on cognitive control in psychosis. These studies indicate that personality traits such as hyperarousal (see table 1) and diminished self-regulation may specifically affect the interaction of emotion perception and attention and suggest that these factors may represent an index of psychosis vulnerability.

As mentioned in Section 3.1, PP are not only oversensitive to negative emotional stimuli but they also assign salience to neutral stimuli, which may contribute to the formation of positive symptoms such as hallucinations or delusions. On the contrary, under-sensitivity to positive emotional stimuli has been associated with alterations in motivation and reward processing, which may result in negative symptoms such as social withdrawal, apathy, and anhedonia (Abbas, Ramzan, Emad, & Rehman, 2019; Barch, 2005, 2008; Barch et al., 2008; Carra et al., 2019; Gard et al., 2009; Kapur & Mamo, 2003). Positive and negative symptoms tend to influence each other in a causative manner, where hallucinations can cause social withdrawal and lead to other negative symptoms and vice versa (Carra et al., 2019; Fowler et al., 2012; Messinger et

al., 2011; Millan, Fone, Steckler, & Horan, 2014; Wickham, Taylor, Shevlin, & Bentall, 2014).

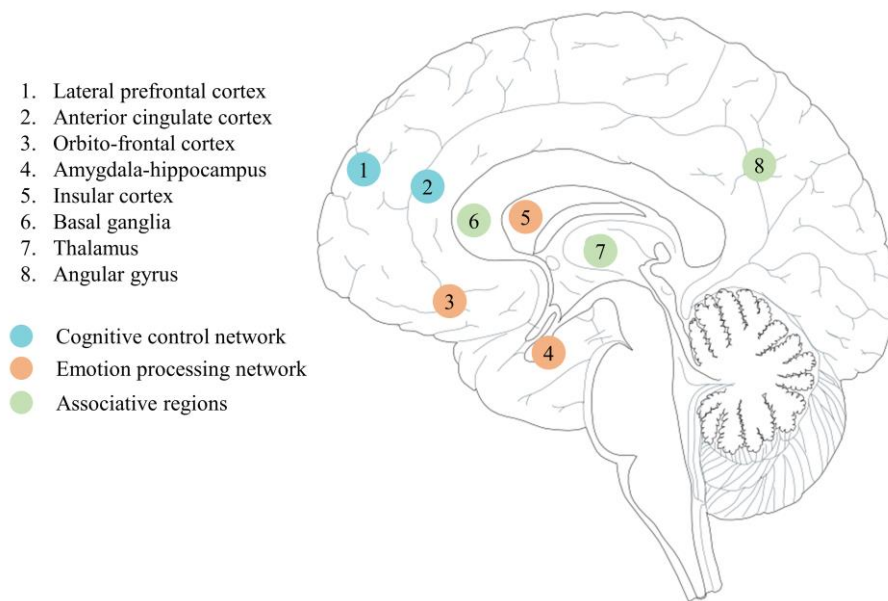
Altered neurochemical mechanisms associated with the dopamine neurotransmitter system have been shown to influence cognitive control and emotion processing in psychosis and an imbalance in dopamine is associated with psychosis pathology (Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001; Juckel et al., 2006; Kapur, 2004; Kapur, Arenovich, et al., 2005; Kapur, Mizrahi, & Li, 2005; McCutcheon, Abi-Dargham, & Howes, 2019; Ott & Nieder, 2019). Dopamine is primarily associated with detecting motivational salience in the environment and using it to form associations in a goal-directed fashion (Berridge & Robinson, 1998; Braver & Cohen, 2000; Schultz, 2002). Under normal physiological circumstances, healthy individuals show a balance between dopamine release in the mid-brain regions and the ability to understand context and to assign salience to environmental stimuli (Kapur, 2004; Kapur, Arenovich, et al., 2005). In psychosis, however, genetic or environmental predispositions lead to a dysregulation of dopamine release/firing (Kapur, 2004; Schulze, Schulze, Renneberg, Schmahl, & Niedtfeld, 2019). Dopamine dysregulation has been associated with the formation of delusions (“a way to advocate false beliefs”) and abnormal perceptual experiences such as hallucinations (“anomalous salience of internal representations of percepts, memories” (Kapur, 2004)). As dopaminergic systems (e.g., nucleus accumbens and ventral tegmental area) are extensively connected to both PFC and amygdala, alterations in dopaminergic transmission not only affect the ability of the amygdala to assign salience to a stimulus but also the PFC's ability to guide goal-directed behavior (Braver & Cohen, 2000; Ott & Nieder, 2019; Rosenfeld, Lieberman, & Jarskog, 2011). In the healthy population, increased dopamine firing is associated with successful cognitive control by the PFC, with activation of the striatum by positive affect (mood, motivation, and reward processes) and increased amygdala activation by negative stimuli (Ashby & Isen, 1999; Kienast et al., 2008; Kumakura et al., 2007; Ott & Nieder, 2019). However, antipsychotic medication blocks the effect of dopamine and, consequently, dampens the salience of preoccupying symptoms in psychosis. This may result in hyperactivity of the amygdala due to overprocessing of negative stimuli, in PFC hypoactivity due to failed attentional control, and altered basal ganglia activity resulting in low motivation and inadequate

processing of reward/positive stimuli (Berridge & Robinson, 1998; Juckel et al., 2006; McCutcheon et al., 2019). As there is a long prodromal phase prior to the onset of clinically relevant psychotic symptoms (McGlashan, Walsh, & Woods, 2010), it is likely that dopamine dysregulation also characterizes the prodrome phase manifested as pre-clinical psychotic symptoms. This is supported by studies reporting salience misattribution in CHR-P individuals (Abubaker et al., 2008; Amminger, Schafer, Klier, et al., 2012; Amminger, Schafer, Papageorgiou, et al., 2012; Dickson et al., 2014; Kohler et al., 2014; Roddy et al., 2012; van Rijn, Aleman, et al., 2011; van Rijn, Schothorst, et al., 2011). As dopamine seems to be involved in all key processes underpinning the interaction of emotion and cognitive control at the brain level (PFC, amygdala and striatum), it is likely that alterations in the mesocorticolimbic dopamine pathways play a critical role in the dysfunctional emotion-cognitive control interaction in psychosis (Mueller, 2011; Xu et al., 2019). However, this hypothesis warrants further investigation.

## **5. Neural underpinnings of emotion-cognitive control interaction in psychosis**

In line with the second aim of this review, this section focuses on the neurofunctional changes associated with the cognitive control and emotion processing systems in psychosis. Alterations in the dorsal cognitive control system and in the ventral emotion processing system have been repeatedly associated with alterations of emotion-cognitive control interaction in psychosis (table 2; (Alustiza, Radua, Pla, Martin, & Ortuno, 2017; Lin, Ding, & Zhang, 2018; Minzenberg et al., 2009; Modinos et al., 2010; Modinos et al., 2015; Mohanty et al., 2005; Ruocco et al., 2014)). However, it is unclear if such changes manifest in (i) the emotion processing neural network independent of cognitive control, (ii) the cognitive control system, or (iii) some intermediary neural circuitry leading to disruptions in emotion-cognitive control interaction along the psychosis continuum (figure 4).

**Figure 4:** Cortical and subcortical brain regions involved with emotion-cognitive control interactions (adapted from Kotz, Ravignani, & Fitch, 2018).



### *Impaired emotion processing?*

Alterations in emotion processing have long been recognized as a core feature of psychosis pathology. They include disturbances in emotion regulation, perception, recognition, and salience attribution in all sensory modalities (Hoekert, Kahn, Pijnenborg, & Aleman, 2007; Kohler, Walker, Martin, Healey, & Moberg, 2010; Kring & Elis, 2013; Taylor et al., 2012; Thaler et al., 2013; Wynn, Jahshan, Altshuler, Glahn, & Green, 2013; Wynn, Lee, Horan, & Green, 2008). Relative to healthy controls, both reduced (Comte et al., 2017; Diaz et al., 2011; Kim, Jeong, et al., 2015; Park et al., 2018; Townsend et al., 2013) and increased (Baarendse, Counotte, O'Donnell, & Vanderschuren, 2013; Bertocci et al., 2012; Brotman et al., 2014; Corbalan et al., 2015; Eack et al., 2016; Favre et al., 2015; Passarotti et al., 2011; Pauly et al., 2008; Pavuluri et al., 2008) activity in emotion processing regions such as the amygdala, orbitofrontal cortex, insula, thalamus, and hippocampus have been observed in PP. The discrepant findings may be attributed to either task demands (such as the type of cognitive control tasks, implicit or explicit processing of emotional stimuli during these tasks) or individual differences (such as symptom severity or differences in personality traits, including anxiety and arousal).

When emotional stimuli are processed implicitly in cognitive control tasks, participants detect and perceive the emotion but are required to ignore/disengage from them the cognitive control task. As PP are (over-) sensitive to (negative)

emotional stimuli as a function of symptom severity, the detection and perception of these stimuli would result in either intact or increased neural activity in the amygdala. In this case, the cognitive control system would be required to exert greater “attentional control” to allow the participant to disengage from the emotional stimuli in order to deliver appropriate task performance. This is supported by neuroimaging studies, in which emotional stimuli were presented implicitly in cognitive control tasks in PP (Dichter, Felder, et al., 2010; Favre et al., 2015; Pauly et al., 2008; Pavuluri et al., 2008). However, a few other studies reported decreased activity in the amygdala and in other emotion processing brain regions (Anticevic et al., 2011; Diaz et al., 2011; Kim, Jeong, et al., 2015). Decreased amygdala activity in PP may be a result of reduced motivation and increased negative symptoms. Indeed, PP presented higher negative symptom scores compared to positive symptoms in these studies (Anticevic et al., 2011; Diaz et al., 2011; Kim, Jeong, et al., 2015). This suggests that the ability to detect and perceive emotional stimuli at an early processing stage during the implicit presentation of emotional stimuli in emotion-cognitive control tasks depends on the severity of positive and negative symptoms in psychosis.

In the case of CHR-P individuals, the implicit presentation of emotional stimuli during a working memory task elicited increased activity in the frontal operculum (lateral PFC) in these participants as compared to healthy controls, in addition to increased activity in the thalamus and decreased activity in the basal ganglia and insula (Pauly et al., 2010). While an increase in PFC activity can be ascribed to facilitated performance, altered activity in the basal ganglia, insula, and thalamus can be ascribed to compensatory activity to maintain task performance in CHR-P individuals. Like PP, these individuals also scored high on negative symptoms. Non-clinical individuals with psychotic-like experiences also showed increased activity in the amygdala and basal ganglia, as well as decreased activity in the PFC in an emotional Stroop task (Mohanty et al., 2005). Although task performance was not significantly different from healthy controls, it significantly correlated with “anxiety apprehension and sensitivity” (Mohanty et al., 2005). This indicates that although they may be sensitive to emotional stimuli, they recruit additional brain regions to maintain task performance.

Reduced activation in emotion processing regions (e.g., amygdala, hippocampus, insula) was observed in PP when emotion was task-relevant and required explicit attention during cognitive control tasks (Comte et al., 2017; Park et al., 2018; Townsend et al., 2013). For example, during the variable attention and congruency task, emotional pictures of same or opposite valence were overlaid with each other and participants had to determine the emotion of the target or the background picture ((Comte et al., 2017) see explicit tasks in figure 2). Similarly, during a modified version of the Simon task, positive and negative emotional pictures were serially presented on the left or right side of the screen and participants were asked to indicate the emotion of the picture via a button press ((Park et al., 2018) see explicit tasks in figure 2). In these tasks, attention is either divided between two competing emotional pictures of opposite valence at the same time point or switched between these emotions continuously during the course of the task, which may overburden the emotion processing system resulting in reduced activation. Task performance (reaction time) was also significantly impaired in PP as compared to healthy individuals in these tasks (Comte et al., 2017; Park et al., 2018). However, reduced activity in emotion processing regions during the explicit presentation of emotional stimuli was not replicated (Brotman et al., 2014; Corbalan et al., 2015; Favre et al., 2015). We did not observe any such pattern in studies with non-clinical populations (table 2).

Overall, changes in emotion processing regions can be ascribed to either (i) oversensitivity towards negative emotional stimuli or (ii) severity of positive symptoms. However, most of the studies discussed above reported decreased activity or altered connectivity of the PFC with the amygdala and associated these changes with impaired task performance in psychosis. An open and relevant question therefore is whether impaired emotion processing occurs independent of cognitive control in psychosis.

#### *Dysfunctional context-sensitive attentional control?*

Successful conflict resolution requires the efficient use of attentional control on emotion regulation, attribution of salience to relevant stimuli, and inhibition of distracting emotional cues to maintain task performance. This is reflected in increased activation in the frontal-cingulate-parietal (cognitive control) network comprising the



anterior cingulate cortex, lateral PFC and inferior parietal gyrus and intact activation in emotion processing regions such as the amygdala (Alustiza et al., 2017; Egner et al., 2008; Minzenberg et al., 2009). In PP, hypoactivation in the cognitive control network is observed regardless of the task-relevance of emotional stimuli and is often paired with altered activity in emotion processing regions such as the amygdala, hippocampus, and insula (table 2). Similarly, CHR-P individuals and non-clinical individuals with psychotic-like symptoms show hyperactive amygdala and hypoactive prefrontal regions (Modinos et al., 2010; Mohanty et al., 2005; Pauly et al., 2010). Further, decreased connectivity between the cognitive control and the emotion processing networks has been observed along the psychosis continuum (Modinos et al., 2010; Park et al., 2018). These findings suggest that alterations in attentional control (hypoactive PFC) result in emotion dysregulation (hyperactive amygdala) in emotion-cognitive control tasks. As both emotion regulation and orienting/inhibiting attention require an intact prefrontal attentional control system, alterations in the emotion processing network engaged in tasks probing emotion-cognitive control interaction, may not be independent of cognitive control dysfunction in PP.

Another aspect of attentional control in cognitive control tasks is the ability to comprehend and actively maintain instructions required to execute a task (Cohen & Servan-Schreiber, 1992). Studies with PP have consistently documented difficulties in interpreting lexical ambiguities due to an inability to understand the context of a situation in language tasks (Baez et al., 2013; Cohen & Servan-Schreiber, 1992; Salzinger, 1971). PP also show difficulties in constructing and maintaining internal representations of context during information-processing and attention-related tasks (Park, Kim, Kim, Kim, & Lee, 2011; Rizzo, Danion, Van Der Linden, Grangé, & Rohmer, 1996; Schenkel, Spaulding, & Silverstein, 2005; Servan-Schreiber, Cohen, & Steingard, 1996; Uhlhaas, Phillips, Schenkel, & Silverstein, 2006; Waters et al., 2004). The concept of “internal representation of context” refers to the process of maintaining either task-instructions (e.g., during Flanker/Stroop tasks, in which participants have to respond to one aspect and ignore other aspects of the stimulus) or a particular stimulus (e.g., during n-back tasks, in which participants need to remember and update each stimulus so as to match it with the -nth preceding stimulus), or processing stimulus sequences (e.g., during language-related tasks, in which one needs to interpret the

meaning by processing a sequence of words) (Cohen & Servan-Schreiber, 1992). A failure to understand context leads to the selection of inappropriate responses in the respective task. Alternatively, PP may understand the context but either fail to maintain it over longer periods of time or fail to integrate the contextual information in the presence of emotional cues/distractors. Several frontal brain regions such as the medial PFC, ventro-medial PFC, inferior frontal, precentral gyrus, and orbito-frontal cortex, associated with difficulties in understanding, maintaining, and using an internal representation of context in emotion and non-emotion tasks in PP, are also a part of an interaction between emotion-cognitive control (Hoenig & Scheef, 2009; Kircher, Leube, Erb, Grodd, & Rapp, 2007; Rapp, Leube, Erb, Grodd, & Kircher, 2004; Smith, Henson, Dolan, & Rugg, 2004). Impairments in contextual processing are subtler in psychosis-prone non-clinical individuals. Task interference by emotional stimuli in emotional Stroop or n-back working memory tasks in CHR-P and psychosis-prone non-clinical individuals is higher than in healthy controls, even though not significantly different. These individuals often show an enhanced processing effort by recruiting additional brain regions (e.g., thalamus, basal ganglia, angular gyrus) in order to maintain task performance (Ettinger et al., 2014; Mohanty et al., 2005; Pauly et al., 2010).

Furthermore, neurochemical changes involving the PFC also support a dysfunctional attentional control hypothesis and the impact of emotion on cognitive control. As mentioned in Section 4, alterations in midbrain dopamine projections to several cortical and subcortical regions have been repeatedly associated with psychotic symptoms (McCutcheon et al., 2019). In addition, increased dopamine levels in the PFC and anterior cingulate cortex are thought to promote successful cognitive control and improve cognitive flexibility (Ashby & Isen, 1999; Braver & Cohen, 2000; Ott & Nieder, 2019). This suggests that a dopaminergic imbalance in the PFC could, in turn, down-regulate attentional control. With respect to emotion-cognitive control interaction, altered prefrontal activity could therefore impair context-sensitive attentional processing and the ability to assess the emotional significance of a stimulus.

More recent meta-analyses of fMRI studies, investigating various aspects of cognitive control (e.g., attentional control, inhibition, sustained and selective attention, working

memory), also showed reduced activation in this network in PP irrespective of the presence of emotional stimuli (table 2; (Alustiza et al., 2017; Mueller, 2011)). Reductions in cortical thickness in the PFC and cingulate cortex have also been observed in CHR-P (Gisselgard et al., 2018). The observation of functional and structural changes, particularly in prefrontal regions, indicates high replicability of cognitive control impairments in psychosis. Overall, the presence of impairment in the cognitive control network in tasks with or without emotional stimuli along the psychosis continuum indicates that dysfunctional context-sensitive attentional control may be inherent to psychosis pathology. Changes in cognitive control may reflect an increased vulnerability to psychosis and could thus serve as neuroimaging biomarkers of psychosis vulnerability (Falkenberg et al., 2015; Gisselgard et al., 2018).

#### *Alterations in the functioning of intermediary associative regions*

When emotion interacts with cognitive control, altered patterns of activation and connectivity within cognitive control and emotion processing networks have been observed in intermediary cortical and subcortical brain regions, such as the thalamus, basal ganglia, and angular gyrus. These regions are implicated in the integration of converging information in both clinical and non-clinical individuals with psychotic-like experiences (Bertocci et al., 2012; Brotman et al., 2014; Pauly et al., 2008; Takahashi et al., 2004). In the following paragraphs, we will discuss the role of these regions with respect to emotion-cognitive control interaction along the psychosis continuum.

The thalamus acts as a filter and integrates information from different sensory systems and relays it to higher cortical regions involved in emotional and cognitive processes (Pauly et al., 2008; Takahashi et al., 2004). Activity in this region increases with increased cognitive load, which may indicate greater effort and efficiency required to orchestrate increased amounts of sensory information (Pauly et al., 2008). Disruption in thalamic activity may result in the discoordination of the transmission of information between emotion processing and cognitive control regions. Neuroimaging studies have shown not only reduced functional activity but also decreased thalamic volume in schizophrenia patients (Kemether et al., 2003; Pinault,

2011). Structural and functional changes in the thalamus have been associated with attention and emotion impairments but also with positive symptoms in schizophrenia and bipolar disorder (Caetano et al., 2001; Chen, Ye, Jin, Zhu, & Wang, 2019; Gilbert et al., 2001; Gong et al., 2019; John, Zikopoulos, Bullock, & Barbas, 2018; Pergola, Selvaggi, Trizio, Bertolino, & Blasi, 2015; Yamamoto et al., 2018). These findings suggest that the ability of the thalamus to process sensory input may be reduced and lead to impaired perceptual processing particularly in situations of increased arousal, ultimately resulting in anomalous perceptual experiences such as hallucinations in schizophrenia (Behrendt, 2006; Pinault, 2011). Whereas reduced thalamus activity was observed in schizophrenia when emotion interacts with cognitive control, increased bilateral thalamus activity was observed in CHR-P individuals during an n-back working memory task with emotional stimuli (Pauly et al., 2008; Pauly et al., 2010). Similarly, a positive correlation was observed between thalamus activity and psychosis-prone personality factors in healthy individuals (Ettinger, Corr, Mofidi, Williams, & Kumari, 2013). The increase in thalamus activity in the prodromal phase of psychosis, which stands in contrast to the reduced activity in schizophrenia, points towards a compensatory role of thalamus to maintain task performance (Ettinger et al., 2014; Ettinger et al., 2015). These data support the concept of a psychosis continuum (Diederen, Daalman, et al., 2012; Ettinger et al., 2014; Ettinger et al., 2015).

The basal ganglia have close associations with sensorimotor, associative/cognitive, and limbic systems, and alterations in this region could be associated with psychiatric disorders such as psychosis (Macpherson & Hikida, 2019). This region has been implicated in tasks involving broadening the scope of thoughts and actions leading to positive experiences and exploratory behavior on the one hand, and developing strategies to deal with threat during aversive situations on the other hand (Fredrickson, 2001; Wager, Phan, Liberzon, & Taylor, 2003; Wager & Smith, 2003). Aberrant activity in sub-parts of the basal ganglia (e.g., striatum, putamen, caudate nucleus) has been reported in psychosis in tasks testing emotion-cognitive control interaction (Bertocci et al., 2012; Brotman et al., 2014; Mohanty et al., 2008; Pauly et al., 2010). Relative to healthy controls, decreased neural activity during negative and increased neural activity in positive emotion-cognitive control tasks were reported in the striatum and PFC in an emotional n-back task in schizophrenia (Eack et al., 2016).

Diminished striatal function in schizophrenia was proposed to reflect fronto-limbic disengagement so as to process fearful stimuli in moderation to maintain working memory task performance (Eack et al., 2016). On the other hand, increased functionality of the striatum and PFC during positive emotion-cognitive control interaction correlated with longer reaction times, suggesting effortful regulation of positive emotion (Eack et al., 2016). Reduced basal ganglia activity was also observed in CHR-P individuals whereas increased activity was seen in healthy individuals with high psychosis proneness in negative emotion-cognitive control interaction (Mohanty et al., 2005; Pauly et al., 2010). As task performance was not significantly different from healthy controls, alteration in basal ganglia activity can be attributed to compensatory emotion regulation to maintain task performance.

Further, cortical regions such as the angular gyrus, precuneus, and posterior cingulate have shown structural abnormalities, in addition to aberrant activity in emotion-cognitive control tasks in psychosis (Nierenberg et al., 2005; Niznikiewicz et al., 2000; Pauly et al., 2008; Thompson et al., 2001). Due to their location at the junction of temporal, parietal, and occipital cortex, these regions play a significant role in the integration of incoming information and channeling this information to frontal-cingulate regions (Pauly et al., 2008). Reduced activity in these regions in negative emotion-cognitive control tasks may also be ascribed to compensatory emotion regulation to maintain task performance. We did not find any studies that looked into these brain regions in non-clinical individuals with psychotic-like experiences in emotion-cognitive control tasks. Altered communication between these intermediary and associative brain regions may result in disruption of emotion-cognitive control coupling. As most studies focused on lateral PFC, anterior cingulate cortex and amygdala, the role of these intermediary regions in emotion-cognitive control interactions along the psychosis continuum warrants further investigation.

## **6. Summary and future directions**

Based on the reviewed studies, factors affecting the interaction between emotion and cognitive control in psychosis include the valence of emotional stimuli, severity of positive and negative symptoms, and individual differences (e.g., arousal). The early stages of psychosis experience may not be distinguishable from healthy individuals;

however, at later stages (CHR-P, first-episode schizophrenia, or chronic schizophrenia) a significant decline in emotion-cognitive control interaction is observed regarding neural activation and task performance (table 2). This decline can be ascribed to an altered interplay between emotion processing and context-sensitive attentional control mediated by intermediary associative regions (thalamus, basal ganglia, and angular gyrus). As a true course of this decline along the postulated psychosis continuum may be obscured by differences in methodological aspects, heterogeneity and severity of symptoms, individual differences in personality traits and medication, future efforts to understand the emotion-cognitive control interaction in psychosis should focus on longitudinal follow-up studies, taking into account these variables and using the same tasks and paradigms.

Further, impairments reflecting the impact of emotion on cognitive control have been described in many other neurological disorders and mental illnesses such as major depressive disorder, autism, borderline personality disorder, anxiety disorder, post-traumatic stress disorder, obsessive-compulsive disorder, and even Alzheimer's disease (Liao et al., 2012; Loveland, Bachevalier, Pearson, & Lane, 2008; Mayberg, 1997; Minzenberg, Fan, New, Tang, & Siever, 2007; Trzepacz et al., 2013). Similarities and differences in these impairments may be associated with subtle differences in illness-specific symptoms or concerns. Comparable to PP, patients suffering from major depression, post-traumatic stress disorder, borderline personality disorder, and anxiety disorders show a negative attentional bias in emotion-cognitive control tasks (aan het Rot, Hogenelst, & Schoevers, 2012; Minzenberg et al., 2007; Nica & Links, 2009; Santangelo et al., 2014). However, this bias is associated with specific illness-related negative emotional stimuli and not with general negative emotions (Joyal et al., 2019). For example, stimuli related to war and abuse caused greater performance impairment (slower responses) in post-traumatic stress disorder, whereas major depressive disorder patients were more affected by stimuli showcasing sadness and discouragement in an emotional Stroop task (Joyal et al., 2019). Similarly, illness-specific abnormalities were observed in neural activations of emotion processing and cognitive control systems in these disorders (Schulze et al., 2019). Borderline personality disorder and post-traumatic stress disorder patients showed hyperactivity in amygdala-hippocampus regions as compared to major

depressive disorder during the processing of negative emotional stimuli (Schulze et al., 2019). This is similar to PP with increased positive symptoms (e.g., hallucinations, (Dichter, Bellion, et al., 2010; Favre et al., 2015; Pacheco-Unguetti & Parmentier, 2016; Pauly et al., 2008; Pavuluri et al., 2008), who showed hyperactive amygdala as compared to PP with increased negative symptoms (e.g., apathy, anhedonia (Anticevic et al., 2011; Diaz et al., 2011; Kim, Yang, et al., 2015)). Diagnosis-specific differences were also observed in lateral PFC regions where both patients with borderline personality disorder and post-traumatic personality disorder showed hyperactive ventrolateral PFC, reflecting enhanced regulation of negatively affected emotion. However, reductions in dorsolateral PFC specifically in borderline personality disorder may be due to increased impulsivity and difficulties in attentional control of emotion (Joyal et al., 2019; Schulze et al., 2019). Taken together, while these studies highlight the significance of diagnosis-specific effects on the attentional control of emotion, relevant questions such as whether and to what extent different types of negative emotions recruit distinct neural systems in different disorders remain an open issue for future investigations. Future research should therefore dissociate the dysfunctions of emotion-cognitive control interaction considering differences in illness-specific etiology.

## **7. Conclusion**

The current review discussed and integrated fMRI evidence examining the influence of emotion on cognitive control along the psychosis continuum to identify alterations in emotion-cognitive control interaction and its connection to psychopathology. Specific processes and corresponding neuroanatomical correlates such as diminishing context-sensitive attentional control of prefrontal regions on the emotion processing network, and structural and functional alterations in subcortical and cortical associative brain regions have been identified as underlying changes affecting emotion-cognitive control interaction. However, open questions concerning the valence-specificity of these brain regions and the relationship between them remain to be further specified. A comprehensive understanding of the neural mechanisms underlying emotion-cognitive control interaction across the psychosis continuum is critical to achieve finer insights into the psychopathology of the illness. This would

further help develop therapies and tools focusing on improving attentional control and ultimately improving socio-cognitive functioning of people with psychosis in their everyday life.

**Declaration of competing interest**

Authors report no conflict of interest.

**Acknowledgment**

This work was funded by Portuguese Science and Technology Foundation (Fundação para a Ciência e a Tecnologia-FCT; grant number PTDC/MHC-PCN/0101/2014).







# **Chapter 4**

## **Hallucination proneness alters sensory feedback processing in self-voice production**

*Under review as:* Duggirala, S. X., Schwartze, M., Goller, L. K., Linden, D. E., Pinheiro, A., & Kotz, S. A. (2023). Hallucination proneness alters sensory feedback processing in self-voice production. bioRxiv, 2023-07. <https://doi.org/10.1101/2023.07.28.550971>

## **Abstract**

**Background:** Sensory suppression occurs when hearing one's self-generated voice, as opposed to passively listening to one's own voice. Quality changes of sensory feedback to the self-generated voice can increase attentional control. These changes affect the self-other voice distinction and might lead to hearing non-existent voices in the absence of an external source (i.e., auditory verbal hallucinations (AVH)). However, it is unclear how changes in sensory feedback processing and attention allocation interact and how this interaction might relate to hallucination proneness (HP).

**Study Design:** Participants varying in HP self-generated and passively listened to their voice that varied in emotional quality and certainty of recognition — 100% neutral, 60-40% neutral-angry, 50-50% neutral-angry, 40-60% neutral-angry, 100% angry, during EEG recordings.

**Study Results:** The N100 auditory evoked potential was more suppressed for the self-generated than externally generated voices. Increased HP was associated with (i) an increased N100 response to the self- compared to externally generated voices, (ii) a reduced N100 response for angry compared to neutral voices, and (iii) a reduced N200 response to unexpected voice quality in sensory feedback (60-40% neutral-angry) compared to neutral voices.

**Conclusions:** The current study highlights an association between increased HP and systematic changes of the emotional quality and certainty in sensory feedback processing (N100) and attentional control (N200) in self-voice production in a non-clinical population. Considering that voice hearers also display these changes, these findings support the continuum hypothesis. However, additional research is needed to validate this conclusion.

**Keywords:** N100-P200-N200, Launay Slade Hallucination Scale, Sensory suppression, Motor-auditory task, Attentional control

## 1. Introduction

Sensations arise inevitably and incessantly from various internal and external sources. As we can predict the sensory consequences of self-generated actions, we suppress these sensations. For example, we perceive the sound of our own footsteps as less intense than those of another person. Accordingly, self- and externally-generated events differ in how we respond and adjust to them in a dynamic environment. The internal forward model provides a mechanistic explanation for such “sensory suppression”(Blakemore, Rees, et al., 1998; Blakemore, Wolpert, & Frith, 1998; Wolpert & Miall, 1996). The model suggests that an internal copy of a motor plan (efference copy) is used to predict the sensory consequences of self-generated actions to prepare the brain for incoming sensory information. The perceived sensory feedback (reafference signal) is processed by comparison to this prediction, resulting either in a match or a mismatch (prediction error)(Wolpert & Kawato, 1998; Wolpert, Miall, & Kawato, 1998). Prediction errors, in turn, allow adaptation and updating of predictions to continuously optimize behavior.

In audition, these processes have been studied in voice production and perception. Neural activity in the auditory cortex (AC) is suppressed when we speak and hear our voice as compared to when we listen to someone else’s voice(Behroozmand, Karvelis, Liu, & Larson, 2009). This suppression is the result of comparing expected and actual sensory feedback to self-voice. Electrophysiologically, this phenomenon is captured by the N100 event-related potential (ERP) suppression effect, i.e., the difference in the N100 amplitude for self-generated and externally-generated voices during real-time talking(Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford, Mathalon, et al., 2001a) but also when self-voices are “self-generated” via a button-press(Pinheiro et al., 2018). Changes in the acoustic properties of the self-generated voice, for example, during a cold or vocal strain, can result in a mismatch between the expected and the actual sensory feedback to the self-voice. These mismatches reduce the N100 suppression effect and may lead to the allocation of additional attentional resources to sensory feedback processing and to the attribution of higher prominence to the self-generated voice(Hu et al., 2015; Lange, 2013; Schroger et al., 2015). This likely explains why empirical studies have reported both an increased N100 and N200

response in unexpected sensory feedback processing(Chen et al., 2011; Escera et al., 2000; Knolle et al., 2012; Knolle et al., 2013b; Melara, Varela, & Baidya, 2021). Unexpected changes in sensory feedback might evoke a surprise response (increased N100; (Lange, 2013; Schroger et al., 2015)) that, in turn, can increase error awareness and attentional control (increased N200; (Pinheiro, Schwartz, et al., 2019; Scheerer, Behich, Liu, & Jones, 2013a)).

### *Hallucination proneness and sensory suppression*

Auditory verbal hallucinations (AVH) can occur in healthy individuals with a prevalence of 6-13%,(Baumeister et al., 2017; Johns et al., 2014) implying a continuum of proneness ranging from no to infrequent or frequent AVH experiences(Badcock & Chhabra, 2013; van Os, 2003; van Os, Hanssen, Bijl, & Ravelli, 2000; van Os et al., 2009; van Os et al., 1999). Altered sensory feedback processing, resulting from insufficient monitoring or inaccurately attributing the self-generated voice to an external source, likely forms the core of AVH experiences(Allen, Aleman, & McGuire, 2007; Allen et al., 2004; Griffin & Fletcher, 2017; Heinks-Maldonado et al., 2007; Heinks-Maldonado et al., 2006; Nelson, Whitford, Lavoie, & Sass, 2014a, 2014b). These alterations were reported for voice hearers with a psychotic disorder and non-clinical voice hearers, suggesting that aberrant sensory feedback processing in self-voice production is a common feature associated with AVH, regardless of the clinical status of the participant(Allen, Aleman, et al., 2007; Allen, Amaro, et al., 2007; Allen, Freeman, Johns, & McGuire, 2006; Allen et al., 2004; Brookwell et al., 2013; Heinks-Maldonado et al., 2007; Jones & Fernyhough, 2007; Pinheiro, Farinha-Fernandes, Roberto, & Kotz, 2019; Stephan-Otto et al., 2023). For example, the N100 suppression effect is reduced in playback in participants with increased hallucination proneness (HP; (Pinheiro et al., 2018)), and it is reversed in real-time voice production tasks in persons with a psychotic disorder(Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford, Mathalon, et al., 2001a, 2001b; Ford, Roach, et al., 2007). While the underlying cognitive and neural mechanisms of AVH seem to somehow overlap in voice hearers with and without a psychotic disorder(Allen, Aleman, et al., 2007; Allen et al., 2004; Brebion et al., 2016; Diederer, van Lutterveld, & Sommer, 2012), differences pertain to the perceived emotional quality, appraisal, controllability, and related

distress(Daalman, Boks, et al., 2011; Johns et al., 2014). Unlike non-clinical voice hearers, voice hearers with a psychotic disorder often experience negative, derogatory, and life-threatening voices(Baumeister et al., 2017; Nayani & David, 1996; Waters, Allen, et al., 2012; Waters, Woodward, Allen, Aleman, & Sommer, 2012). This distinction in emotional voice quality and the potentially resulting distress are linked to deficits in identification and appraisal of vocal emotions in both voice hearers with (Alba-Ferrara et al., 2013; Rossell & Boundy, 2005; Shea et al., 2007) and without a psychotic disorder (Phillips & Seidman, 2008). Unlike non-clinical voice hearers, voice hearers with a psychotic disorder not only tend to ascribe more attention to negative emotions and perceive them more strongly but also misattribute negative meaning to neutral stimuli to maintain positive symptoms such as AVH (Amorim, Roberto, Kotz, & Pinheiro, 2022; Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001b; Kapur, 2003; Liddle et al., 2016). Misattributions of salience and the source of a self-generated stimulus in voice hearing were linked to aberrant predictive processing (Corlett et al., 2019; Davies et al., 2018; Nelson et al., 2014a, 2014b; Sterzer et al., 2018). Abnormally strong top-down predictions might generate attentional biases, causing an imbalance between expected and actual sensory input(Alderson-Day et al., 2017; Daalman, Verkooijen, Derks, Aleman, & Sommer, 2012; Powers et al., 2016; Vercammen & Aleman, 2010). This imbalance might lead to difficulties in perception, for example, the misattribution of negative meaning to neutral stimuli and perceiving meaningful information (e.g., speech) in noise (Alderson-Day et al., 2017; Barkus, Stirling, Hopkins, McKie, & Lewis, 2007a; Powers, Mathys, & Corlett, 2017; Vercammen & Aleman, 2010; Vercammen, de Haan, & Aleman, 2008), ultimately leading to false perceptions - AVH. Taken together, these findings emphasize the interdependence and mutual influence between alterations in sensory perception and predictive processing in voice hearers. Therefore, by manipulating emotional quality and thereby altering the perceptual certainty of recognizing one's own voice, one can probe both changes in sensory predictive processing as well as attention allocation in high HP persons, highlighting transitions along the HP spectrum.

#### *The current study and rationale*

Using a well-validated EEG motor-auditory task and building on own prior work (figure 1)(Pinheiro et al., 2018), the current study examined whether systematic changes in sensory feedback processing of the self-voice as a function of HP lead to altered sensory suppression (N100, P200) and attentional control (N200). The emotional quality of the self-voice was manipulated to change the level of certainty in sensory feedback processing (100% neutral, 60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry and 100% angry). For the self-voice that is most certain (100% neutral and 100% angry), we expected a reduction of the classical N100 suppression effect (self- < externally-generated) with higher HP(Pinheiro et al., 2018). For the uncertain self-voice (60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry), we expected a reversed N100 suppression effect (self- > externally-generated) with increasing levels of uncertainty regarding sensory feedback, in persons with low compared to high HP. Similar effects were expected for the P200 response that indicates the conscious detection of self-generated stimuli (Knolle et al., 2013a, 2013b; Knolle et al., 2019). Considering that the presumed alterations are linked to attentional control and error awareness, a reduced or reversed N200 suppression effect (self- > externally-generated) was expected for the certain compared to uncertain self-voice with higher HP.

## **2. Methods**

### *Participants*

Twenty-nine healthy adults (age range 18-27 years) were recruited. All participants were first invited for a voice recording, followed by the EEG session. Three participants did not participate in the EEG sessions due to time constraints, whereas one participant was excluded from further analysis due to technical issues during the EEG data collection. Therefore, the final participant number was 25 (21 females, mean age = 21.24, s.d. = 2.49 years; 21 right-, 3 left-handed, and 1 ambidextrous) varying in HP (Launay Slade Hallucinations Scale (LSHS)(Castiajo & Pinheiro, 2017; Larøi & Van der Linden, 2005a, 2005b; Launay & Slade, 1981) total scores: mean = 18.56, s.d. = 10.17, max = 42, min = 3; LSHS AVH scores [sum of items: “In the past, I have had the experience of hearing a person’s voice and then found no one was there”, “I often hear

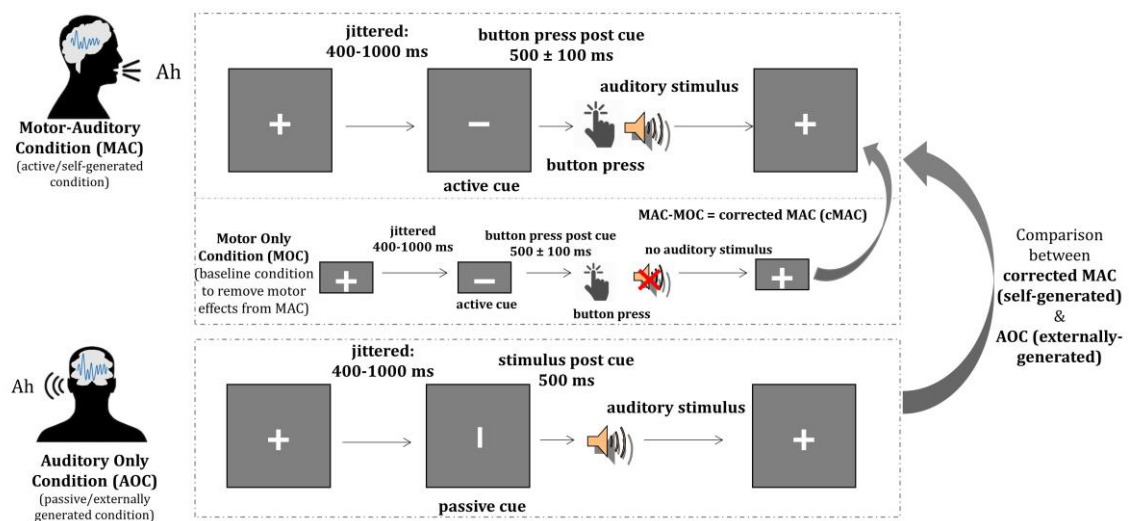


a voice speaking my thoughts aloud”, and “I have been troubled by voices in my head”]: mean = 2.40, s.d. = 2.62, min = 0, max = 11). All participants provided their written informed consent before the start of the study. They either received financial compensation (vouchers) or study credits for their participation. All participants self-reported normal or corrected-to-normal visual acuity and normal hearing. The study was approved by the Ethics Committee of the Faculty of Psychology and Neuroscience at Maastricht University and performed in accordance with the approved guidelines and the Declaration of Helsinki (ERCPN-176\_08\_02\_2017\_S2).

### *Procedure*

All participants underwent two study sessions conducted on separate visits. During the first voice recording session, “ah” and “oh” vocalizations from each participant were recorded and morphed (see section A of the supplementary document) to create final (100% neutral, 60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry and 100% angry) voice morphs for the EEG experiment. During the second session, EEG was recorded while the participants performed the auditory-motor task (figure 1; see section A of the supplementary document). The task was programmed and presented using the Presentation software (version 18.3; Neurobehavioral Systems, Inc.). Stimuli were presented via ear inserts. Button presses were recorded via the spacebar button on the keyboard. Participants were given an overview of the procedure and the principles of EEG at the start of the session. They sat comfortably in an electrically shielded soundproof chamber in front of a screen placed about 100 cm away. Participants filled in the LSHS questionnaire while the EEG cap was prepared.

**Figure 1:** Graphical representation of the Motor-auditory task. Abbreviations: MA = Motor Auditory Condition; AO = Auditory Only Condition; MO = Motor Only Condition. Motor activity from MA condition was removed by subtracting MO from MA to obtain MA corrected condition. Statistical analyses were performed with ERPs from MAc and AO conditions.



The paradigm was presented in a fully randomized event-related design over 12 runs. Each run consisted of 80 trials (40 AO, 40 MA, and 10 MO). Each trial started with a fixation cross, after which the presentation (vertical or horizontal) of a cue was jittered between 400-1000 ms. The cue was then followed by an auditory stimulus (after 500 ms for AO) or a button press that may (MA) or may not (MO) elicit an auditory stimulus. Five types of voice morphs consisting of “ah” and “oh” vocalizations, respectively, were presented in the AO and MA conditions. Thus, each run consisted of 4 trials of 10 stimulus types each (“ah” and “oh” for 5 voice morphs). This included 96 trials per voice morph (“ah” and “oh” combined, supplementary table 1). Participants were given short breaks after each run. To minimize potential influences of lateralized motor activity, participants were asked to switch their response hand every three runs. Prior to the experiment, participants were trained to press the button within  $500 \pm 100$  ms after the cue (horizontal bar) to align the presentation of auditory stimuli in the MA and AO conditions and to avoid overlap of cue-elicited and motor activation.

### Stimulus Rating

At the end of the EEG session, participants rated their voices for arousal and valence (supplementary figure 1). They additionally rated the voices on perceived ownness, meaning how much they identified their self-voice on a Likert scale (0-10). This was done to ensure that participants recognized their own voice and perceived the

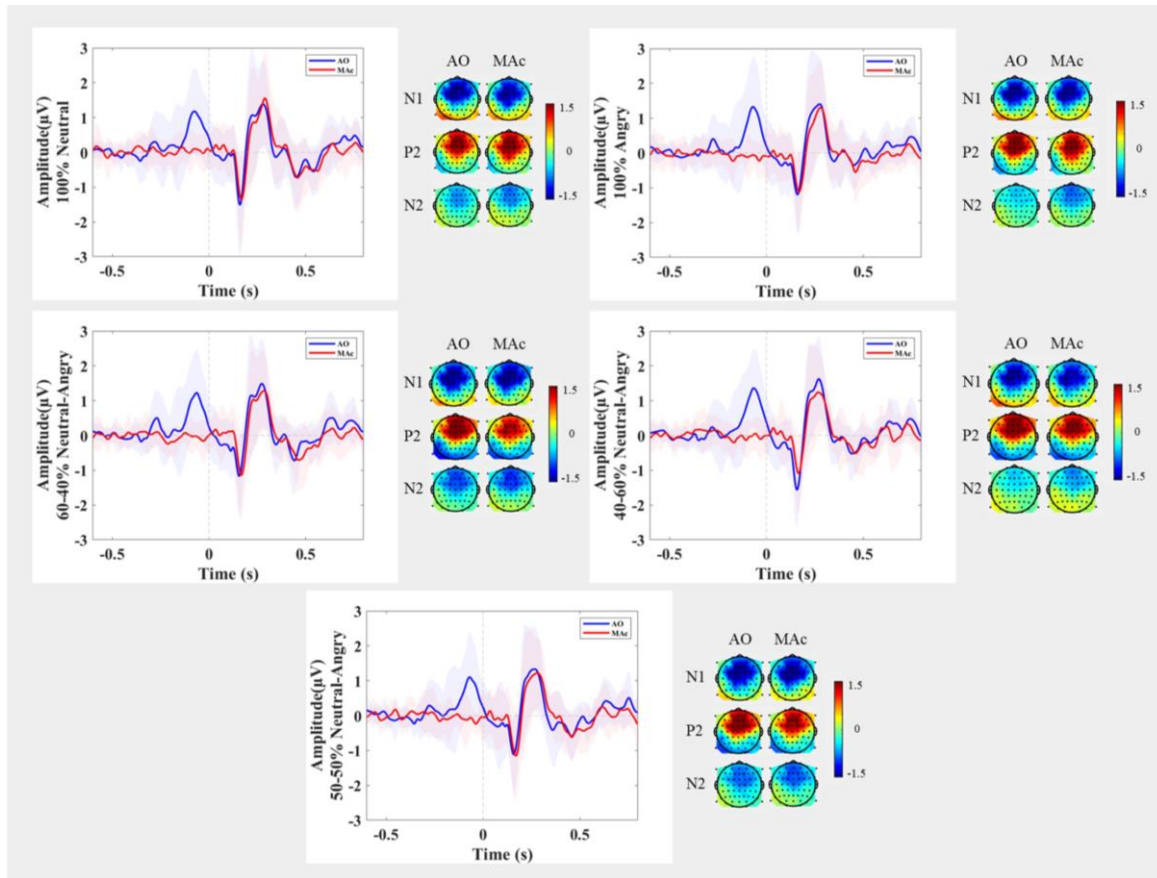
emotion expressed by it. Participants were debriefed after the experiment was finished.

### *EEG data acquisition and preprocessing*

EEG data were recorded with BrainVision Recorder (Brain Products, Munich, Germany) using an ActiChamp 128-channel active electrode set-up while participants performed the auditory-motor task. Data were acquired with a sampling frequency of 1000 Hz, an electrode impedance below 10 k $\Omega$ , using TP10 as online reference. During the EEG recording, participants were seated in a comfortable chair about 100 cm away from the screen in an acoustically and electrically shielded chamber.

EEG data were pre-processed (see section A the supplementary document) using the Letswave6 toolbox (<https://github.com/NOCIONS/letswave6>) running on MATLAB 2019a. The grand averaged waveforms revealed three ERP components, two negative components peaking at approximately 164 ms and 460 ms respectively and one positive component peaking at 286 ms. As the latencies of the ERP responses varied significantly (supplementary table 2), peak amplitudes as an outcome measure were chosen for data quantification. The N100 peak amplitude was defined as the largest negative peak occurring between 80-230 ms, the P200 peak amplitude was defined as the following positive peak between N100 and 380 ms, and the N200 peak amplitude as the negative peak between the P200 and 600 ms (Swink & Stuart, 2012a, 2012b). Previous research showed that the ERP components of interest all have prominent fronto-medial and fronto-central topographies (Behroozmand et al., 2009; Chen et al., 2012; Korzyukov, Karvelis, Behroozmand, & Larson, 2012). Therefore, the N100, P200, and N200 responses were extracted from the same fronto-central region of interest (ROI) that included 21 electrode locations: AFF1h, AFF2h, F1, Fz, F2, FFC3h, FFC1h, FFC2h, FFC4h, FC3, FC1, FCz, FC2, FC4, FCC3h, FCC1h, FCC2h, FCC4h, C1, Cz, C2 (figure 2).

**Figure 2:** Grand average ERP waveforms  $\pm$  standard error of mean and topographic maps showing voltage distribution at the peak ERPs, comparing self-generated and externally-generated voices for the five self-voice types originating from a fronto-central ROI. Abbreviations: MAc = Motor Auditory Corrected; AO = Auditory Only.



### Statistical analyses

Statistical analyses on N100, P200, and N200 data were performed in R version 4.2.2 (2022-10-31) Copyright (C) 2022, using linear mixed modeling with lmer and lmerTest packages (Bates, 2016; Kuznetsova, Brockhoff, & Christensen, 2017). We used linear mixed modeling to control for the random effects of participants influencing the outcome measure. Additionally, since HP measured by the LSHS is a continuous variable, Linear mixed modeling was considered more appropriate than classical ANOVA to analyze the impact of HP on sensory feedback (condition) and voice quality (stimulus type). Amplitude values of the ERPs (N100/P200/N200) were used as outcome measures, while participants were used as random effects, and condition (2 levels: MAc and AO), stimulus type (5 levels: 100% neutral, 60-40% neutral-angry, 50-50% neutral-angry, 40-60% neutral-angry, 100% angry) and LSHS total or LSHS

AVH scores (continuous variable) were included as fixed effects in the models. For all models, the Gaussian distribution of model residuals and quantile-quantile plots confirmed their respective adequacy.

### 3. Results

We followed a hypothesis-driven approach to probe changes in voice quality (stimulus type) and sensory prediction (condition) as a function of HP.

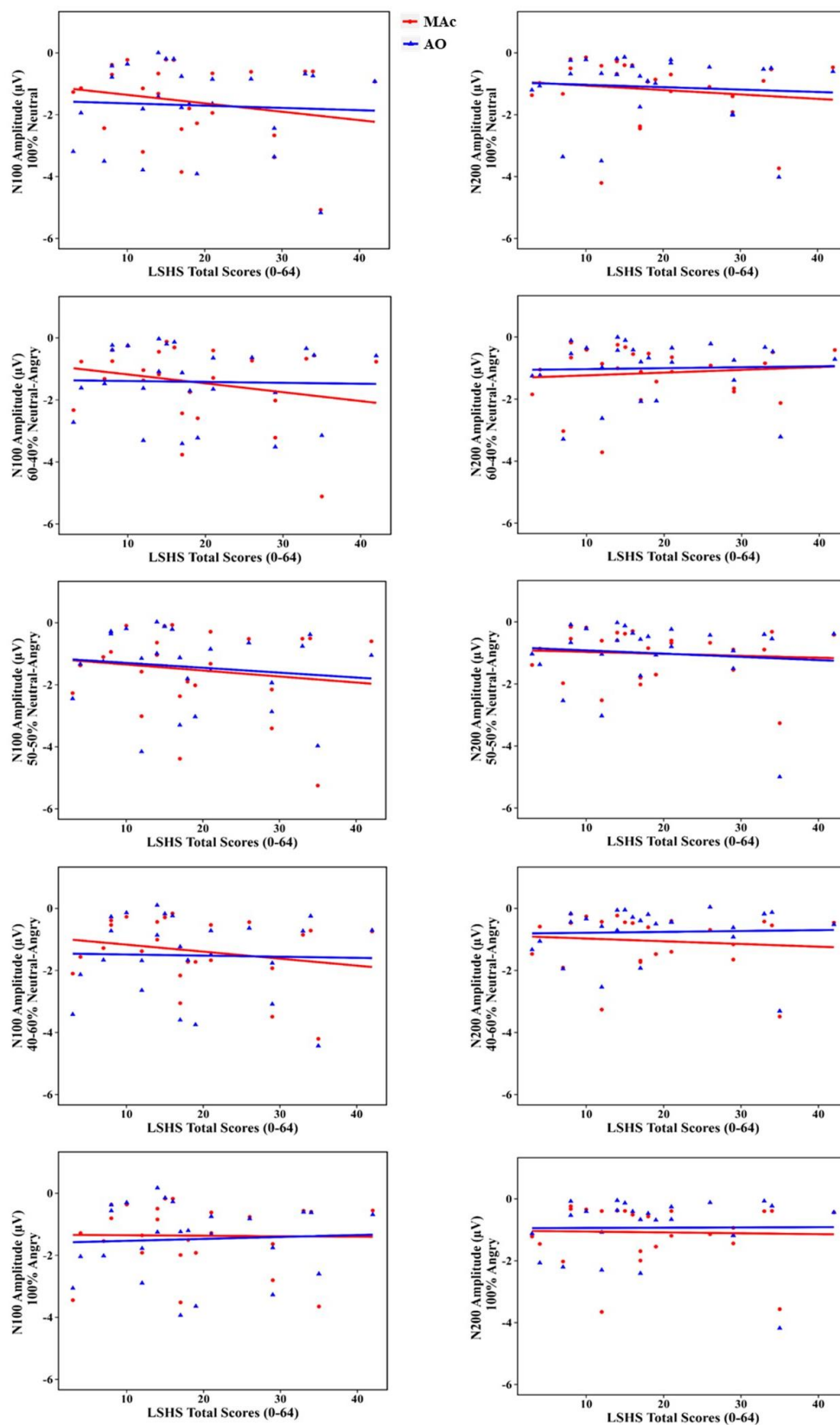
*N100*: To probe the influence of HP (based on LSHS total scores) on condition and stimulus type, we tested the model [m1\_N100 <- lmer(N100 ~ + Condition \* LSHS total + Stimulus Type \* LSHS total + (1|ID), data=data, REML = FALSE)] against the null model [m0\_N100], which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 24.072$ ,  $p = 0.01243^*$ ; AIC = 432.93; table 1, figure 3). We thus replicated the N100 sensory suppression effect where externally generated (AO) voices lead to a larger (more negative) N100 response than self-generated (MAc) voices. We also observed an overall decrease (less negative) in the N100 response independent of condition (AO or MAc) with increased HP (LSHS total scores) for the angry compared to neutral voice (table 1, figure 3).

**Table 1:** Linear mixed effects model for the N100 including the effect of HP based on LSHS total scores. Abbreviations: SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 225.0$  (except Intercept:  $df = 29.03$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-1.153e+00	4.832e-01	-2.386	0.02379 *
AO	-3.381e-01	1.162e-01	-2.909	0.00399 **
LSHS total	-2.489e-02	2.283e-02	-1.090	0.28461
60N	1.943e-01	1.838e-01	1.057	0.29148
50N	1.766e-01	1.838e-01	0.961	0.33747
40N	1.258e-01	1.838e-01	0.685	0.49427
Angry	-1.445e-01	1.838e-01	-0.786	0.43247

AO*LSHS total	1.519e-02	5.491e-03	2.767	0.00614 **
60N*LSHS total	1.545e-03	8.682e-03	0.178	0.85895
50N*LSHS total	-2.256e-04	8.682e-03	-0.026	0.97929
40N*LSHS total	4.225e-03	8.682e-03	0.487	0.62698
Angry*LSHS total	1.962e-02	8.682e-03	2.260	0.02481 *
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	1.2318	1.1099	
Residual		0.1951	0.4417	
Number of observations: 250, Subjects: 25				

**Figure 3:** Scatter plots depicting N100 and N200 modulations as a function of HP based on LSHS total scores, for each stimulus type. The N100 response for the self-generated voice increased (more negative) with an increase in HP (table 1). The N200 response decreased with an increase in HP for the most uncertain self-voice, regardless of the conditions (table 2).



*P200*: Analysis of the P200 followed the same procedure as for the N100. However, the results indicated that HP (based on LSHS total or AVH scores) did not significantly affect sensory prediction (condition) or voice quality (stimulus type) (see section B of the supplementary document).

*N200*: The model that showed the best goodness of fit [ $m1\_N200 \leftarrow lmer(N200 \sim + \text{Condition} * \text{LSHS total} + \text{Stimulus Type} * \text{LSHS total} + (1|ID), \text{data}=\text{data}, \text{REML} = \text{FALSE})$ ] also yielded a significant difference ( $\chi^2(11) = 27.44, p = 0.003941^{**}$ ; AIC = 323.15; table 2, figure 3) when compared against the null model [ $m0\_N200$ ; AIC = 328.59]. The N200 for the self-generated (60N) self-voice compared to neutral self-voice decreased (less negative) with an increase in HP (LSHS total scores).

**Table 2:** Linear mixed effects model for the N200, including the effect of HP based on LSHS total scores. Abbreviations: SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 225.0$  (except Intercept:  $df = 29.2785$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-0.980059	0.378787	-2.587	0.0149 *
AO	0.092415	0.093585	0.988	0.3245
LSHS total	-0.012202	0.017896	-0.682	0.5007
60N	-0.260779	0.147970	-1.762	0.0794 .
50N	0.075303	0.147970	0.509	0.6113
40N	0.085665	0.147970	0.579	0.5632
Angry	-0.052273	0.147970	-0.353	0.7242
AO*LSHS total	.002340	0.004421	0.529	0.5971
60N*LSHS total	0.017018	0.006991	2.434	0.0157 *
50N*LSHS total	0.002683	0.006991	0.384	0.7015
40N*LSHS total	0.008063	0.006991	1.153	0.2500
Angry*LSHS total	0.010041	0.006991	1.436	0.1523
<b>Groups</b>	<b>Name</b>	<b>Variance</b>	<b>SD</b>	



Random Effects				
Subjects	Intercept	0.7530	0.8678	
Residual		0.1265	0.3557	
Number of observations: 250, Subjects: 25				

#### 4. Discussion

This EEG study investigated how changes in sensory feedback processing of the self-voice link to HP and might engage attentional resources by manipulating the emotional quality of the self-voice, thereby altering the certainty of recognizing one's own voice. The data analyses focused on the N100, P200, and N200 ERP components elicited by the self- and externally-generated [certain (100% neutral, 100% angry) and uncertain (60-40% neutral-angry, 50-50% neutral-angry, 40-60% neutral-angry)] self-voice (figure 1). The results replicated previous findings (Knolle et al., 2019; Pinheiro et al., 2018), confirming an N100 suppression effect when comparing sensory feedback processing for the self- and externally-generated voice (table 1). Critically, this N100 suppression effect was reduced in high HP (based on both LSHS total and AVH scores), confirming a link between HP and altered sensory feedback processing (figure 3). Moreover, regardless of condition, high HP (based on LSHS total scores) was associated with reduced attention allocation indicated by a reduced N100 response to angry compared to neutral voice and lower error awareness demonstrated in a reduced N200 response to the uncertain (60-40% neutral-angry morph) compared to neutral voice (table 2, figure 3). However, HP did not modulate the P200 responses. Overall, these results confirm that HP influences sensory feedback processing, and it suggests that attention allocation for the self-generated voice varies with HP in a group of healthy individuals.

##### *Sensory feedback processing and attention allocation as a function of HP*

Replication of the classical N100 sensory suppression effect (Bass et al., 2008; Knolle et al., 2012; Knolle et al., 2013a, 2013b; Knolle et al., 2019; Pinheiro et al., 2018) (table 1) likely indicates that the auditory cortex is prepared for the sensory consequences of the self-generated voice. However, increased HP was associated with an increased

N100 response for the self-generated voice (figure 3), thus reducing the N100 suppression effect. This may indicate altered sensory feedback processing for the self-generated voice as well as increased attentional resource allocation towards sensory feedback processing in high HP individuals. One may consider that this alteration and the need for additional resources stem from a less efficient comparison of expected and actual sensory input and the resultant error signal, which might lead to hyper-accentuation of the self-voice. This perspective is supported by previous studies with voice hearers with (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford, Mathalon, et al., 2001b; Ford et al., 2013) and without psychotic disorder (Pinheiro et al., 2018) using similar paradigms. Altered responses to the self-generated voice might indicate that subtle changes in self-monitoring might already be present in healthy persons with high HP.

Furthermore, regardless of condition (AO or MAc), the N100 response to the angry compared to neutral self-voice was reduced in high HP participants (table 1), likely indicating differences in their response when the emotional quality of their voice becomes (fully) negative. Prior research indicates that high HP persons tend to show a dampened negative emotion perception, based on their ability to control attentional bias towards negative cues (Amorim et al., 2022). Therefore, the current results may point to a link between high HP and reduced appraisal of and inhibition of attention allocation to negative emotions in a non-clinical sample.

Contrary to expectations, HP did not modulate the P200 in sensory feedback processing of the self-voice. The N100 and P200 have been linked to different effects when attributing a sensory event to one's own action. Whereas the N100 suppression effect seems to reflect the outcome of the comparison of expected and actual sensory input, the P200 was associated with the more conscious realization that a finger tap elicited a related auditory stimulus (Knolle et al., 2012; Knolle et al., 2013a, 2013b). The present task, which involved the pseudo-random interweaving of conditions (MA, AO, MO) and stimuli (5 types of "ah" and "oh" vocalizations each), may have precluded sufficient opportunity for the P200 to engage in conscious retrospective processing of a button-press eliciting the self-voice.

The N200 was reduced for the 60-40% neutral-angry compared to the 100% neutral self-voice in high HP individuals regardless of the condition (table 2, figure 3). Prior pilot data showed that anger expressed in “ah” vocalizations was already recognized in the initial morphing steps, i.e., the 70-30% neutral-anger voice on the neutral-angry continuum. It is therefore possible that the 60-40% neutral-angry self-voice, among the five presented voice types marks a distinct shift from perceiving something as neutral to detecting anger in the voice imbuing the perception of an uncertain voice. Consequently, this specific self-generated voice may have yielded the most equivocal outcome regarding perceptual uncertainty of the self-voice. Functionally, the N200 has been linked to error awareness, attentional control, and conscious processing of perceptual novelty (Folstein & Van Petten, 2008; Folstein, Van Petten, & Rose, 2008). Thus, the reduced N200 to this uncertain self-voice in high HP individuals might suggest an altered response to unexpected change or error awareness. Additionally, the N200 has been linked to heightened emotional reactivity to negative rather than neutral stimuli (Gardener, Carr, MacGregor, & Felmingham, 2013). Taken together, the reduced N200 in high HP individuals may thus indicate down-regulation of negative emotional reactivity, reduced error awareness, and processing of an uncertain self-voice.

Although the N100 suppression effect was observed for the self-generated voice (table 1), there was no significant interaction between condition (AO, MAc) and stimulus type (five types of self-voice). This suggests that the self-voice manipulations were still within the acceptable range of feasible acoustic changes and therefore, we did not find differential suppression effects for the different types of self-voices (supplementary figure 1). Further, the lack of this interaction in the N100 could be the result of stimulus type probability (2:3 for certain: uncertain). Previous studies showed that higher probability and stimulus repetition result in a stimulus-specific memory trace reflected in early auditory processing as a pronounced N100 suppression (Baldeweg, 2006; Costa-Faidella, Baldeweg, Grimm, & Escera, 2011; Friston, 2005a). Taken together, the unexpected self-voices might not have induced sufficiently different perceptions either because they were presented more frequently, or because they did not differ sufficiently in their acoustic profile. Consequently, there was no difference in the N100 suppression effect among self-voices.

Some specifics of the task design should be noted. Unlike the classical ERP suppression paradigm, where different conditions are presented in a blocked design (Knolle et al., 2012; Knolle et al., 2013a, 2013b; Pinheiro et al., 2018), here all conditions and stimuli were presented in a fully event-related design. Due to the mixing of conditions, a cue was introduced to indicate whether the participant was required to press a button to generate a self-voice or to passively listen to the self-voice (figure 1). While this cue was removed from the MA by subtracting the MO condition for the final analysis, it remained present in the AO condition resulting in a pre-stimulus positive potential (figure 2). Next to the presence of the cue, the duration between the cue and the auditory stimulus was constant (500 ms). Both factors caused the participants to pay close attention and made them anticipate the onset of the voice in the externally generated condition. However, even though the temporal delay was similar in the self- and externally-generated conditions, we observe a significant N100 suppression effect ( $AO > MAc$ , table 1). This could be attributed to a confluence of factors. Studies have reported that it is not the motor-action per se, but the voluntary intention, involving motor planning, to self-generate an action (e.g., a voice) that leads to sensory suppression (Jack et al., 2021; Timm, SanMiguel, Keil, Schröger, & Schönwiesner, 2014). Further, the increased N100 response in the cued listening condition (AO), excluding motor planning, could be attributed to explicit attention allocation to a self-relevant stimulus (e.g., self-voice) (Hillyard, 1981; Horváth & Winkler, 2010; Nobre, 2001; Slagter, Van Wouwe, et al., 2016; Slagter, Prinssen, Reteig, & Mazaheri, 2016). Together, the performance of a motor action in the self-generated condition may take away attention from listening to the generated stimulus, which differs from a cued listening condition (Baess, Horvath, Jacobsen, & Schroger, 2011; Griffiths, Jack, Pearson, Elijah, Mifsud, Han, Libesman, Barreiros, et al., 2023). These factors together may influence how attentional resources are directed towards diverse sensory input and to the different N100 responses to the self- versus externally-generated voice.

Taken together, the current results link increased HP to changes in sensory feedback processing and attentional engagement to the self-voice in a healthy participant group. Specifically, these findings suggest that the processing of sensory consequences of one's own actions are attenuated, however, this attenuation decreases with an increase in HP. Further, high HP is associated with reduced attention allocation to the

angry compared to neutral voice, demonstrating their ability to effectively manage negative content (Amorim et al., 2022). The current findings thus support the continuity perspective regarding changes in sensory feedback processing and attention allocation previously reported in voice hearers (Ford, Mathalon, Heinks, et al., 2001; Ford, Mathalon, et al., 2001b; Ford et al., 2013; Ford, Roach, Faustman, & Mathalon, 2008; Ford et al., 2010). Nevertheless, to strengthen this concept, further investigations involving participants across the psychosis continuum, including healthy persons who do not hear voices, voice-hearers with and without psychotic disorders, are warranted.

**Acknowledgements:** The authors would like to thank Joseph Johnson for sharing the paradigm code that was adapted and used in the current experiment. We would also like to thank Alexandra Emmendorfer for sharing her scripts that were modified and used for EEG preprocessing as well as for engaging in valuable discussions on data analysis.

**Funding:** The current study was supported by BIAL Foundation (BIAL 238/16).

**Data availability:** The data that support the findings of this study are available from the corresponding author upon reasonable request.

**Author Contributions:** SXD, MS, DL, AP, SK conceptualized and designed the experiment, SXD prepared materials, collected and analyzed the data, and wrote the first draft of the manuscript, SXD, MS, LG, DL, AP, SK refined the manuscript. AP, MS, SK procured funding for the project. All authors have approved the final version of the manuscript.

**Competing Interests:** The authors declare that they have no competing interests.

## **Supplementary Document**

### **SECTION A: Methods**

#### **Stimulus generation**

##### *Voice recording*

Participants comfortably sat inside an acoustically and electrically shielded chamber with the recording equipment, while the researcher sat outside this chamber. Recordings were made using a Rode NTKb microphone powered by a Rode NTK microphone power supply (<http://www.ode.com/microphones/ntk>) and processed with the Praat software (<https://www.praat.org>). Participants were instructed to repeatedly vocalize “ah” and “oh” in a neutral (no emotion) and in an angry voice. Vowels were chosen to eliminate semantic content (Cook & Wilding, 2001; Schweinberger, Herholz, & Sommer, 1997; Ventura, Freitas, & Tavares, 2009). Participants were asked to vocalize the vowels for 500 ms, and were provided with examples to familiarize them with the target duration of the vocalization. This duration was chosen to properly capture the emotionality while maintaining self-voice recognition. The best voice samples were selected once the participants confirmed that they recognized their recorded voice, that the anger intensity was the highest that they could produce, that they perceived no emotion in the neutral recording, and if the vowels were pronounced clearly. Background noise was eliminated from the recordings using Audacity software (<https://audacityteam.org/>) and a Praat script was applied to normalize the intensity to 70 dB. The duration of the final neutral and angry “ah” and “oh” vocalizations for each participant was 500 ms.

##### *Morphing*

To create voice samples with varying degrees of emotional content, the pre-recorded neutral and angry self-voices for each individual participant were parametrically morphed to create neutral-to-angry and angry-to-neutral continua. These continua consisted of 11 stimuli with a 10% stepwise increase (neutral-to-angry)/decrease (angry-to-neutral) in emotionality along the continuum (see supplementary table 1). Morphing was performed using the TANDEM-STRAIGHT software (Kawahara, 2006;

Kawahara & Irino, 2005; Kawahara et al., 2008) running on MATLAB (R2019a, v9.6.0.1072779, The MathWorks, Inc., Natick, MA). For the final EEG experiments, 100% neutral, 60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry and 100% angry voice morphs were selected. The intermediate voice morphs were selected based on pilot data that revealed that the maximum uncertainty to differentiate a neutral from an angry self-voice fell in the range of 35-65% morphing. The increase in emotional voice quality (as self-voice stepwise changes from fully neutral to fully angry) and manipulations of uncertainty (most certain: 100% neutral and angry; uncertain: 60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry self-voice morphs) would probe both changes in sensory feedback to the self-voice and attention allocation resulting from these changes.

#### *Auditory-motor task*

A variant of an established button-press task was employed to investigate differences between self- and externally-generated auditory stimuli (Pinheiro et al., 2018) (figure 1). This task comprises three conditions: a motor-auditory condition (MAC), where participants pressed a button to generate their pre-recorded voice; an auditory-only condition (AOC), where participants passively listened to their pre-recorded voice; and a motor-only condition (MOC), where they pressed a button but did not hear their voice. This latter condition was used to control for motor activity resulting from the button-press in the MA condition (MAC-MOC = corrected MAC [cMAC]). Previous studies have consistently shown that there is a reduction in the N100 amplitude in response to self-generated sound via a button-press compared to passively listening to the same sound (Baess, Widmann, Roye, Schroger, & Jacobsen, 2009; Hughes et al., 2013), indicating that button-presses can be used as a motor-act to self-generate a stimulus (for voices see Knolle et al., 2019).

#### **EEG data preprocessing**

Data were first cleaned to remove false button presses (e.g., trials with button presses during AO), downsampled to 500 Hz, and then bandpass filtered (1-30 Hz). All channels were re-referenced to the average of the mastoid electrodes. Eye blinks and movements and noisy electrodes were removed using an independent component

analysis (ICA) with the runica algorithm in combination with Rajan and Rayner (PICA) as implemented in Letswave6 (<https://github.com/NOCIONS/letswave6>). ICs representing noise were removed for each participant based on the IC time course and topography. The resulting data were segmented with a pre-stimulus time window of -600 to 800 ms, time-locked to the onset of the auditory stimulus. The segmented data were baseline corrected to a window of -600 to -400 ms relative to the onset of the respective auditory stimulus. This remote baseline window was selected due to a cue-related ERP modulation before the onset of the auditory stimulus in AO, which could not be removed using high-pass filtering. After baseline correction, an automatic artifact rejection algorithm was applied with an amplitude criterion of  $\pm 65\mu\text{V}$  to remove epochs/trials with remaining artifacts. The resulting data were then averaged for each participant and each condition.



## SECTION B: Table and table legends

**Supplementary table 1:** Neutral-Angry continua with 11 voice morphs.

a) Neutral-to-angry

Emotion /Morphs	1*	2	3	4	5*	6*	7*	8	9	10	11*
Neutral	<b>100 %</b>	90 %	80 %	70 %	<b>60 %</b>	<b>50 %</b>	<b>40 %</b>	30 %	20 %	10 %	<b>0%</b>
Angry	<b>0%</b>	10 %	20 %	30 %	<b>40 %</b>	<b>50 %</b>	<b>60 %</b>	70 %	80 %	90 %	<b>100 %</b>

b) Angry-to-neutral

Emotion /Morphs	1*	2	3	4	5*	6*	7*	8	9	10	11*
Angry	<b>100 %</b>	90 %	80 %	70 %	<b>60 %</b>	<b>50 %</b>	<b>40 %</b>	30 %	20 %	10 %	<b>0%</b>
Neutral	<b>0%</b>	10 %	20 %	30 %	<b>40 %</b>	<b>50 %</b>	<b>60 %</b>	70 %	80 %	90 %	<b>100 %</b>

c) Final Stimuli for Ah and Oh vocalizations.

100% Neutral = Ah (a1 + b11) + Oh (a1 + b11)
60-40% Neutral-Angry = Ah (a5 + b7) + Oh (a5 + b7)
50-50% Neutral-Angry = Ah (a6 + b6) + Oh (a6 + b6)
40-60% Neutral-Angry = Ah (a7 + b5) + Oh (a7 + b5)
100% Angry = Ah (a11 + b1) + Oh (a11 + b1)

Note: a1 refers to the specific voice morph from table a, voice morph 1.

**Supplementary table 2:** Latency range of N100, P200 and N200 amplitudes.

	Min (ms)	Max (ms)
N100	0.08	0.23
P200	0.20	0.38
N200	0.25	0.6

The influence of proneness to auditory verbal hallucinations on condition and stimulus type was tested based on LSHS AVH scores. The respective model [m2\_N100 <- lmer (N100 ~ + Condition \* LSHS AVH + Stimulus Type + (1|ID), data=data, REML = FALSE)] showed the best goodness of fit and yielded a significant difference ( $\chi^2(7) = 14.071$ ,  $p = 0.04993^*$ ; AIC = 434.94) compared to the null model [m0\_N100] (supplementary table 3, and figure 3). A more negative N100 response was observed for external compared to self-generated voices. The N100 response decreased (i.e., it was less negative) in response to externally-generated compared to self-generated voices with increased HP (LSHS AVH scores). Further, compared to neutral voices, other voices lead to a decreased (less negative) N100 response in the self-generated condition.

**Supplementary table 3:** Linear mixed effects model for the N100 response including the effect of HP based on LSHS AVH scores. Abbreviations: SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects: df = 225.0 (except Intercept: df = 27.632).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-1.47223	0.09030	-4.722	6.11e-05 ***
AO	-0.1700	0.07741	-2.196	0.0291 *
LSHS AVH	-0.05934	0.08622	-0.688	0.4974
60N	0.22298	0.09030	2.469	0.0143 *
50N	0.17245	0.09030	1.910	0.0574
40N	0.20424	0.09030	2.262	0.0247 *

Angry	0.21959	0.09030	2.432	0.0158 *
AO*LSHS AVH	0.04744	0.02177	2.179	0.0304 *
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	(Intercept)	1.2377	1.1125	
Residual		0.2038	0.4515	
Number of observations: 250, Subjects: 25				

**Supplementary table 4:** Model comparisons with P200 response as output and HP, Condition and Stimulus. Abbreviations: SE = standard error; SD = standard deviation; NP = number of parameters; AIC = Akaike information criterion; BIC = Bayesian information criterion; Chisq = chi square; Df = degree of freedom; M1 = Condition \* Stimulus type \* LSHS total/AVH; M2 = Condition \* LSHS total/AVH + Stimulus type \* LSHS total/AVH; M3 = Condition \* LSHS total + Stimulus type; \*p < 0.05; \*\*p < 0.01; \*\*\*p < 0.001.

Models with LSHS total								
Model	NP	AIC	BIC	Log Lik	Deviance	Chisq	Df	Pr(>Chisq)
Null model	3	382.48	393.05	-188.24	376.48			
M1	22	405.55	483.03	-180.78	361.55	14.931	19	0.727
M2	14	391.02	440.32	-181.51	363.02	13.468	11	0.2638
M3	10	385.52	420.73	-182.76	365.52	10.966	7	0.1401
Models with LSHS AVH								
Model	NP	AIC	BIC	Log Lik	Deviance	Chisq	Df	Pr(>Chisq)
M1	22	403.75	481.23	-179.88	359.75	16.729	19	0.6082
M2	14	388.50	437.80	-180.25	360.50	15.98	11	0.1419

M3	10	384.58	419.79	-182.29	364.58	11.904	7	0.1038
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The influence of HP was tested based on LSHS AVH. The model [m2\_N200 <- lmer (N200 ~ + Condition \* LSHS AVH + Stimulus Type\*LSHS AVH + (1|ID), data=data, REML = FALSE)] also yielded a significant difference ( $\chi^2(11) = 22.32$ ,  $p = 0.022^*$ ; AIC = 328.28) from the null model ([m0\_N200]; AIC = 328.59) (supplementary table 5, and figure 6).

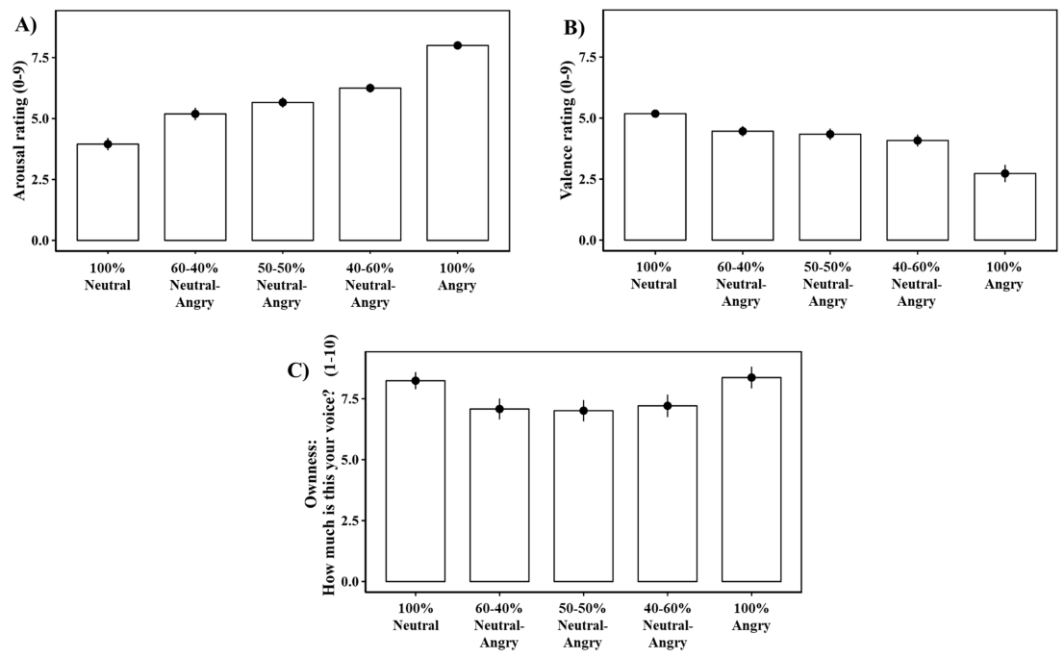
**Supplementary table 5:** Linear mixed effects model for the N200, including the effect of HP based on LSHS AVH scores. Abbreviations: SE = standard error; SD = standard deviation;  $^*p < 0.05$ ;  $^{**}p < 0.01$ ;  $^{***}p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 225.0$  (except Intercept:  $df = 29.39$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-1.236426	0.246880	-5.008	2.4e-05 ***
AO	0.148456	0.061680	2.407	0.0169 *
LSHS AVH	0.012458	0.069441	0.179	0.8589
60N	-0.016236	0.097525	-0.166	0.8679
50N	0.132913	0.097525	1.363	0.1743
40N	0.238609	0.097525	2.447	0.0152 *
Angry	0.095991	0.097525	0.984	0.3260
AO*LSHS AVH	-0.005252	0.017349	-0.303	0.7624
60N*LSHS AVH	0.029712	0.027431	1.083	0.2799
50N*LSHS AVH	-0.003256	0.027431	-0.119	0.9056
40N*LSHS AVH	-0.001369	0.027431	-0.050	0.9602
Angry*LSHS AVH	0.015870	0.027431	0.579	0.5635
Groups	Name	Variance	SD	
<b>Random Effects</b>				

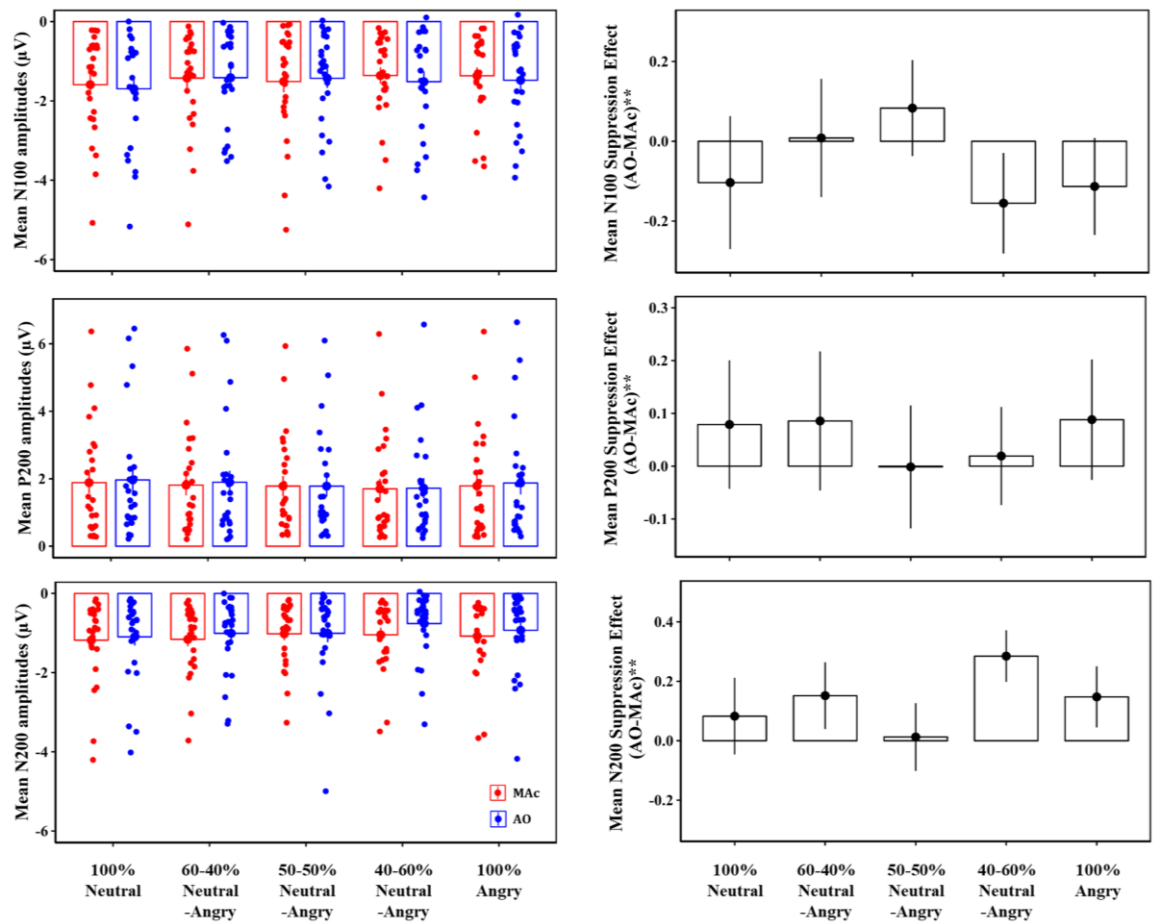
Subjects	(Intercept)	0.7517	0.8670	
Residual		0.1294	0.3598	
Number of observations: 250, Subjects: 25				

**SECTION C: Figure and figure legends**

**Supplementary figure 1:** Post experiment stimuli rating. A) Arousal rating on a scale of 0-9 for each voice stimulus. B) Valence rating on a scale of 0-9 for each voice stimulus. C) Ownness rating on a scale of 1-10 for each voice stimulus. Vertical bars represent standard error of mean.

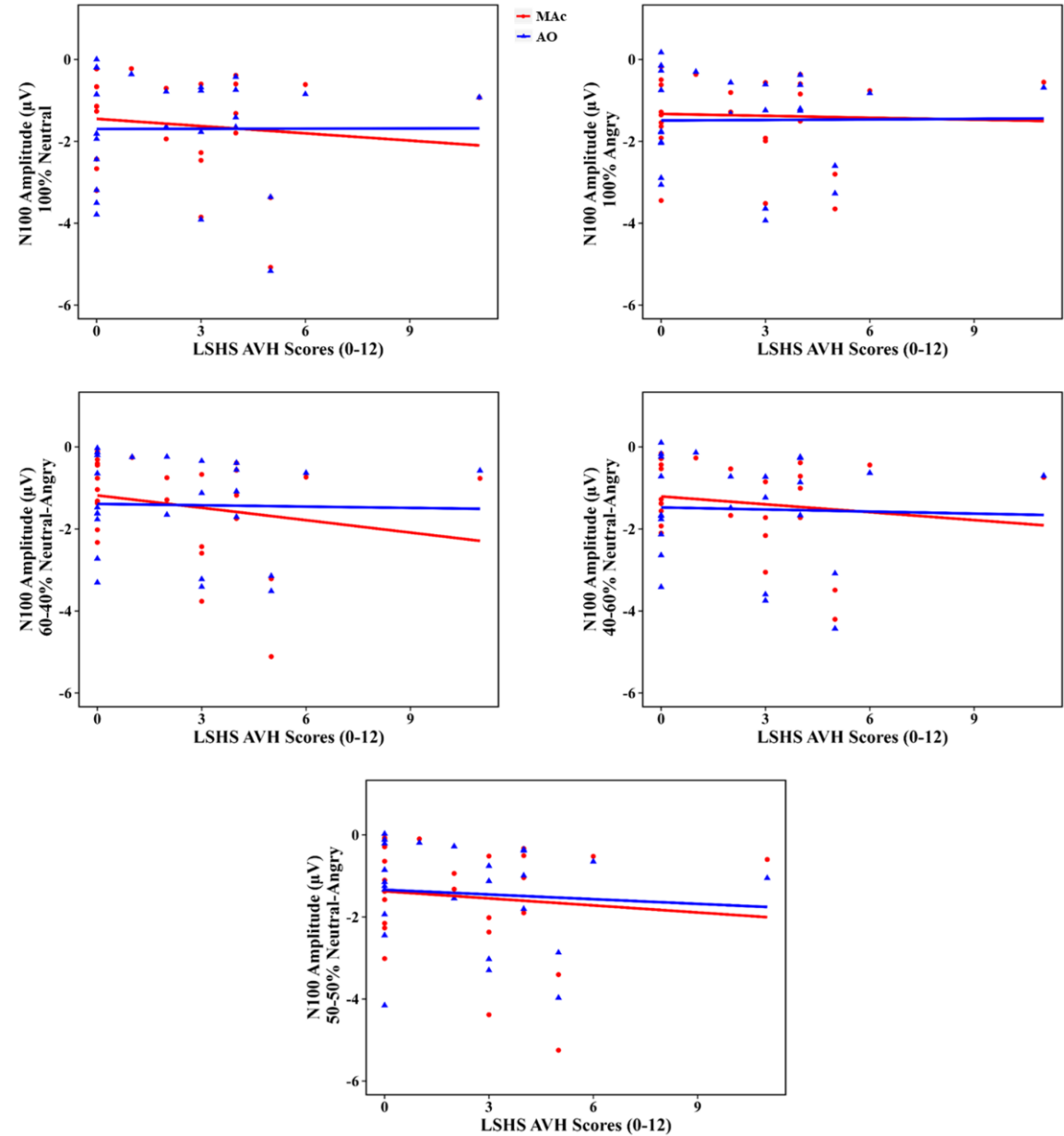


**Supplementary figure 2:** Mean ERP amplitudes for MAc and AO, and suppression effects (AO - MAc) per voice stimulus type. Note: Negative N100 suppression values depict AO > MAc whereas positive N100 suppression values depict MAc > AO. Vertical bars represent standard error of mean.



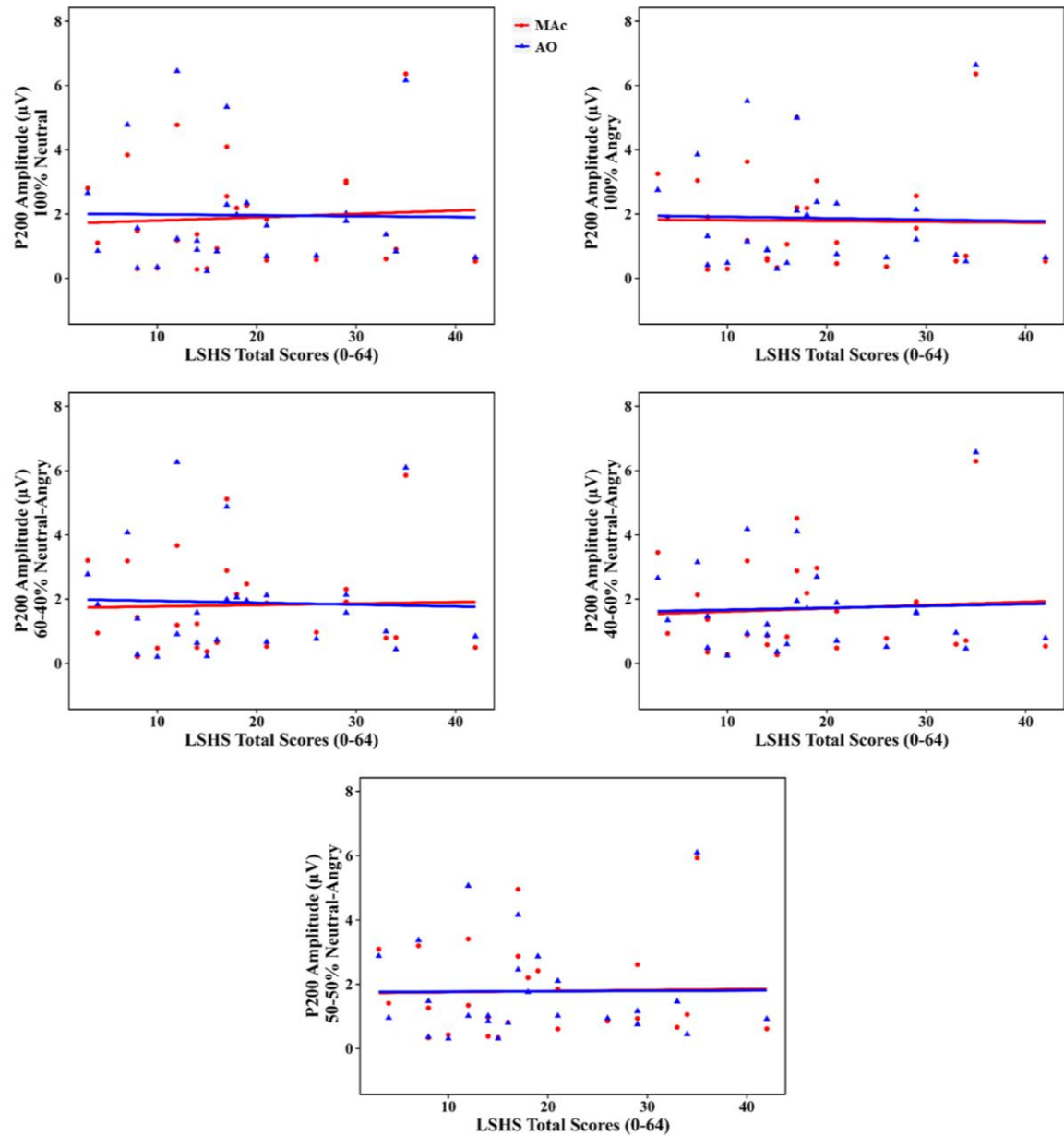
\*\*Negative N1 suppression values depict AO > MAc whereas positive N1 suppression values depict MAc > AO

**Supplementary figure 3:** Scatter plots depicting N100 modulation as a function of HP based on LSHS AVH scores for each stimulus type. Increase (more negative) in N100 response for self-generated voice with increase in HP scores (supplementary table 3).

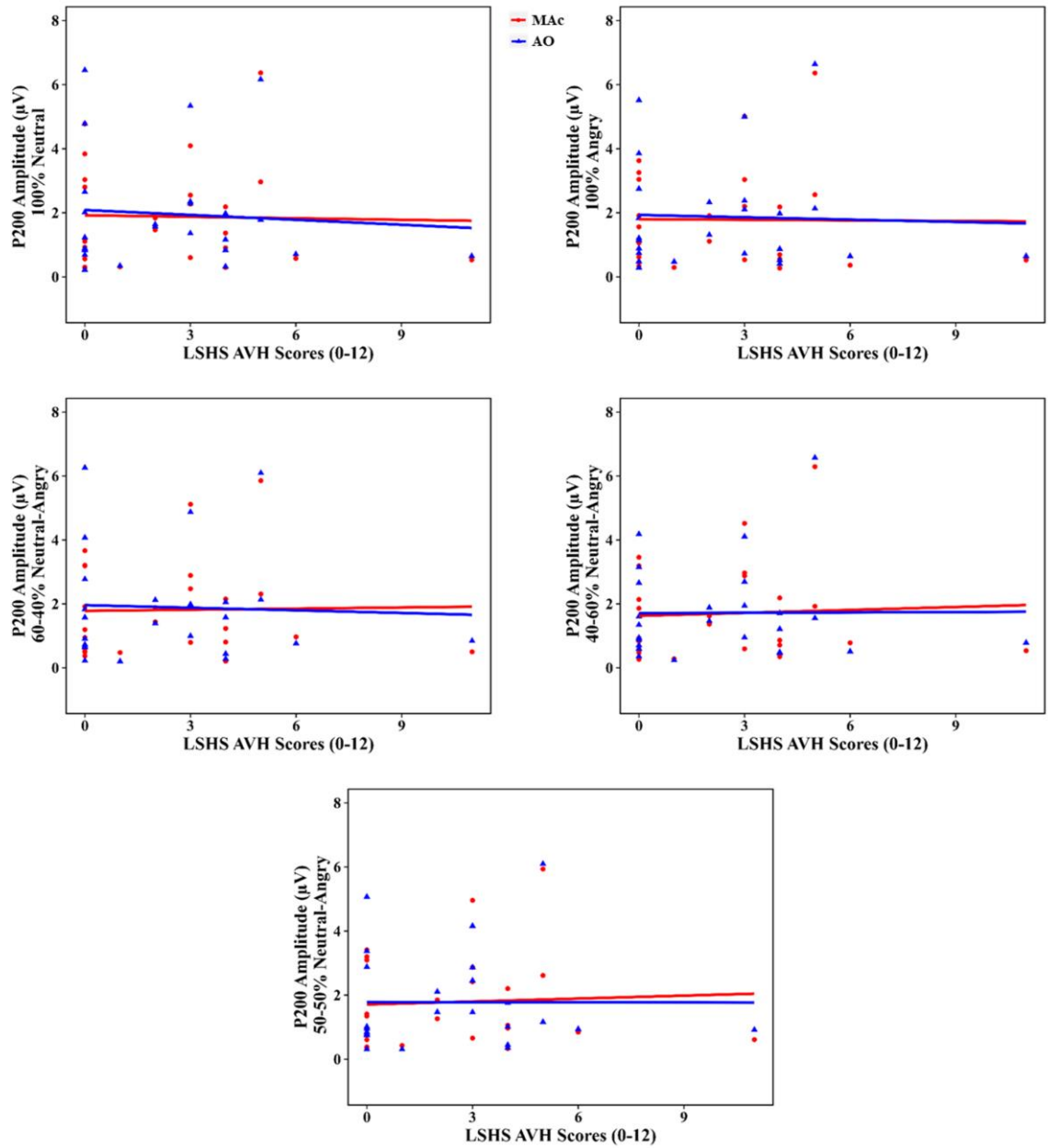




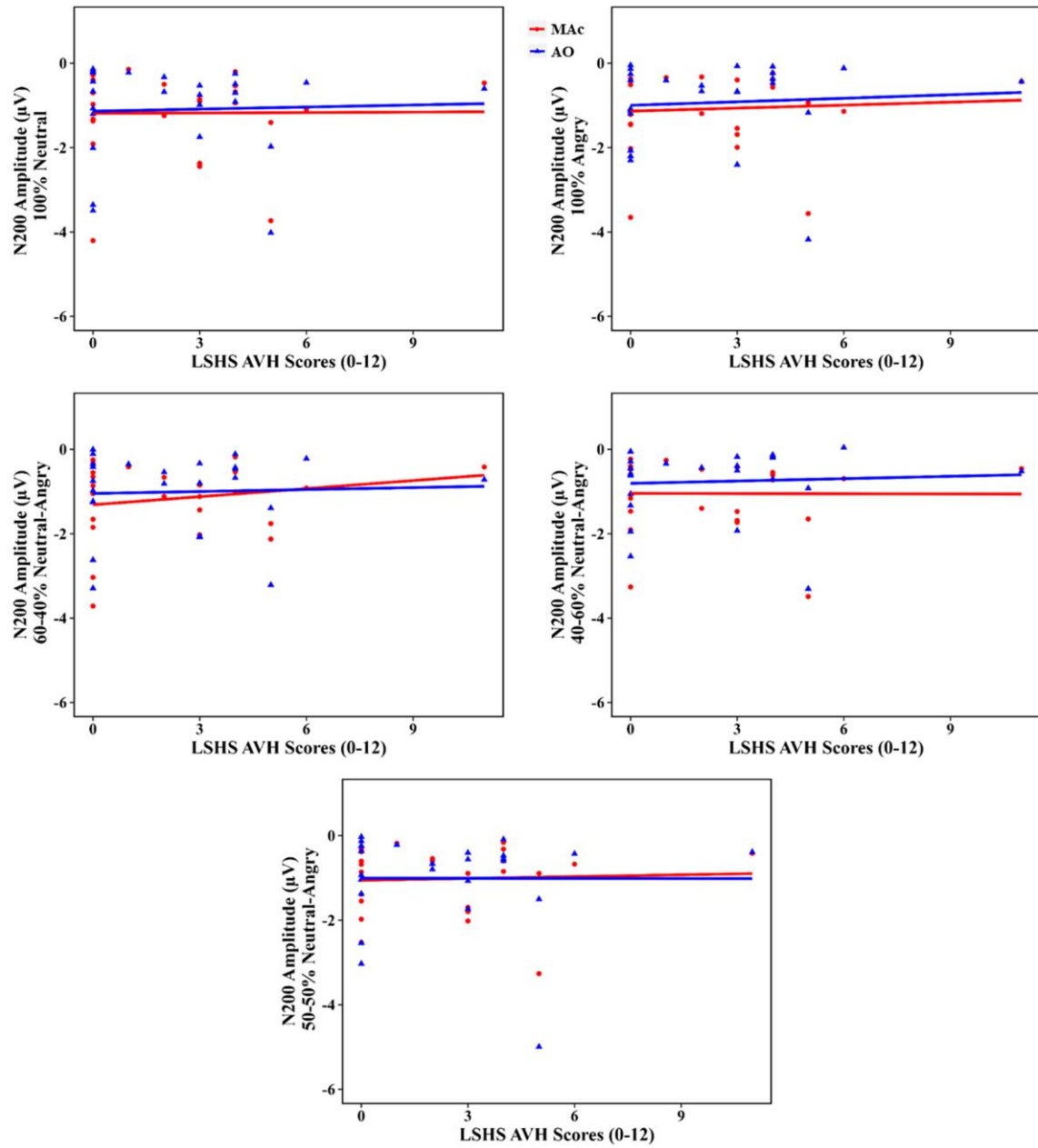
**Supplementary figure 4:** Scatter plots depicting the change in P200 response as a function of HP (based on LSHS total scores) for each stimulus type. No effect of condition (AO, MAc), stimulus type or HP on P200 responses (supplementary table 4).



**Supplementary figure 5:** Scatter plots depicting the change in P200 response as a function of HP based on LSHS AVH scores for each stimulus type. No effect of condition (AO, MAc), stimulus type or HP on P200 responses (supplementary table 4).



**Supplementary figure 6:** Scatter plots depicting the change in N200 responses as a function of HP based on LSHS AVH scores for each stimulus type (supplementary table 5).





# **Chapter 5**

## **Not enough pleasure? Influence of hallucination proneness on sensory feedback processing of positive self-voice**

*To be submitted as:* Duggirala, S. X., Schwartz, M., Goller, L. K., Linden, D. E., Pinheiro, A., & Kotz, S. A. (2023). Not enough pleasure? Influence of hallucination proneness on sensory feedback processing of position self-voice.

## **Abstract**

Ample research explored changes in sensory feedback processing of the self-voice as well as the control of attention allocation in voice hearers, including both non-clinical voice hearers and voice hearers with a psychotic disorder. While the attentional bias toward negative emotional information in voice hearers with a psychotic disorder due to heightened sensitivity towards threat/danger is well established, it remains unclear how positive emotion captures or controls attention. Manipulating the certainty of sensory feedback to the self-voice, transitioning from fully neutral to entirely positive (100% neutral, 60-40% neutral-pleasure, 50-50% neutral-pleasure, 40-60% neutral-pleasure, 100% pleasure), provides an opportunity to investigate attentional control and sensory feedback processing in positive self-voice as a function of hallucination proneness (HP). Participants with different HP scores self-generated and passively listened to their own voices during EEG recordings. N100 or P200 responses to self-generated and externally-generated self-voices did not differ. Further, HP did not modulate N100/P200 responses. These null findings might result from the minimal perceptual discriminability among the five types of voices varying in pleasure content. This might have led to less variation in certainty regarding the sensory feedback to self-voice, and consequently a lack of differential engagement of attentional resources. The lack of a global N100 suppression effect prompts inquiry into the association of sense of ownership/agency and pleasure.

**Keywords:** N100-P200-N200, motor-auditory task, pleasure, certainty, hallucination proneness

## **1. Introduction**

Although typically associated with a clinical diagnosis of psychiatric (e.g., schizophrenia, bipolar disorder, or major depressive disorder) or neurological (e.g., Alzheimer's disease, Parkinson's disease) disorder, auditory verbal hallucinations (AVH) are also present in healthy individuals not in need of clinical care (Daalman, Boks, et al., 2011; Johns et al., 2002; Johns et al., 2014; Larøi, 2012; Maijer, Begemann, Palmen, Leucht, & Sommer, 2018; van Os, 2003). The differences between psychotic and non-clinical AVH pertain to their emotional valence, controllability, and related distress (Daalman, Boks, et al., 2011; Johns et al., 2002; Johns et al., 2014; Larøi, 2012; Maijer et al., 2018). A growing body of evidence suggests that the neural underpinnings (Allen, Aleman, et al., 2007; Barkus et al., 2007a; Brebion et al., 2016; Diederen, Daalman, et al., 2012) and the experience of AVH in terms of characteristics such as sound location, loudness, and source are similar in psychotic and non-clinical voice hearers (Daalman, Boks, et al., 2011; Johns et al., 2002; Johns et al., 2014; Larøi, 2012; Maijer et al., 2018). This evidence and the prevalence rates of AVH may reflect a continuum of susceptibility in the general population ranging from low to high hallucination proneness (HP; (Baumeister et al., 2017; Johns et al., 2002; van Os et al., 2009).

One of the theories embedded in the 'forward model' framework that accounts for AVH relates to self-monitoring and inner-speech (Feinberg, 1978; Frith et al., 1992; Frith et al., 2000; Jones & Fernyhough, 2007). According to this theory, alterations in self-monitoring may lead to the attribution of one's own actions to an outside agent. This means that if AVH are a form of inner speech, voice hearers may fail to recognize them as self-generated. The forward model suggests that self- compared to externally-generated actions and their sensory consequences lead to a suppression of neural activity as individuals can fully predict the sensory consequences of their own actions (Blakemore et al., 2000; Frith et al., 2000; Wolpert et al., 1995). Any disruption of this internal 'self-monitoring' system may result in the misattribution of internally-generated speech to an external source, and consequently lead to the manifestation of experiences such as AVH. Electroencephalographic (EEG) studies in voice hearers provide support for this conception (Ford & Mathalon, 2004; Ford, Mathalon, Heinks,

et al., 2001; Ford, Mathalon, et al., 2001a, 2001b; Ford et al., 2013; Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018). Voice hearers with a psychotic disorder and non-clinical voice hearers both showed altered processing of self-generated voices, as indicated by an increased N100 event-related potential (ERP) response to self- compared to externally-generated voices (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford, Mathalon, et al., 2001a, 2001b; Ford et al., 2013; Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018). Alternatively, it might indicate error in attention allocation and control whereby voice hearers allocate attention to a contextually irrelevant stimulus (e.g., self-generated own voice). Given that these alterations are observed in both voice hearers with and without a diagnosis of a psychotic disorder, they potentially support the psychosis continuum hypothesis.

Emotions provide cues that can crucially influence the memory of an event in the source monitoring framework (Johnson, Hashtroudi, & Lindsay, 1993). Emotional quality of the hallucinated voices not only offers contextual information about the event's source but also imparts details about the distinctiveness of a voice. Any alteration in processing emotional characteristics may therefore increase the risk of misattribution of internally-generated sensations to external sources. The emotional quality of hallucinated voices is one of their main characteristics. They are more often negative and derogatory for voice hearers with a psychotic disorder and more positive and neutral for non-clinical voice hearers (Daalman, Boks, et al., 2011; Johns et al., 2014; Larøi, 2012). This difference in the emotional quality of AVH in non-clinical voice hearers and voice hearers with a psychotic disorder has also been reported in vocal emotion processing (Amminger, Schafer, Klier, et al., 2012; Amminger, Schafer, Papageorgiou, et al., 2012; Amorim et al., 2022; van 't Wout et al., 2004). Voice hearers with a psychotic disorder tend to perceive negative emotions more intensely and to misattribute negative meaning to a neutral stimulus, potentially due to altered predictions causing them to constantly anticipate negative sensations and pay involuntary attention to negative cues (Mohanty et al., 2008). Further, voice hearers with a psychotic disorder tend to show low positive affect, reduced pleasure expression and recognition (Cohen & Minor, 2010; Horan, Blanchard, Clark, & Green, 2008; Kring & Moran, 2008; Li, Fung, Moore, & Martin, 2019; Watson & Naragon-Gainey, 2010). This reduced ability to process positive emotions is closely associated



with negative symptoms such as social aloofness and constricted affect (Watson & Naragon-Gainey, 2010). If the heightened perception of negative emotions and the tendency to attribute negative connotations to neutral stimuli among voice hearers with a psychotic disorder are linked to their inherent attentional bias favoring negative cues (Alba-Ferrara et al., 2013; Birchwood & Chadwick, 1997; Galdos et al., 2011; Nelson et al., 2014a, 2014b; Rossell & Boundy, 2005), it can be hypothesized that non-clinical voice hearers, who frequently encounter positive and neutral voices, may have an enhanced ability to discern positive content within neutral stimuli. By manipulating positive emotional quality it may be possible to vary certainty about sensory feedback to self-voice production as well as probe control of attention allocation. This manipulation may allow disentangling sensory feedback processing and control of attention allocation in non-clinical individuals who are highly prone to hallucinations. Understanding these processes within the context of positive emotion as a function of HP in non-clinical individuals, might help develop interventions to improve diminished pleasure perception and motivation in voice hearing (Foussias, Agid, Fervaha, & Remington, 2014; Nguyen et al., 2016).

The excellent temporal resolution of EEG allows sensitive monitoring of dynamical changes in voice quality. Here, we extended prior work (Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018), using a well-validated EEG motor-auditory task (figure 1) where the self-voice changes from fully neutral to fully pleasure - 100% neutral, 60-40% neutral- pleasure; 50-50% neutral- pleasure; 40-60% neutral-pleasure and 100% pleasure. We focused on the N100, P200, and N200 ERP responses as established indicators of sensory feedback processing, conscious differentiation between self-generated and externally-generated events, and attention allocation and error awareness. For 100% neutral and 100% pleasure self-voice, we expected to observe reduced N100 and P200 suppression effects (self- > externally-generated) with increased HP. The hypotheses for the direction of the effect of uncertain/ambiguous self-voices were more exploratory. Previous studies (Addington et al., 2008; Amminger, Schafer, Klier, et al., 2012; Amminger, Schafer, Papageorgiou, et al., 2012; Amorim et al., 2022) showed that participants scoring high on HP tend to miscategorize vocal emotions, in particular, ambiguous voices from the neutral-pleasure continuum were categorized as 'angry'. Therefore, we expected an increase

in N100, P200 as well as N200 amplitude for self- than externally-generated ambiguous (uncertain: 60-40%: neutral-pleasure; 50-50%: neutral-pleasure; 40-60%: neutral-pleasure) self-voices in individuals with low compared to high HP individuals. The underlying rationale was that individuals characterized by low HP would display changes in both sensory feedback processing and error perception and awareness when listening to unexpected and ambiguous self-voices.

## **2. Methods**

### *Participants*

Twenty-nine healthy adults (age range 18-27 years) were recruited. All participants were first invited for a voice recording, followed by the EEG session. Three participants did not participate in the EEG sessions due to time constraints, whereas one participant was excluded from further analysis due to technical issues during the EEG data collection. Therefore, the final participant number was 25 (21 females, mean age = 21.24, s.d. = 2.49 years; 21 right-, 3 left-handed, and 1 ambidextrous) varying in HP (Launay Slade Hallucination Scale (LSHS)(Castiajo & Pinheiro, 2017; Larøi & Van der Linden, 2005a, 2005b; Launay & Slade, 1981) total scores: mean = 18.56, s.d. = 10.17, max = 42, min = 3; LSHS AVH scores [sum of items: “In the past, I have had the experience of hearing a person’s voice and then found no one was there”, “I often hear a voice speaking my thoughts aloud”, and “I have been troubled by voices in my head”]: mean = 2.40, s.d. = 2.62, min = 0, max = 11). All participants provided their written informed consent before the start of the study. They either received financial compensation (vouchers) or study credits for their participation. All participants self-reported normal or corrected-to-normal visual acuity and normal hearing. The study was approved by the Ethics Committee of the Faculty of Psychology and Neuroscience at Maastricht University and performed in accordance with the approved guidelines and the Declaration of Helsinki (ERCPN-176\_08\_02\_2017\_S2).

### *Procedure*

All participants went through the following sessions performed in two separate visits.

Session 1: Voice recording and stimulus generation

### *Voice recording*

Participants comfortably sat inside an acoustically and electrically shielded chamber with the recording equipment, while the researcher sat outside this chamber. Recordings were made using a Rode NTKb microphone powered by a Rode NTK microphone power supply (<http://www.rote.com/microphones/ntk>) and processed with the Praat software (<https://www.praat.org>). Participants were instructed to repeatedly vocalize “ah” and “oh” in a neutral (no emotion) and in a pleasure voice. Vowels were chosen to eliminate semantic content (Cook & Wilding, 2001; Schweinberger et al., 1997; Ventura, Nagarajan, & Houde, 2009). Participants were asked to vocalize the vowels for 500 ms, and were provided with examples to familiarize them with the target duration of the vocalization. This duration was chosen to properly capture the emotionality while maintaining adequate length for self-voice recognition. The best voice samples were selected once the participants confirmed that they recognized their recorded voice, that the pleasure intensity was the highest that they could produce, that they perceived no emotion in the neutral recording, and if the vowels were pronounced clearly. Background noise was eliminated from the recordings using Audacity software (<https://audacityteam.org/>) and a Praat script was applied to normalize the intensity to 70 dB. The duration of the final neutral and pleasure “ah” and “oh” vocalizations for each participant was 500 ms.

### *Morphing*

To create voice samples with varying degrees of emotional content, the pre-recorded neutral and pleasure self-voices for each individual participant were parametrically morphed to create neutral-to-pleasure and pleasure-to-neutral continua. These continua consisted of 11 stimuli with a 10% stepwise increase (neutral-to-pleasure)/decrease (pleasure-to-neutral) in emotionality along the continuum (see supplementary table 1). Morphing was performed using the TANDEM-STRAIGHT software (Kawahara, 2006; Kawahara & Irino, 2005; Kawahara et al., 2008) running on MATLAB (R2019a, v9.6.0.1072779, The MathWorks, Inc., Natick, MA). For the final EEG experiments, 100% neutral, 60-40%: neutral-pleasure; 50-50%: neutral-pleasure; 40-60%: neutral-pleasure and 100% pleasure voice morphs were selected.

The intermediate voice morphs were selected based on pilot data that revealed that the maximum uncertainty to differentiate a neutral from a pleasure self-voice fell in the range of 35-65% morphing. The increase in emotional voice quality (as self-voice stepwise changes from fully neutral to fully pleasure) and manipulations of uncertainty (most certain: 100% neutral and pleasure; uncertain: 60-40%: neutral-pleasure; 50-50%: neutral-pleasure; 40-60%: neutral-pleasure self-voice morphs) would probe both changes in sensory feedback to the self-voice and attention allocation resulting from these changes.

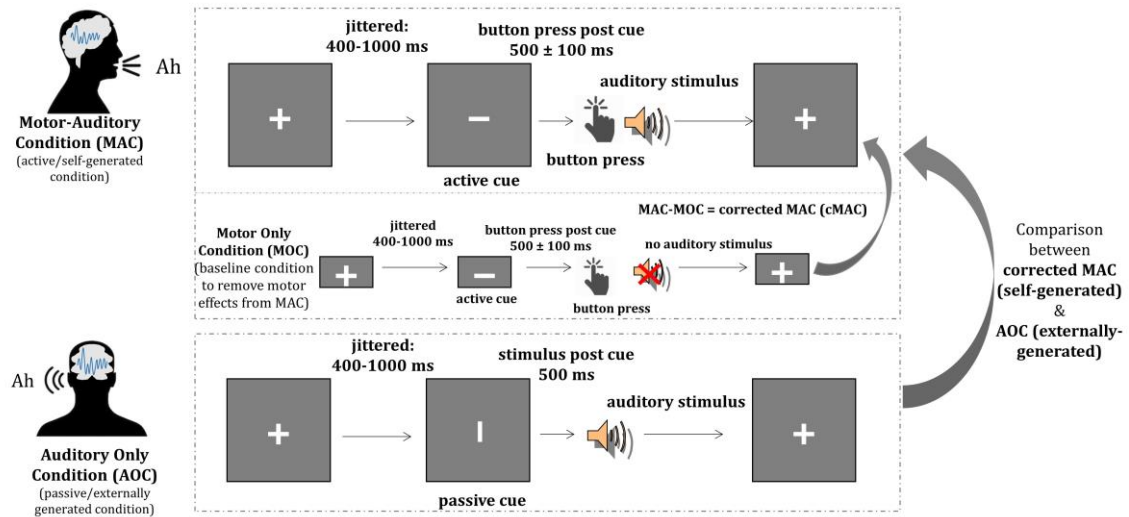
## Session 2: EEG

Participants were given an overview of the procedure and the principles of EEG at the start of the session. They sat comfortably in an electrically shielded soundproof chamber in front of a screen placed about 100 cm away. Participants filled in the LSHS questionnaire while the EEG cap was prepared.

### *Auditory-motor task*

A variant of an established button-press task was employed to investigate differences between responses to self- and externally-generated auditory stimuli (Pineiro, Schwartze, Amorim, et al., 2020; Pineiro et al., 2018) (figure 1). This task comprises three conditions: a motor-auditory condition (MAC), where participants pressed a button to generate their pre-recorded voice; an auditory-only condition (AOC), where participants passively listened to their pre-recorded voice; and a motor-only condition (MOC), where they pressed a button but did not hear their voice. This latter condition was used to control for motor activity resulting from the button-press in the MA condition ( $MAC - MOC = \text{corrected MAC [cMAC]}$ ). Previous studies have consistently shown that there is a reduction in the N100 amplitude in response to self-generated sound via a button-press compared to passively listening to the same sound (Baess et al., 2009; Hughes et al., 2013), indicating that button-presses can be used as a motor-act to approximate self-generation of a speech stimulus (for voices see Knolle et al., 2019).

**Figure 1:** Graphical representation of the Motor-auditory task. Abbreviations: MA = Motor Auditory Condition; AO = Auditory Only Condition; MO = Motor Only Condition. Motor activity from MA condition was removed by subtracting MO from MA to obtain MA corrected condition. Statistical analyses were performed with ERPs from MAC and AO conditions.



The paradigm was presented in a fully randomized event-related design over 12 runs. Each run consisted of 80 trials (40 AO, 40 MA, and 10 MO). Each trial started with a fixation cross, after which the presentation (vertical or horizontal) of a cue was jittered between 400-1000 ms. The cue was then followed by an auditory stimulus (after 500 ms for AO) or a button press that may (MA) or may not (MO) elicit an auditory stimulus. Five types of voice morphs consisting of “ah” and “oh” vocalizations, respectively, were presented in the AO and MA conditions. Thus, each run consisted of 4 trials of 10 stimulus types each (“ah” and “oh” for 5 voice morphs). This included 96 trials per voice morph (“ah” and “oh” combined, supplementary table 1). Participants were given short breaks after each run. To minimize potential influences of lateralized motor activity, participants were asked to switch their response hand every three runs. Prior to the experiment, participants were trained to press the button within 500 ± 100 ms after the cue (horizontal bar) to align the presentation of auditory stimuli in the MA and AO conditions and to avoid overlap of cue-elicited and motor activation.

The task was programmed and presented using the Presentation software (version 18.3; Neurobehavioral Systems, Inc.). Auditory stimuli were presented via in ear inserts. Button presses were recorded via the spacebar button on the keyboard.

### *Stimulus Rating*

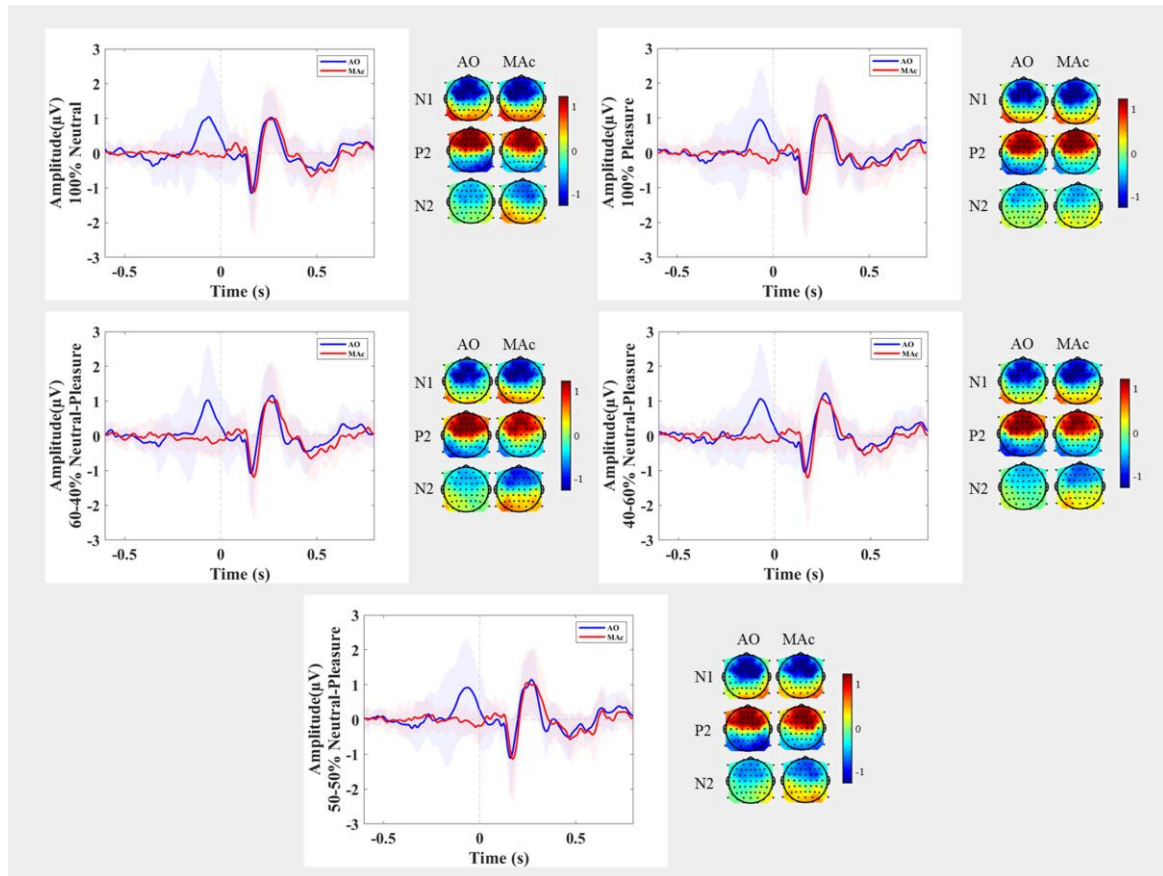
At the end of the EEG session, participants rated their voices for arousal and valence (supplementary figure 1). They additionally rated the voices on perceived ownness, i.e., how much they identified their self-voice on a Likert scale (0-10). This was done to ensure that participants recognized their own voice and perceived the emotion expressed by it.

### *EEG data acquisition and preprocessing*

EEG data were recorded with BrainVision Recorder (Brain Products, Munich, Germany) using an ActiChamp 128-channel active electrode setup while participants performed the auditory-motor task. Data were acquired with a sampling frequency of 1000 Hz, an electrode impedance below 10 k $\Omega$ , using TP10 as online reference. EEG data were pre-processed using the Letswave6 toolbox (<https://github.com/NOCIONS/letswave6>) running on MATLAB 2019a. Data were first cleaned to remove false button presses (e.g., trials with button presses during AO), downsampled to 500 Hz, and then bandpass filtered (1-30 Hz). All channels were re-referenced to the average of the mastoid electrodes. Eye blinks and movements and noisy electrodes were removed using an independent component analysis (ICA) with the runica algorithm in combination with Rajan and Rayner (PICA) as implemented in Letswave6 (<https://github.com/NOCIONS/letswave6>). ICs representing primarily noise were removed for each participant based on the IC time course and topography. The resulting data were segmented using a -600 to 800 ms time-window relative to the onset of the auditory stimulus. The segmented data were baseline corrected to a -600 to -400 ms window. This remote baseline was selected due to a cue-related ERP modulation before the onset of the auditory stimulus in AO. After baseline correction, an automatic artifact rejection algorithm was applied with an amplitude criterion of  $\pm 65\mu\text{V}$  to remove epochs/trials with remaining artifacts. The resulting data were then averaged for each participant and each condition. The grand averaged waveforms

revealed three ERP components, two negative components peaking at 164 ms and 460 ms and one positive component peaking at 286 ms. As the latencies of the ERP responses varied significantly (supplementary table 2), peak amplitudes as an outcome measure were chosen for data quantification. The N100 peak amplitude was defined as the largest negative peak occurring between 80-230 ms, the P200 peak amplitude was defined as the following positive peak between N100 and 380 ms, and the N200 peak amplitude as the negative peak between the P200 and 600 ms (Swink & Stuart, 2012a, 2012b). Previous research showed that the ERP components of interest all have prominent fronto-medial and fronto-central topographies (Behroozmand et al., 2009; Chen et al., 2012; Korzyukov et al., 2012). Therefore, the N100, P200, and N200 responses were extracted from the same fronto-central region of interest (ROI) that included 21 electrode locations: AFF1h, AFF2h, F1, Fz, F2, FFC3h, FFC1h, FFC2h, FFC4h, FC3, FC1, FCz, FC2, FC4, FCC3h, FCC1h, FCC2h, FCC4h, C1, Cz, C2 (figure 2).

**Figure 2:** Grand average ERP waveforms  $\pm$  standard error of mean and topographic maps showing voltage distribution at the peak ERPs, comparing self-generated and externally-generated voices for the five self-voice types originating from a fronto-central ROI. Abbreviations: MAc = Motor Auditory Corrected; AO = Auditory Only.



### *Statistical analyses*

Statistical analyses on N100, P200 and N200 data were performed in R version 4.2.2 (2022-10-31) Copyright (C) 2022, using linear mixed modeling with lmer and lmerTest packages (Bates, 2016; Kuznetsova et al., 2017). We used linear mixed modeling (LMM) to control for the random effects of participants influencing the outcome measure. Additionally, since HP measured by the LSHS is a continuous variable, LMMs are considered more appropriate than classical ANOVA to analyze the impact of HP on sensory prediction (condition) and salience (stimulus type). Amplitude values of the event related potential (N100/P200/N200) were used as an outcome measure, participants were used as random effects, and condition (2 levels: MAc and AO), stimulus type (5 levels: 100% neutral, 60-40% neutral-pleasure, 50-50% neutral-pleasure, 40-60% neutral-pleasure, 100% pleasure) and LSHS total or LSHS AVH scores (continuous variable) were included as fixed effects, respectively, in



the hypothesized models. For all the models, the Gaussian distribution of model residuals and quantile-quantile plots confirmed their respective adequacy.

### 3. Results

We followed a hypothesis-driven approach to specifically probe the interaction of sensory feedback processing (condition) and emotional voice quality (stimulus type) with HP.

*N100 & P200*: None of the models testing the interaction of HP with sensory feedback processing (condition) and emotional quality (stimulus type) showed significant differences from the null models (supplementary table 2 and 3).

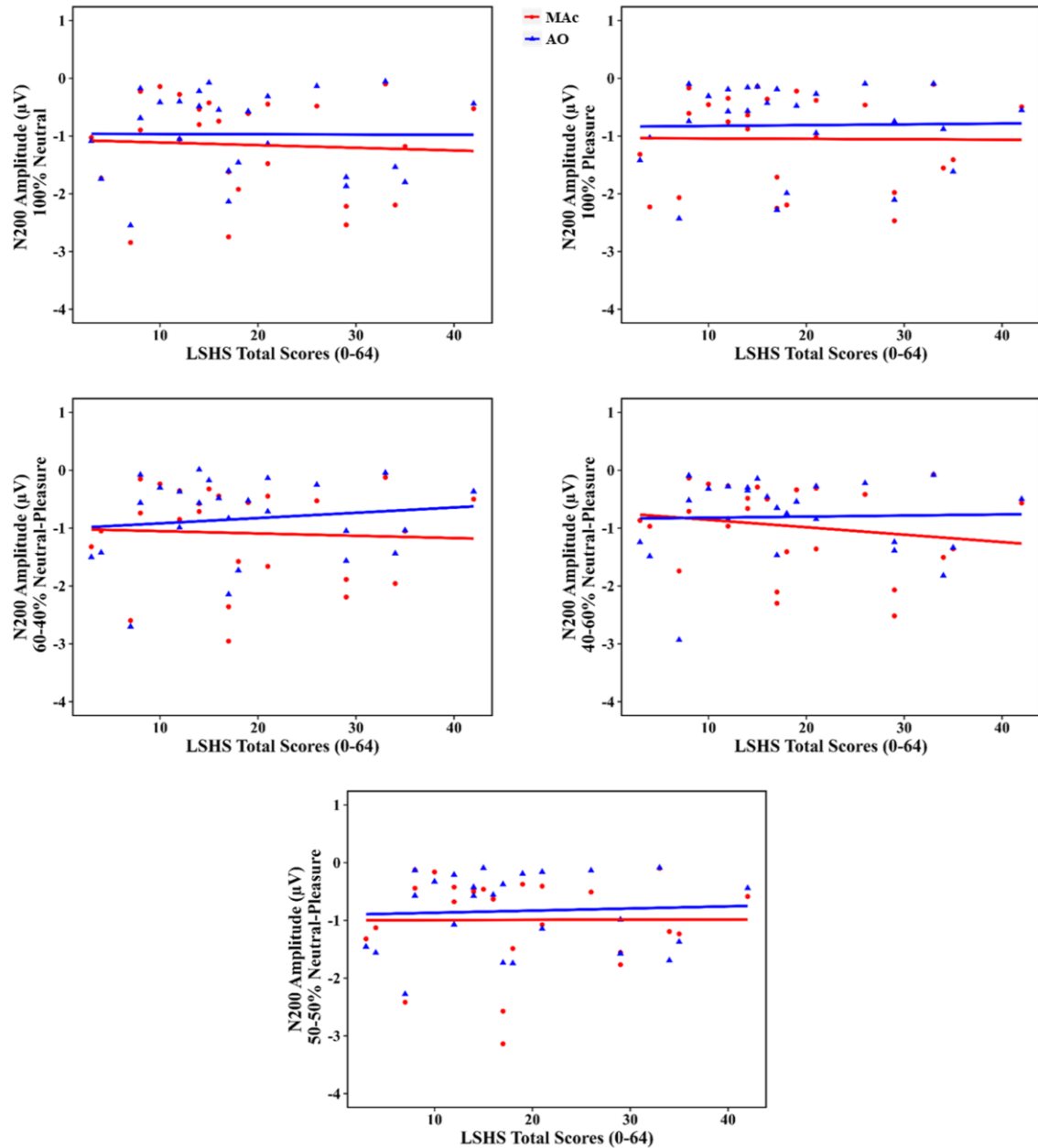
*N200*: The model that showed best goodness of fit [ $m1.1\_N200 \leftarrow \text{lmer}(N200 \sim + \text{Condition} * \text{LSHS total} + \text{Stimulus Type} + (1|ID), \text{data}=\text{data}, \text{REML} = \text{FALSE})$ ] also yielded a significant difference ( $\chi^2(7) = 34.621, p = 0.000^{**}$ ; AIC = 245.43; table 1, figure 3) against the null model [ $m0\_N200$ ; AIC = 266.05]. There was no notable impact of the condition, and the influence of HP on the N200 suppression effect was not statistically significant.

**Table 1:** Linear mixed effects model of N200 amplitude including the effect of hallucination proneness based on LSHS total scores. Notes: SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 225.0$  (except Intercept:  $df = 26.07$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-1.077133	0.287934	-3.741	0.000871 ***
AO	0.057689	0.082453	0.700	0.484863
LSHS total	-0.004347	0.013474	-0.323	0.749531
60N	0.096563	0.062670	1.541	0.124767
50N	0.145844	0.062670	2.327	0.020845 *
40N	0.174131	0.062670	2.779	0.005922 **

Pleasure	0.128594	0.062670	2.052	0.041335 *
AO*LSHS total	0.007476	0.003896	1.919	0.056223 .
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	0.45025	0.6710	
Residual		0.09819	0.3134	
Number of observations: 250, Subjects: 25				

**Figure 3:** Scatter plots depicting the change in N200 amplitudes as a function of HP (based on LSHS total scores) for each stimulus type. While it appeared as if the N200 response differed for self- and externally-generated self-voices with increase in HP, this result only approached statistical significance ( $p = 0.056$ ).



#### 4. Discussion

This EEG study investigated changes in sensory feedback processing of one's own voice and attention allocation as a function of HP by manipulating the positive emotionality of self-voice stimuli. Specifically, we examined the auditory N100, P200, and N200 responses for the self- and externally-generated self-voice, using a modified version of a previously established motor-auditory paradigm (figure 1; (Pinheiro, Schwartze, Amorim, et al., 2020; Pinheiro et al., 2018)). However, contrary to

expectations, the ERP responses for self- and externally-generated voices were similar, leading to no discernable suppression effects. Moreover, HP did not modulate N100, P200 or N200 responses for the self- and externally-generated voices. Our findings thus question the notion of changes in attentional engagement or sensory feedback processing of the self-voice along the neutral-pleasure emotion expression continuum as a function of HP.

The recognition of vocal emotions relies on identifying physical characteristics such as base frequency, intensity, duration and pitch variability (Banse & Scherer, 1996; Juslin & Laukka, 2001; Kanske et al., 2013; Sauter, Eisner, Calder, & Scott, 2010). Specifically, vocal emotions characterized by moderate or low emotional intensity lead to diminished accuracy in recognizing emotions and require a longer processing time (Banse & Scherer, 1996; Juslin & Laukka, 2001; Kanske et al., 2013; Sauter, 2017; Sauter et al., 2010). For example, pleasure vocalizations are most often associated with long duration, low spectral center of gravity and high spectral variation (Sauter et al., 2010). These characteristics may pose challenges for capturing the impact of these vocalizations in early ERPs. Further, pleasure vocalizations are often confused with other positive emotions such as relief and contentment, leading to unclear and ambiguous overall perceptions (Sauter, 2017; Sauter et al., 2010). Notably, these emotions share similar physical properties as well as arousal and valence ratings, even though they differ semantically, meaning they are not synonyms (Sauter, 2017; Sauter et al., 2010). Based on these aspects, it is likely that the perceptual discriminability among the five types of self-vocalizations varying in pleasure content used here might have been low. Further, using the same sample of participants and design but a self-voice continuum from neutral to angry yielded a significant global N100 suppression effect (Duggirala et al., 2023). Hence, the question arises as to why similar effects did not emerge as a function of a continuum manipulation for positive vocal emotion expressions. Participants in the current study did not exhibit a complete sense of ownership (“my voice or someone else’s voice”) and agency (“feeling associated with the sensory outcome of one’s voluntary action”) of their own 100% pleasure voice. The sense of ownership was lower for the 100% pleasure stimuli compared to the 100% angry self-voice ((Herbert, Herbert, Ethofer, & Pauli, 2011; Yoshie & Haggard, 2013); supplementary figure 1C and supplementary figure 1C of Duggirala et al., 2023)).

Likewise, in the epistemological domain of positive emotions, pleasure vocalizations link to a comparatively diminished sense of agency when contrasted with elation or pride (Sauter, 2017). Likewise, research indicates that emotion perception is susceptible to contextual influence (Liuni, Ponsot, Bryant, & Aucouturier, 2020; Mauchand & Zhang, 2023; Paulmann & Pell, 2010). It is likely that the prevalence of heightened ambiguous expression (3:2 → ambiguous:unambiguous self-voice), next to perceptual discriminability, might have created an overall ambiguous context. Therefore, the lack of perceptual discriminability among the self-voices from the neutral-pleasure continuum and the low sense of ownership and agency, might have established an overall ambiguous context. In turn, these factors might have resulted in no changes in the certainty of sensory feedback in self-voice or differential attentional engagement, leading to no global N100/P200 suppression effect and no discernable effects of voice quality on N100/P200 suppression effects.

Research findings indicate that the processing and attention-capturing functions of positive emotions require a longer duration and are evidenced in the ERP components occurring in approximately the latency range of 200-550 ms (Carretié, Hinojosa, Martín-Loeches, Mercado, & Tapia, 2004; Carretié, Martín-Loeches, Hinojosa, & Mercado, 2001; Xue et al., 2013). The N200 response (regardless of condition and HP; table 1, supplementary table 2, supplementary table 5) showed a global reduction for self-voices with more than 50% pleasure content. Further, while it appeared as if the N200 response differed for self- and externally-generated self-voices with increasing HP, this result only approached statistical significance ( $p = 0.056$ , table 1, figure 3). Considering the initial argument of lack of ownership in pleasure vocalizations and ambiguous context, this reduced N200 response for self-voices high on pleasure content, might reflect a decreased ability to categorize and process these voices in general (compared to 100% neutral voice) but decreased further in high HP individuals. Alternatively, this might also indicate an absence of attentional bias towards pleasure compared to non-emotional neutral expressions. Previous studies have shown that schizotypy is associated with less attentional focus on positive affect and feelings, and experiencing and anticipating less pleasure (Giakoumaki, 2016; Kerns, Docherty, & Martin, 2008; Li et al., 2019; Martin, Becker, Cicero, Docherty, & Kerns, 2011). Likewise, individuals scoring high on HP misidentified pleasure content

in vocalizations from the pleasure-neutral continuum (Amorim et al., 2022). Authors in this study have speculated that the high HP participants' intrinsic attentional bias towards negative emotions leading them to possibly ignore or allocate less attentional resources to non-negative emotional stimuli, i.e., neutral or pleasure.

A limitation related to the task-design is noted. In contrast to the conventional blocked design in auditory-motor tasks, the current mixed design included a visual cue to prompt the respective condition of interest. Furthermore, there was a fixed duration of 500 ms between the visual cue and the onset of the auditory stimulus in the externally-generated condition. While the influence of the visual cue was eliminated in the MA condition by subtracting MO from MA to eliminate motor effects, in the AO condition, the response to the visual cue remained as a pre-stimulus positive potential. Both the fixed delay and the visual cue could have enhanced anticipation of the externally-generated voice and focused attention on it (Heynckes, De Weerd, Valente, Formisano, & De Martino, 2020; Sowman et al., 2012). Consequently, the listener might have been better prepared for the sound to occur, approximating the preparedness for the sensory consequences of a self-generated voice in the MA condition (Costa-Faidella et al., 2011; Heynckes et al., 2020; Sowman et al., 2012). However, it should be noted that previous studies using both cued and uncued externally-generated conditions showed no difference in suppression effects (Griffiths, Jack, Pearson, Elijah, Mifsud, Han, Libesman, Rita Barreiros, et al., 2023; Lange, 2013). Studies have reported that it is not the motor-action per se, but the voluntary intention involving motor planning to self-generate an action (e.g., a voice) that leads to a sensory suppression (Jack et al., 2021; Timm, SanMiguel, Keil, Schroger, & Schonwiesner, 2014). Therefore, the null findings associated with the N100/P200 suppression effects cannot be attributed exclusively to factors associated with the current task-design.

Various factors (e.g., lack of acoustic differences, ambiguous context, task design) discussed earlier are probable contributors to the absence of significant findings in the present study. Future investigations should take these factors into account and analyze voices in distinct blocks. Despite the lack of significant results in this study, the investigation of positive emotions in processing a self-relevant stimulus (e.g., self-voice) is important. Voice hearers with a psychotic disorder have difficulties in

experiencing positive emotions such as pleasure and motivation (Cohen & Minor, 2010; Horan et al., 2008; Kring & Moran, 2008; Li et al., 2019; Watson & Naragon-Gainey, 2010). These difficulties are associated with negative symptoms such as constricted affect and social aloofness, which in turn are influenced by positive symptoms such as hallucinations (Watson & Naragon-Gainey, 2010). Therefore, probing how processing of a positive self-relevant stimulus (e.g., self-voice) affect allocation of attention and sensory feedback processing of own voice in non-clinical individuals who are highly prone to hallucination can contribute to understanding the pre-existing limitations in processing positive emotions within non-clinical samples. Future studies should also include assessments of negative symptoms (e.g., lack of motivation, depression, stress) while studying positive emotions in voice hearing as they may act as a mediating factor in the relationship between positive emotion processing and HP. This, in turn, may provide insights for developing approaches to enhance pleasure perception and increase motivation in voice hearers.

**Funding:** The current study was supported by BIAL Foundation (BIAL 238/16).

**Data availability:** The data that support the findings of this study are available from the corresponding author upon reasonable request.

**Author Contributions:** SxD, MS, DL, AP, SK conceptualized and designed the experiment, SxD prepared materials, collected and analyzed the data, and wrote the first draft of the manuscript, SxD, MS, LG, DL, AP, SK refined the manuscript. MS, AP, SK procured funding for the project. All authors have approved the final version of the manuscript.

**Competing Interests:** The authors declare that they have no competing interests.

**Acknowledgements:** The authors would like to thank Joseph Johnson for sharing the paradigm code that was adapted and used for the current experiment. We would also like to thank Alexandra Emmendorfer for detailed discussions on data analysis.

## Supplementary Document

### Table and table legends

**Supplementary table 1:** Neutral-Pleasure continua with 11 voice morphs.

a) Neutral-to-pleasure

Emotion / Morphs	<b>1*</b>	2	3	4	<b>5*</b>	<b>6*</b>	<b>7*</b>	8	9	10	<b>11*</b>
Neutral	<b>100%</b>	90%	80%	70%	<b>60%</b>	<b>50%</b>	40%	30%	20%	10%	<b>0%</b>
Pleasure	<b>0%</b>	10%	20%	30%	<b>40%</b>	<b>50%</b>	60%	70%	80%	90%	<b>100%</b>

b) Angry-to-pleasure

Emotion / Morphs	<b>1*</b>	2	3	4	<b>5*</b>	<b>6*</b>	<b>7*</b>	8	9	10	<b>11*</b>
Pleasure	<b>100%</b>	90%	80%	70%	<b>60%</b>	<b>50%</b>	40%	30%	20%	10%	<b>0%</b>
Neutral	<b>0%</b>	10%	20%	30%	<b>40%</b>	<b>50%</b>	60%	70%	80%	90%	<b>100%</b>

c) Final Stimuli for Ah and Oh vocalizations.

100% Neutral = Ah (a1 + b11) + Oh (a1 + b11)
60-40% Neutral-Pleasure = Ah (a5 + b7) + Oh (a5 + b7)
50-50% Neutral-Pleasure= Ah (a6 + b6) + Oh (a6 + b6)



40-60% Neutral-Pleasure = Ah (a7 + b5) + Oh (a7 + b5)
100% Pleasure = Ah (a11 + b1) + Oh (a11 + b1)

Note: a1 refers to the specific voice morph from table a, voice morph 1.

**Supplementary table 2:** Mean latencies of N100, P200 and N200 amplitudes.

	Mean (ms)	S.d (ms)	Min (ms)	Max (ms)
N100	162	0.027	0.08	0.23
P200	265	0.033	0.18	0.38
N200	462	0.078	0.25	0.6

**Supplementary table 3:** Model comparisons with N100 amplitude as output and HP, Condition and Stimulus.

Notes: SE = standard error; SD = standard deviation; NP = number of parameters; AIC = Akaike information criterion; BIC = Bayesian information criterion; Chisq = chi square; Df = degree of freedom; M1 = Condition \* Stimulus type \* LSHS total/AVH; M2 = Condition \* LSHS total/AVH + Stimulus type \* LSHS total/AVH; M3 = Condition \* LSHS total + Stimulus type; \*p < 0.05; \*\*p < 0.01; \*\*\*p < 0.001.

Models with LSHS total								
Model	Parameters	AIC	BIC	Log Lik	Deviance	Chisq	Df	Pr(>Chisq)
Null model	3	372.29	382.85	-183.14	366.29			
M1	22	402.08	479.55	-179.04	358.08	8.2081	19	0.9845
M2	14	387.77	437.07	-179.89	359.77	6.5166	11	0.8368
M3	10	380.61	415.83	-180.31	360.61	5.673	7	0.5784

Models with LSHS AVH								
Model	Parameters	AIC	BIC	Log Lik	Deviance	Chisq	Df	Pr(>Chisq)
M1	22	400.02	477.49	-178.01	356.02	10.268	19	0.946
M2	14	385.98	435.28	-178.99	357.98	8.3045	11	0.6858
M3	10	378.66	413.88	-179.33	358.66	7.6243	7	0.3669

**Supplementary table 4:** Model comparisons with P200 amplitude as output and HP, Condition and Stimulus.

Notes: SE = standard error; SD = standard deviation; NP = number of parameters; AIC = Akaike information criterion; BIC = Bayesian information criterion; Chisq = chi square; Df = degree of freedom; M1 = Condition \* Stimulus type \* LSHS total/AVH; M2 = Condition \* LSHS total/AVH + Stimulus type \* LSHS total/AVH; M3 = Condition \* LSHS total + Stimulus type; \*p < 0.05; \*\*p < 0.01; \*\*\*p < 0.001.

Models with LSHS total								
Model	Parameters	AIC	BIC	Log Lik	Deviance	Chisq	Df	Pr(>Chisq)
Null model	3	332.37	342.94	-163.19	326.37			
M1	22	355.08	432.56	-155.54	311.08	15.288	19	0.7041
M2	14	345.81	395.11	-158.91	317.81	8.5623	11	0.6622
M3	10	341.81	377.02	-160.90	321.81	4.5642	7	0.713

Models with LSHS AVH								
Model	Parameters	AIC	BIC	Log Lik	Deviance	Chisq	Df	Pr(>Chisq)
M1	22	360.75	438.23	-158.38	316.75	9.6177	19	0.9618
M2	14	348.73	398.03	-160.36	320.73	5.6463	11	0.8959
M3	10	341.82	377.04	-160.91	321.82	4.5476	7	0.715

The influence of hallucination proneness based on LSHS AVH scores was also tested. The model that showed the best goodness of fit [m2.1\_N200 <- lmer (N200 ~ + Condition \* LSHS AVH + Stimulus Type + (1|ID), data=data, REML = FALSE)] also yielded a significant difference ( $\chi^2(7) = 31.439$ ,  $p = 0.000$  \*\*; AIC = 248.61; supplementary table 5, supplementary figure 7) against the null model [m0\_N200; AIC = 266.05]. The N200 response for self- compared to externally-generated voices is significantly different, regardless of voice quality or HP.

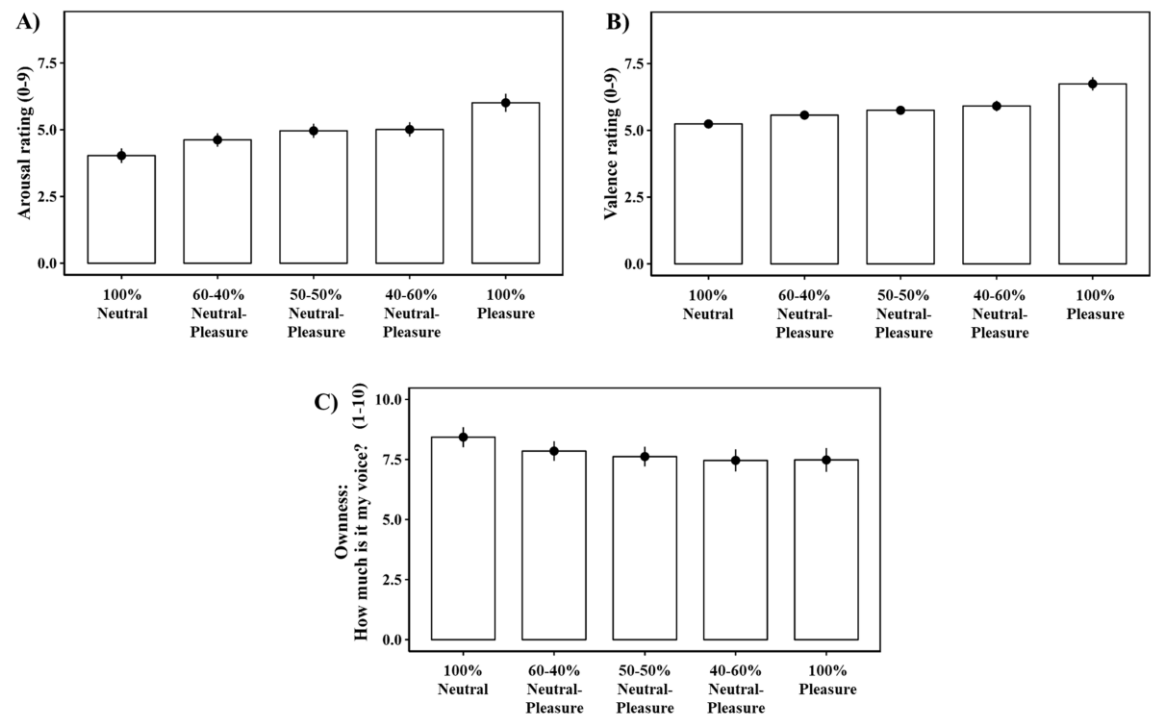
**Supplementary table 5:** Linear mixed effects model of N200 amplitude including the effect of hallucination proneness based on LSHS AVH scores. Notes: SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects: df = 225.0 (except Intercept: df = 28.54). There was a significant difference between the N200 response for self- and externally-generated voices.

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-1.160e+00	1.900e-01	-6.104	1.28e-06 ***
A0	1.717e-01	5.411e-02	3.173	0.00172 **

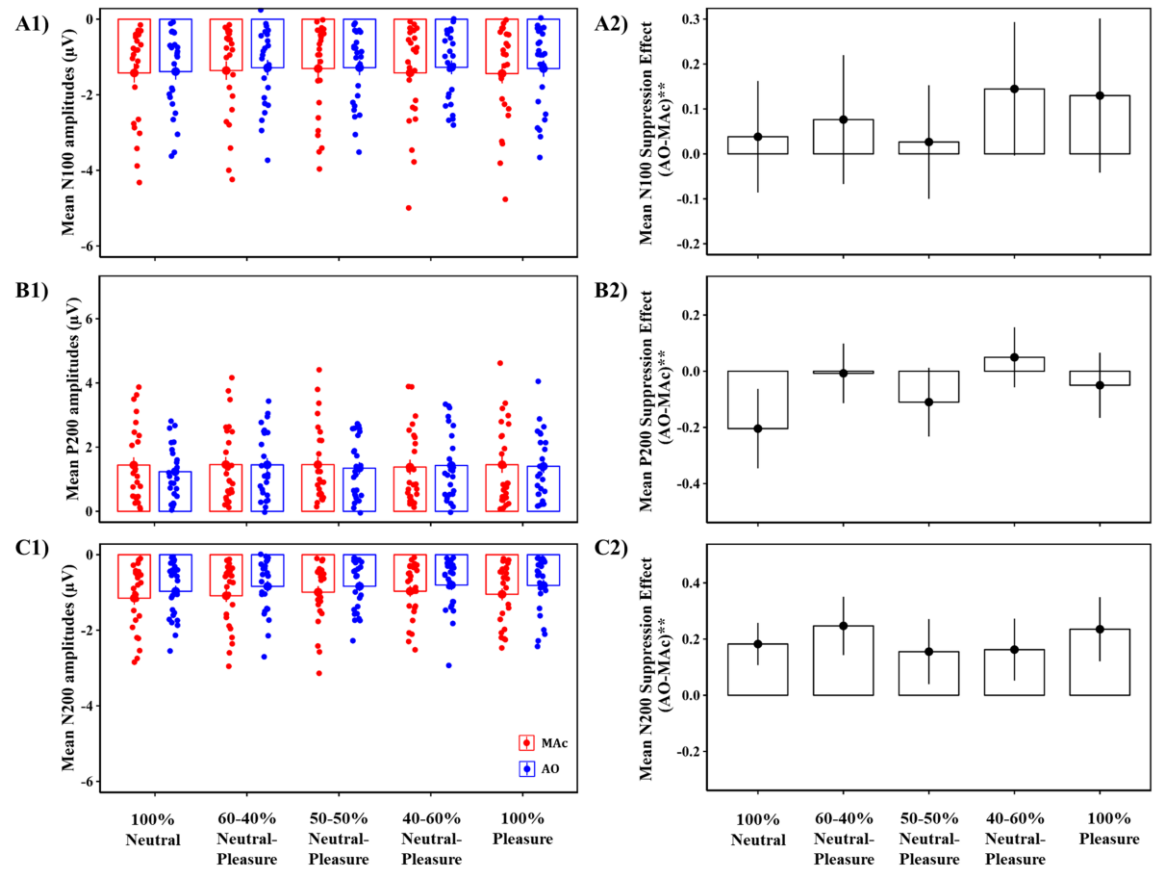
LSHS AVH	9.422e-04	5.226e-02	0.018	0.98575
60N	9.656e-02	6.312e-02	1.530	0.12744
50N	1.458e-01	6.312e-02	2.311	0.02175 *
40N	1.741e-01	6.312e-02	2.759	0.00628 **
Pleasure	1.286e-01	6.312e-02	2.037	0.04278 *
AO*LSHS AVH	1.033e-02	1.522e-02	0.679	0.49802
<b>Groups</b>	<b>Name</b>	<b>Variance</b>	<b>SD</b>	
<b>Random Effects</b>				
Subjects	Intercept	0.44989	0.6707	
Residual		0.09959	0.3156	
Number of observations: 250, Subjects: 25				

Figure and figure legends

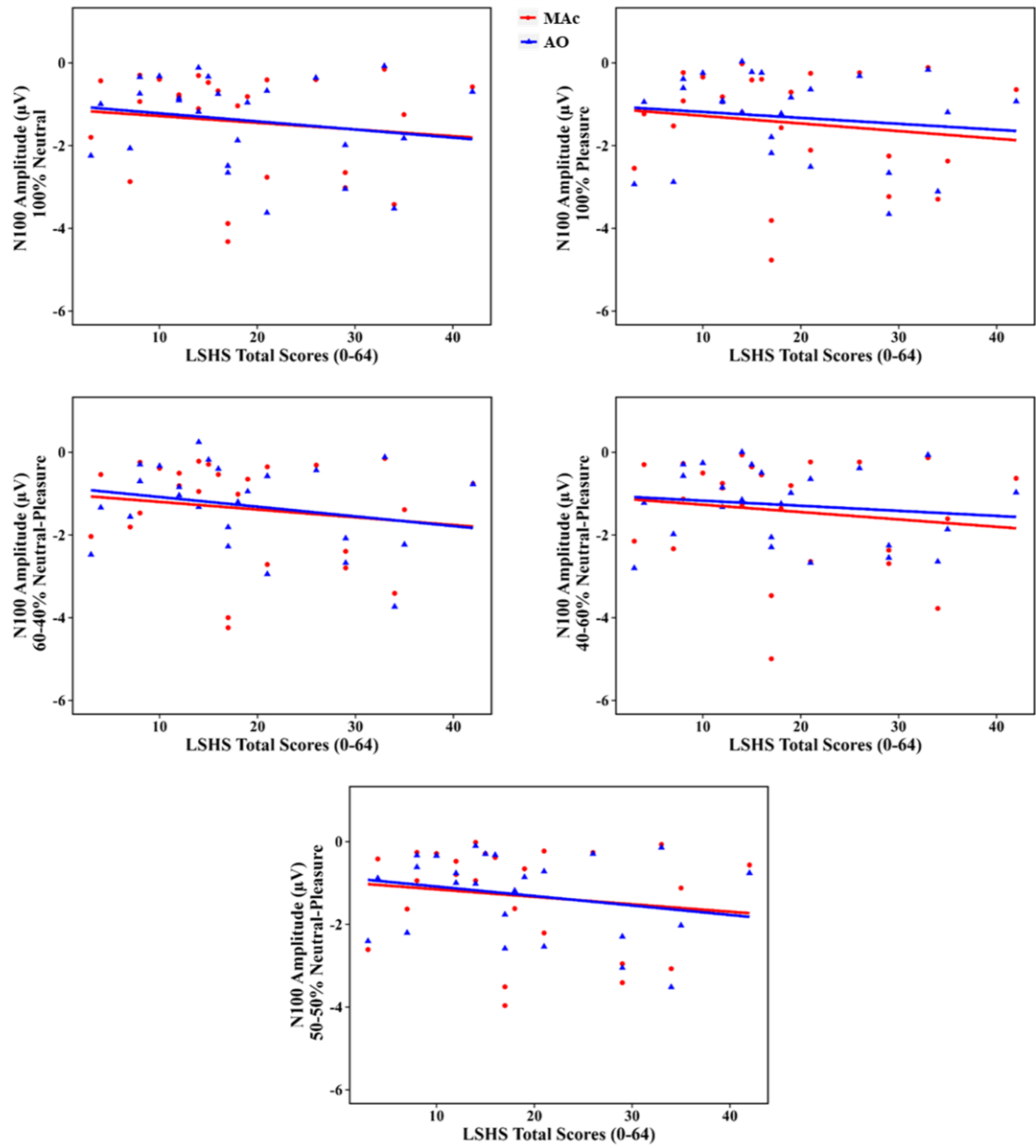
**Supplementary figure 1:** Post experiment stimuli rating. A) Arousal rating on a scale of 0-9 for each voice stimulus. B) Valence rating on a scale of 0-9 for each voice stimulus. C) Ownness rating on a scale of 1-10 for each voice stimulus.



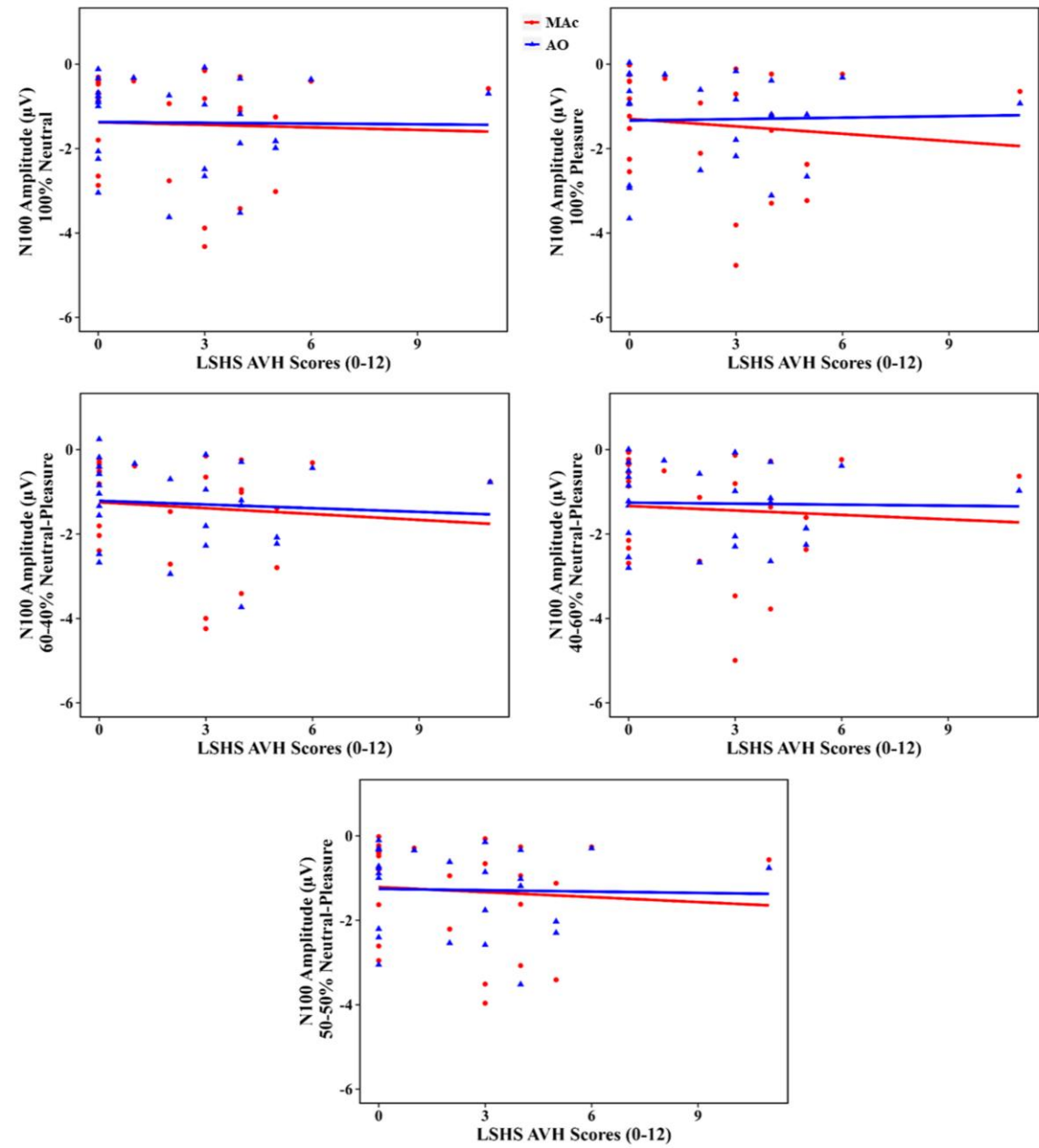
**Supplementary figure 2:** Mean ERP amplitudes for MAc and AO, and suppression effects (AO - MAc) per stimulus type. Note: Negative N100 suppression values depict AO > MAc whereas positive N100 suppression values depict MAc > AO



**Supplementary figure 3:** Scatter plots depicting the change in N100 amplitudes as a function of HP (based on LSHS total scores) for each stimulus type.

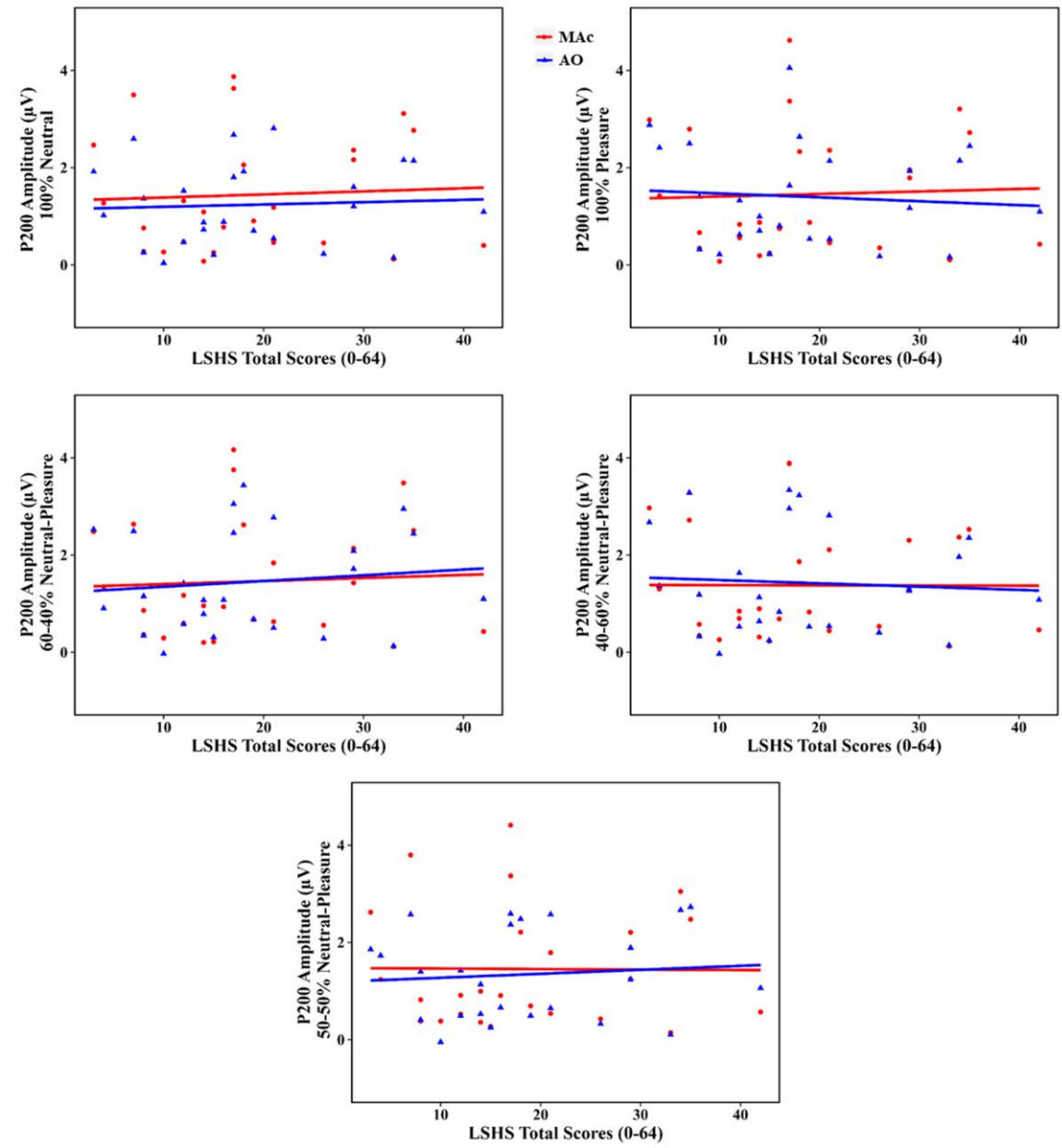


**Supplementary figure 4:** Scatter plots depicting the change in N100 amplitudes as a function of HP (based on LSHS AVH scores) for each stimulus type.

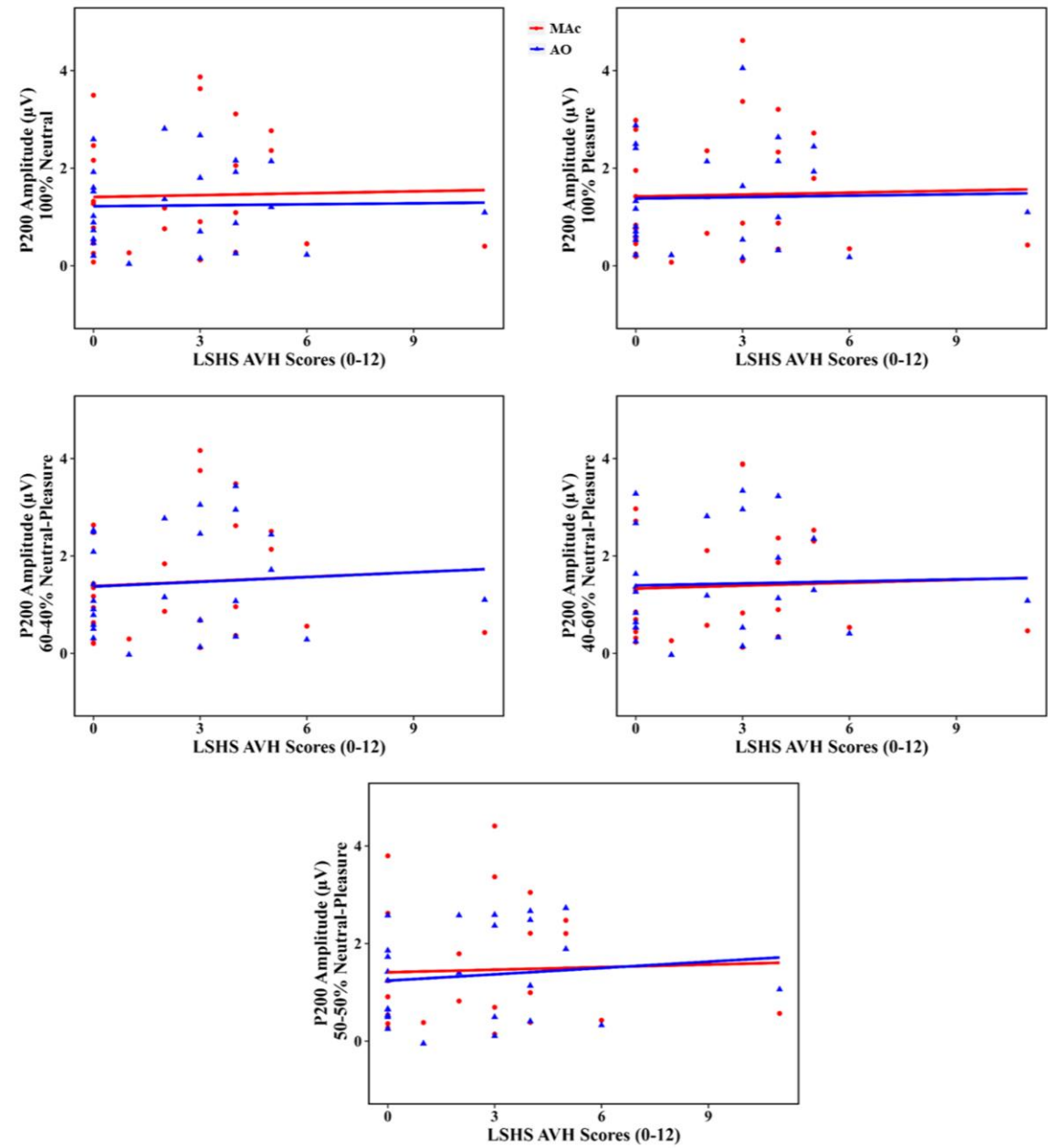




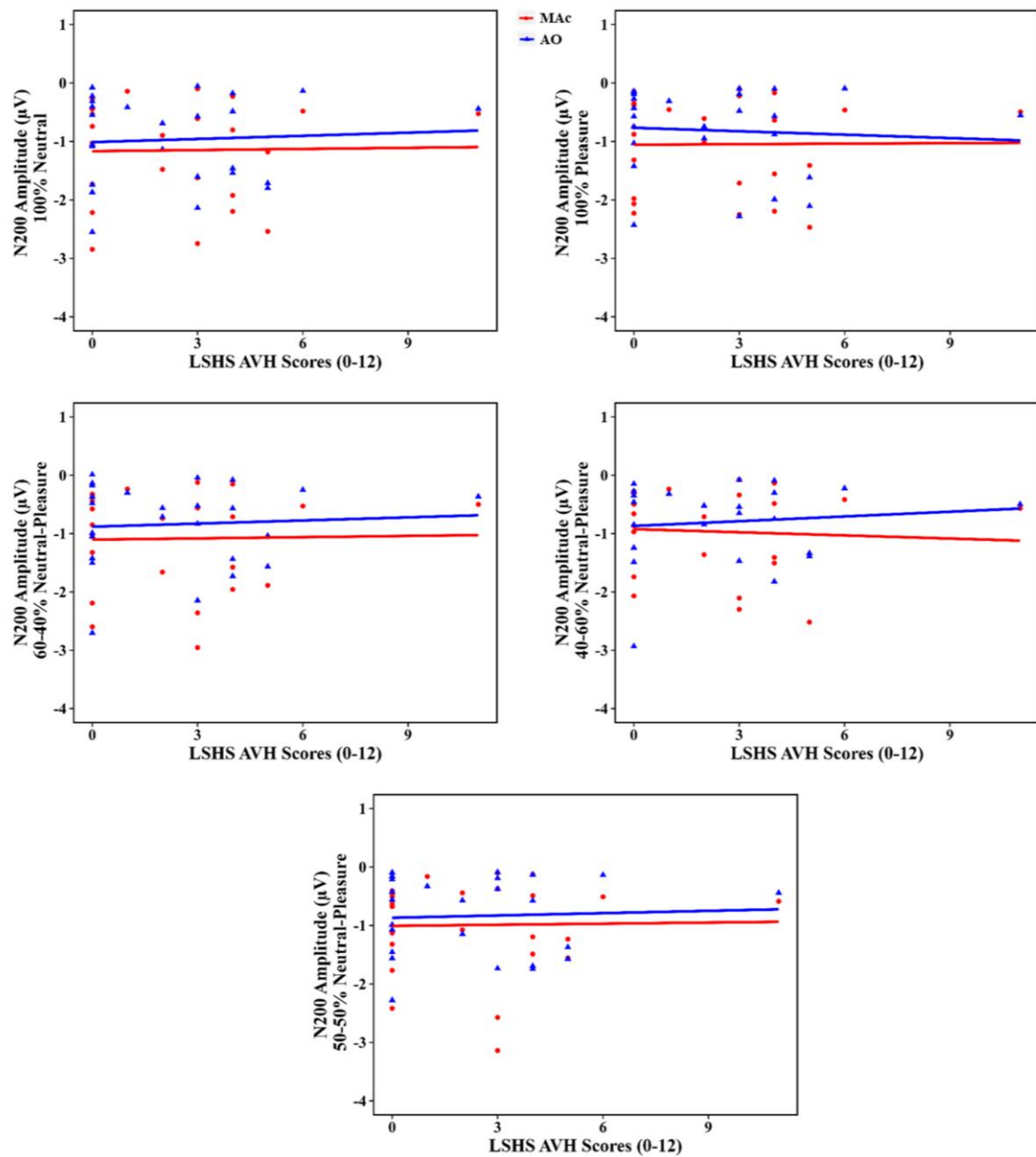
**Supplementary figure 5:** Scatter plots depicting the change in P200 amplitudes as a function of HP (based on LSHS total scores) for each stimulus type.



**Supplementary figure 6:** Scatter plots depicting the change in P200 amplitudes as a function of HP (based on LSHS AVH scores) for each stimulus type.



**Supplementary figure 7:** Scatter plots depicting the change in N200 amplitudes as a function of HP based on LSHS AVH scores for each stimulus type. Regardless of the voice quality, the N200 response for the self- and externally-generated voices were significantly different. However, HP did not modulate the N200 response from self- and externally-generated voices.





# **Chapter 6**

## **Exploring Neural Dynamics in Self-Voice Processing and Perception: Implications for Hallucination Proneness.**

*Under review as:* Duggirala, S. X., Honcamp, H., Schwartz, M., Amelsvoort, T., Pinheiro, A., Linden, D., & Kotz, S. A. (2023). Exploring Neural Dynamics in Self-Voice Processing and Perception: Implications for Hallucination Proneness. bioRxiv, 2023-09. <https://doi.org/10.1101/2023.09.21.558843>

## **Abstract**

Altered sensory feedback processing and attention control are assumed to contribute to auditory verbal hallucinations, which are experienced by the general population and patients with psychosis, implying a continuum of hallucination proneness (HP). However, the interaction of altered sensory feedback processing and attention control along this HP continuum remains unclear. Manipulating the level of certainty of sensory feedback by changing self-voice quality (100% neutral, 60-40% neutral-angry, 50-50% neutral-angry, 40-60% neutral-angry, 100% angry) in individuals varying in HP, we tested this interaction using electroencephalography while participants self-generated or passively listened to their voices. Regardless of voice quality, HP modulated the N100 and P200 suppression effects. High HP individuals showed an increased N100 response to the self-generated voices and an increased P200 response for externally-generated voices. This may indicate increased error awareness and attention allocation in high HP individuals for self-voice generation stemming from altered sensory feedback processing, and/or attentional control. The current findings suggest that alterations of the sensory feedback processing in self-voice production are a fundamental characteristic of the continuum of HP, regardless of the clinical status of voice hearers.

*Keywords:* Sensory feedback, Attentional control, Event related potential, Emotion, Voice hearing

## **1. Introduction**

Auditory verbal hallucinations (AVH) and hearing voices are perceptions without corresponding sensory input. Different cognitive models and theories have been proposed to account for AVH (Allen, Aleman, et al., 2007; Bentall, 1990; Brebion et al., 2000; Brebion, Amador, Smith, & Gorman, 1997; Feinberg, 1978; Fernyhough, 2004; Frith & Done, 1988; Johns et al., 2001; McGuire et al., 1996; Seal et al., 2004). Supported by the 'forward model' framework, source monitoring and inner speech models of AVH postulate that voice hearers misidentify an internally generated action (i.e., speech/voice) as originating from an external source (Blakemore, Goodbody, & Wolpert, 1998; Blakemore, Rees, et al., 1998; Blakemore, Wolpert, et al., 1998; Miall & Wolpert, 1995; Wolpert et al., 1998). However, an efficient perceptual system needs to clearly differentiate internally- from externally-generated sensory events. The forward model postulates that a top-down prediction (efference copy) of the sensory consequences of an internally-generated event is produced, which is then compared to the perceived sensory feedback. Based on the degree of mismatch (prediction error) between expected and actual sensory feedback, the internal model is updated and the event is classified as either internally- or externally-generated. This theory is supported by evidence from the visual (Sommer & Wurtz, 2006, 2008a, 2008b), tactile (Blakemore, Rees, et al., 1998; Blakemore et al., 2000; Shergill, Samson, Bays, Frith, & Wolpert, 2005), and auditory domain (Chang, Niziolek, Knight, Nagarajan, & Houde, 2013; Eliades & Wang, 2003; Houde, Nagarajan, Sekihara, & Merzenich, 2002; Numminen, Salmelin, & Hari, 1999; Ventura, Nagarajan, et al., 2009).

Electroencephalographic (EEG) studies identified the N100 auditory evoked potential as an indicator of the degree of mismatch between expected and actual sensory feedback (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford et al., 2013). The N100 primarily originates in the auditory cortex (AC) (Heinks-Maldonado et al., 2005; Krumbholz, Patterson, Seither-Preisler, Lammertmann, & Lütkenhöner, 2003; Mäkelä & Hari, 1987; Sams et al., 1985) and its amplitude is suppressed when expected and actual sensory feedback to a self-generated voice matches closely (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford et al., 2013). Conversely, a larger degree of mismatch leads to an increased N100 response and potentially also

leads to increased attention allocation (Schroger et al., 2015; Schröger et al., 2015). Altered self-other voice discrimination has been reported in both psychotic as well as non-clinical voice hearers (Allen, Aleman, et al., 2007; Allen, Amaro, et al., 2007; Allen et al., 2004; Pinheiro, Farinha-Fernandes, et al., 2019), which is evidenced by reduced N100 suppression and increased neural activity in AC for self- compared with externally-generated voice (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford et al., 2013; Oestreich et al., 2015; Perez et al., 2012; Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018). This indicates that self-generated sensory events might be processed incorrectly, and might be misattributed to an external source in voice hearers regardless of their need for clinical care. Similarly, the aberrant salience hypothesis puts forward that voice hearers misattribute salience to an irrelevant stimulus because they cannot inhibit attention to them (Alba-Ferrara et al., 2013; Kapur, 2003; Kapur, Mizrahi, et al., 2005). In empirical studies with voice hearers, misattribution of salience by allocating attention to irrelevant stimuli manifests as misattributing negative value to neutral stimuli (Allott et al., 2015) and as perceiving meaningful speech in noise (Galdos et al., 2011). Altered self-monitoring and salience misattribution theories might therefore share a common denominator through predictions (Davies et al., 2018; Nelson et al., 2014a, 2014b). Consequently, the lack of disengagement from prior expectations might result in increased attention allocation and attachment of meaning to non-relevant stimuli. Alterations of sensory feedback processing and attentional control therefore seem to present as two sides of the same coin in voice hearing.

The underlying neural mechanisms seem to be generic to AVH rather than psychosis-specific (Daalman, Boks, et al., 2011; Daalman, van Zandvoort, et al., 2011; Diederer, Daalman, et al., 2012; Sommer et al., 2010). Psychotic, clinically at-risk of psychosis, and non-clinical AVH may accordingly lie on a severity continuum that ranges from low to high hallucination proneness (HP) (Castiajo & Pinheiro, 2017; van Os, 2003; van Os et al., 2009). This suggests that the alterations in sensory feedback processing and attentional control evidenced in psychotic (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford et al., 2013) and non-clinical (Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018) voice hearers should also be present in non-voice hearers with high HP, albeit in an attenuated form. Emotional voice quality can



additionally affect sensory feedback processing and modulate AC activity (Pinheiro et al., 2017). The differences between non-clinical voice hearers and voice hearers with a psychotic disorder pertain to the emotional quality of their voices, controllability, and related distress (Daalman, Boks, et al., 2011; Johns et al., 2014). Voice hearers with a psychotic disorder often perceive derogatory voices that are beyond their control, which causes distress and significantly impacts their daily life (Daalman, Boks, et al., 2011; Johns et al., 2014). In contrast, non-clinical voice hearers tend to perceive neutral or positive voices, can exert control over them, and rarely experience distress (Daalman, Boks, et al., 2011; Johns et al., 2014). These differences between psychotic and non-clinical voice hearers influence how they evaluate and judge negative emotional content (Allott et al., 2014; Rossell & Boundy, 2005; Shea et al., 2007). Contrary to voice hearers with psychotic disorder, non-clinical voice hearers can downregulate negative emotions by dampening their emotional salience (Amorim et al., 2022). A systematic manipulation of negative emotional voice quality could thus provide insight into individuals' certainty about sensory feedback in their own voice, highlighting a potential trade-off with attentional control in persons who vary in HP. In turn, this may be key to a better understanding of the risk of transitioning from high HP to pathological voice hearing.

The current EEG study therefore systematically investigated effects of modulated sensory feedback to one's own voice varying in its degree of emotional quality and certainty of sensory feedback to self-voice as a function of HP. Using a well-validated auditory-motor task, participants self-generated and passively listened to their self-voice, which changed from fully neutral to fully angry: 100% neutral, 60-40% neutral-angry; 50-50% neutral-angry; 40-60% neutral-angry and 100% angry. The high temporal resolution of EEG allowed analysis of short-, mid- and long-latency event-related potential (ERP) markers to differentiate early and later auditory processing stages. Specifically, we probed sensory feedback processing and error awareness/attention allocation using the earlier N100 as primary outcome measure and then focused on the later categorical distinction of the self- from the externally-generated voice using the P200 as secondary outcome. The main hypothesis was that high HP would be linked to alterations of sensory feedback processing of the self-voice and of attentional control (Pinheiro et al., 2018). High HP individuals were expected

to display increased N100 and P200 responses to self- as compared to externally-generated unambiguous self-voice (100% neutral and 100% angry). The self-generated ambiguous self-voice (60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry) was expected to result in increased N100 and P200 responses in low but not high HP individuals.

## **2. Methods**

### *Participants*

45 adult participants, including 2 voice hearers with a diagnosis of a psychotic disorder were recruited (see supplementary document for power calculations). All participants followed the same procedure and three testing sessions. Prior to any assessment, participants were informed about the study procedures via an information letter as well as in person. Five participants could not participate in further sessions. Therefore, the final sample included 40 participants (26 females, 13 males, 1 other; mean age = 24.45, s. d = 2.33 years; range = 20-44 years) varying in HP as measured by the Launay Slade Hallucination Scale (LSHS; (Castiajo & Pinheiro, 2017; Launay & Slade, 1981)) (LSHS total scores: mean = 16.55, s.d. = 12.90, max = 57, min = 0; LSHS AVH scores [sum of items: “In the past, I have had the experience of hearing a person’s voice and then found no one was there”, “I often hear a voice speaking my thoughts aloud”, and “I have been troubled by voices in my head”]: mean = 2.52, s.d. = 3.14, min = 0, max = 11). All participants provided their written informed consent. They either received financial compensation in vouchers (10 euros per hour) or study credits (1 credit per hour) for their participation. All participants self-reported normal or corrected-to-normal visual acuity and normal hearing. The study was approved by the Medical Ethics Review Committee of the azM and Maastricht University (METC azM/UM) and conducted in accordance with the Declaration of Helsinki (METC 20-035; the study was prematurely terminated due to recruitment difficulties concerning voice hearers).

### *Procedure*

All participants went through three testing sessions.

## Session 1: Screening and neuropsychological assessment

The first session consisted of a diagnostic interview (the Mini International Neuropsychiatric Interview)(Lecrubier et al., 1997) and a detailed neuropsychological assessment of voice-hearing (including LSHS). This was done to differentiate non-voice hearers, non-clinical voice hearers, and voice hearers with a psychotic disorder.

## Session 2: Voice recording and stimulus generation

Participants comfortably sat inside an acoustically isolated chamber with the recording equipment (SENNHEISER K6/ME64 condenser microphone coupled with the M-track Eight as the audio interface), while the researcher sat outside this chamber. Recordings were made using a microphone with the Praat software (<https://www.praat.org/>). Participants were instructed to repeatedly vocalize “ah” in a neutral (i.e., without emotion) and in an angry voice for approximately 500 ms. They were provided with examples to familiarize them with the target duration of the vocalization. This specific duration was chosen to properly capture the emotionality while maintaining self-voice recognition. A vowel was chosen (instead of words) to eliminate semantic content processing (Cook & Wilding, 2001; Schweinberger et al., 1997; Ventura, Nagarajan, et al., 2009). The best voice sample was selected if the participants confirmed that (i) they recognized their recorded voice, (ii) the anger intensity was the highest that they could produce, (iii) they perceived no emotion in the neutral recording, and (iv) if the vocalization was pronounced clearly. Background noise was eliminated from the recordings using Audacity software (<https://audacityteam.org/>), and a Praat script was applied to normalize the intensity at 70 dB. The duration of the final neutral and angry “ah” vocalizations for each participant was 500 ms.

## *Morphing*

To create voice samples with varying degrees of emotional content, the pre-recorded neutral and angry self-voices for each individual participant were parametrically morphed to create a neutral-to-angry continuum. These continua consisted of 11 stimuli with 10% stepwise increase (neutral-to-angry) in emotional content along the

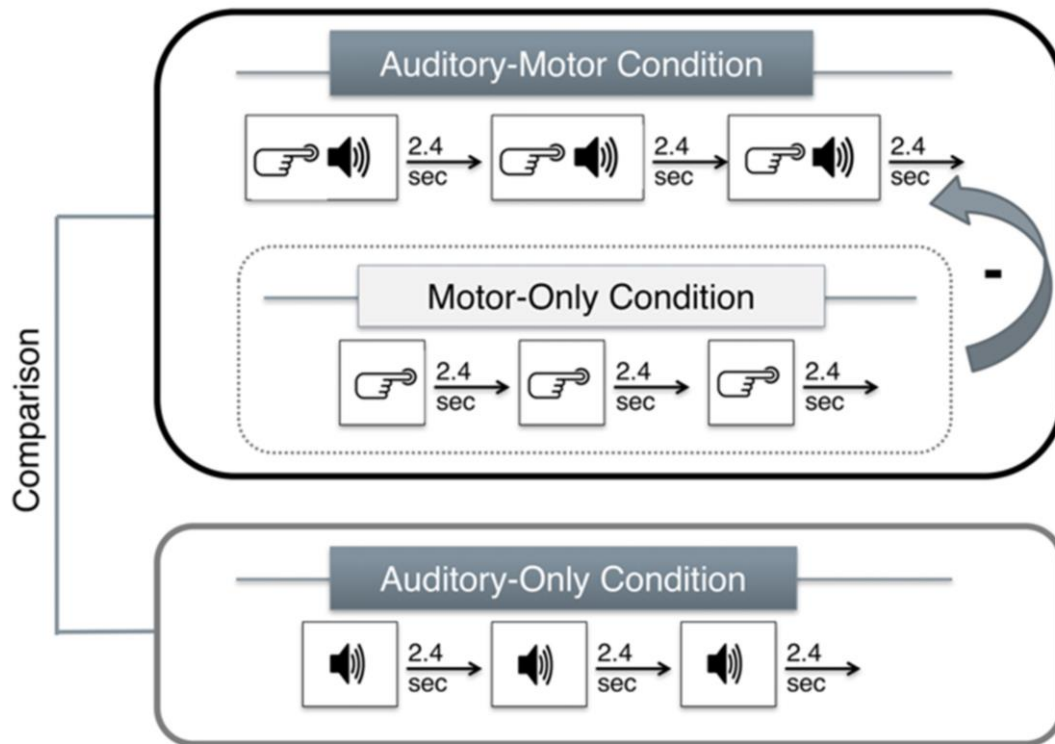
continuum (supplementary table 1). Morphing was performed using TANDEM-STRAIGHT software (Kawahara, 2006; Kawahara & Irino, 2005; Kawahara et al., 2008) running on MATLAB (R2019a, v9.6.0.1072779, The MathWorks, Inc., Natick, MA). For the final EEG experiments, 100% neutral, 60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry and 100% angry voice morphs were selected. The intermediate voice morphs were selected based on pilot data revealing that a maximum of uncertainty to differentiate the neutral from a somewhat angry voice fell in the range of 35-65% morphing. The increase in emotional voice quality (as self-voice changes from fully neutral to fully angry) and manipulations of certainty (as the self-voice quality changes from most certain to somewhat ambiguous to certain again; most certain: 100% neutral, uncertain/ambiguous: 60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry; certain: 100% angry self-voice morphs) allowed probing changes in certainty of sensory voice feedback and attentional control resulting from these changes.

### Session 3: EEG recordings

The third session comprised the EEG recordings. Participants were given an overview of the procedure and the principles of EEG at the start of the session. A previously employed auditory-motor task was used to investigate differences in self- and externally-generated auditory stimuli (Pinheiro et al., 2018) (figure 1). Previous studies have consistently shown an N100 amplitude suppression for sounds generated by a button-press, in contrast to those generated externally (Baess et al., 2009; Hughes et al., 2013; Knolle et al., 2012; Knolle et al., 2013a; Knolle et al., 2019), meaning that button-presses can be reliably used as a motor-act to self-generate a stimulus.

**Figure 1:** Motor-Auditory Task (Pinheiro et al., 2018). The task included three conditions: a motor-auditory (MA) condition, where participants pressed a button to generate a pre-recorded auditory stimulus representing a self-generated stimulus; an auditory-only (AO) condition, where participants passively listened to the pre-recorded auditory stimulus representing an externally-generated stimulus; and a motor-only (MO) condition, wherein they pressed a button but did not hear anything.

This last condition was used to control motor activity resulting from the button-press in the MA condition (MA-MO = MA corrected [MAc]).



The paradigm was presented in a mixed design and consisted of 5 runs in total (supplementary figure 1). All conditions (MA, AO, and MO) were blocked and 5 types of stimuli were randomly presented within the MA and AO blocks. Four of these 5 runs consisted of 2 sections corresponding to MA, followed by AO. Each of these sections consisted of 10 blocks corresponding to right and left hand button presses. Each block consisted of 10 trials. Five types of voice morphs belonging to “ah” vocalizations, respectively, were presented during MA and AO conditions. Overall, this resulted in 80 trials for each stimulus type. Trial durations from the MA section were used to present the voices in the AO section within each run. Therefore, each trial duration varied according to the button press. Prior to the experimental runs, participants were trained to press the button approximately every 2.4 seconds for the MA condition consisting of 100 trials. They received feedback during the training to adjust their tapping speed. Training concluded only if they performed correct taps in 75% of the trials (Pinheiro et al., 2018). No feedback was provided during the experimental

blocks. Participants could take small breaks after each run. The task was programmed and presented using the Presentation software (version 18.3; Neurobehavioral Systems, Inc.). Stimuli were presented via in-ear inserts. Button presses were recorded via the spacebar button on the keyboard.

At the end of the EEG session, participants were asked to rate the auditory stimuli for arousal and valence (supplementary figure 2). In addition, they were asked to rate the stimuli on perceived ownness, meaning perceived certainty of their own voice. This was done to ensure that they recognized their own voices and perceived the respective emotions.

#### *EEG data acquisition and processing*

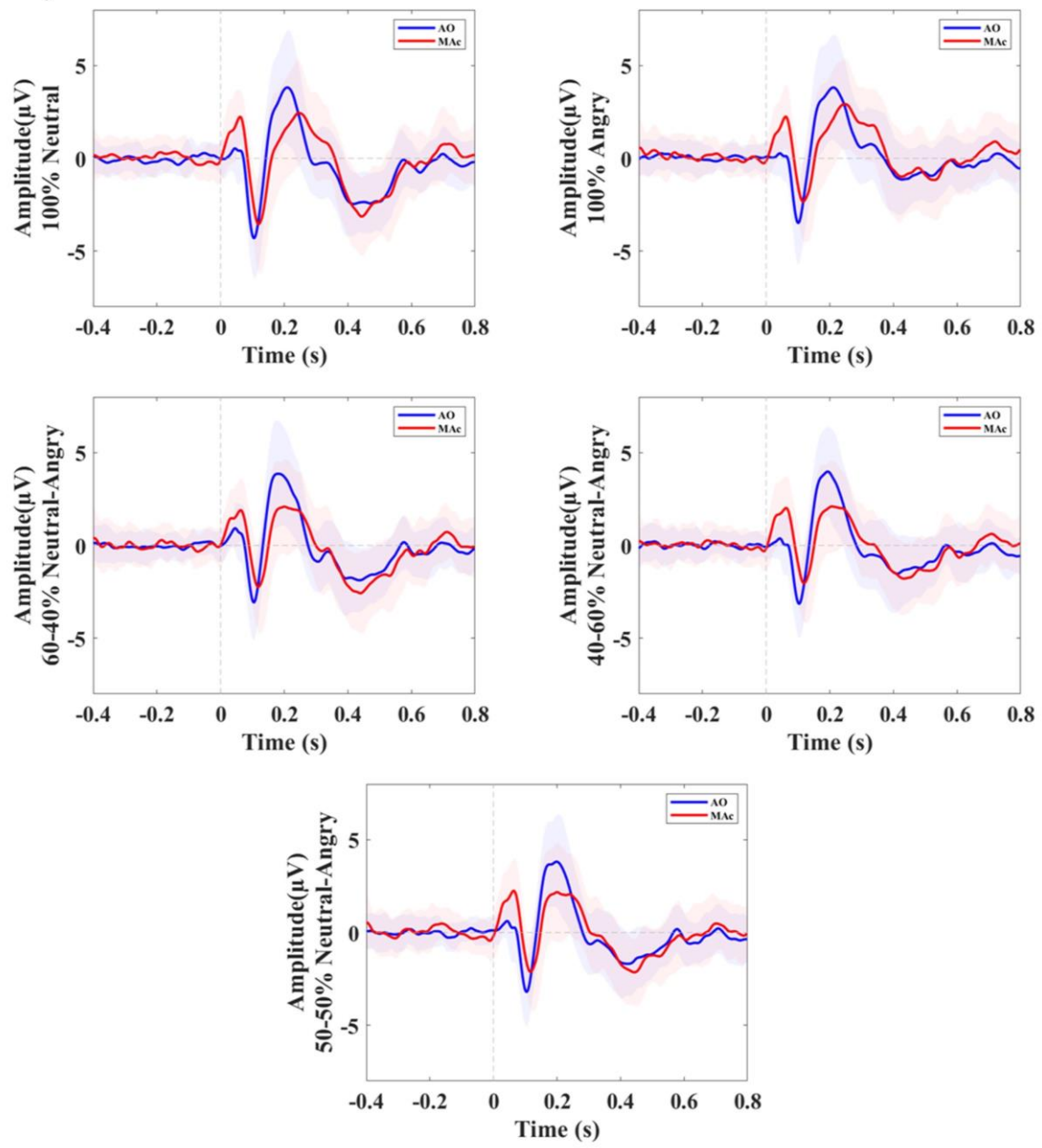
EEG data were recorded in an acoustically and electrically shielded room with BrainVision Recorder (Brain Products, Munich, Germany) using an ActiChamp 128-channel active electrode set-up while participants performed the task. Data were acquired at a sampling frequency of 1000Hz, electrode impedance was kept below 10 k $\Omega$ , and with FCz as online reference. During the EEG recording, participants were seated in a comfortable chair about 100 cm away from a screen in front of them.

EEG data were pre-processed using the MATLAB 2019a based toolbox Letswave 6 (<https://github.com/NOCIONS/letswave6>). Data were first cleaned to remove any repeated blocks/runs of the EEG recordings (e.g., due to hardware issues with the ear inserts leading to noise in the auditory stimuli or complete absence of audition), and trials within the time range of 800-2400 ms were processed further. Data were then downsampled to 500 Hz and bandpass filtered (0.5-30 Hz). All channels were re-referenced to the average of the four (TP7, TP8, TP9, TP10) mastoid electrodes. Noise related to eye blinks and movements, and noisy electrodes were removed using independent component analysis (ICA) with the runica algorithm in combination with principal component analysis (PICA; Rajan and Rayner as implemented in letswave 6). ICs representing the above mentioned noise were removed for each participant based on the IC time course and topography. The resulting data were segmented using a time-window of -400 to 800 ms relative to the onset of the auditory stimuli. The segmented data were baseline corrected to a window of -200 to -0 ms relative to the

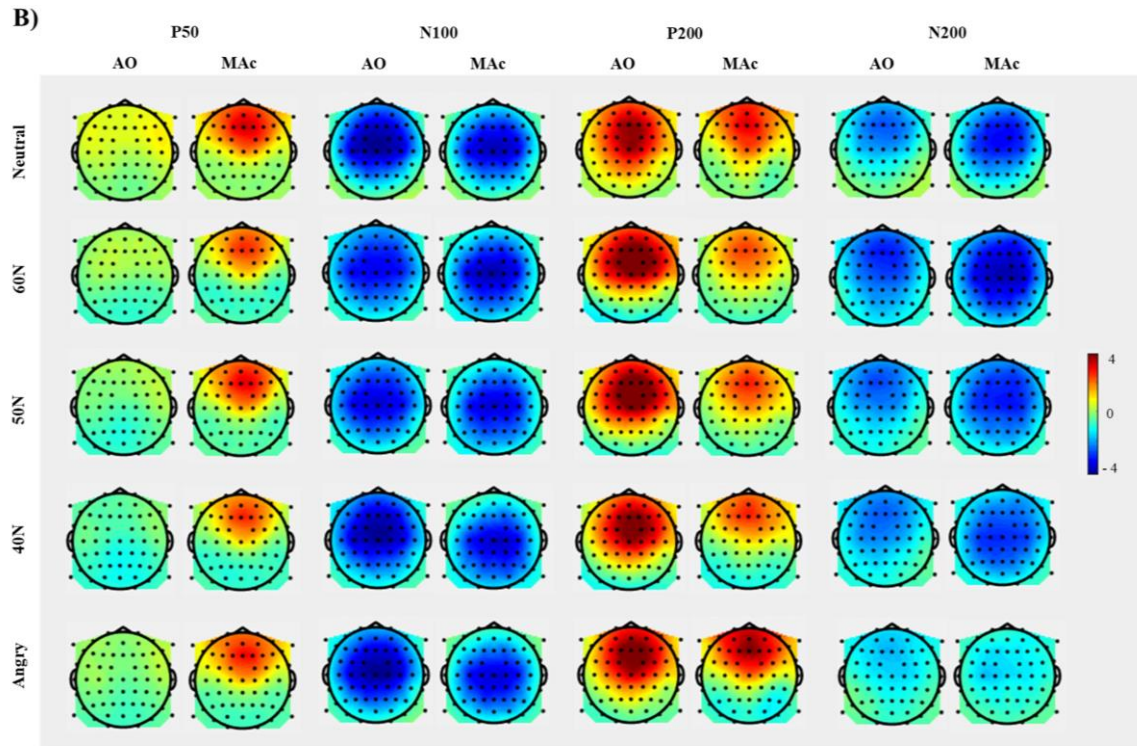
onset of the auditory stimuli. After baseline correction, an automatic artifact rejection algorithm was applied with an amplitude criterion of  $\pm 65\mu\text{V}$  to remove epochs/trials with remaining artefacts. The resulting data were then averaged for each participant, for each condition. The grand average waveforms revealed four distinct ERP components (figure 2), two negative ones peaking at approximately 110 ms and 450 ms respectively and two positive ones peaking at 55 ms and 200 ms approximately. Mean amplitudes (P50: 0.035-0.075, range = 40 ms, N100: 0.08-0.14, range = 60 ms; P200: 0.16-0.24, range = 80 ms; N200: 0.39-0.49, range = 100 ms) extracted from one fronto-central region of interest (Behroozmand et al., 2009; Chen et al., 2012; Folstein & Van Petten, 2008; Knolle et al., 2013a; Timm, SanMiguel, Saupe, & Schroger, 2013) ROI with 20 electrodes: AFF1h, AFF2h, F1, Fz, F2, FFC3h, FFC1h, FFC2h, FFC4h, FC3, FC1, FC2, FC4, FCC3h, FCC1h, FCC2h, FCC4h, C1, Cz, C2) were chosen as the outcome measure (figure 2).

**Figure 2:** A) Grand average waveforms  $\pm$  variance comparing self-generated and externally-generated for five types of self-voice originating from a fronto-central ROI. B) Topographic maps show voltage distribution in the ERP time window. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected

A)







### *Statistical analyses*

Statistical analyses of the ERP data were performed in R version 4.2.2 (2022-10-31) Copyright (C) 2022, using linear mixed models (LMMs) using the lmer and lmerTest packages (Bates, 2016; Kuznetsova et al., 2017). LMMs were used to control for random effects of participants influencing the outcome measure. Additionally, as HP measured by the LSHS is a continuous variable, LMMs were considered more appropriate than classical ANOVA to analyze the impact of HP on sensory feedback (condition) and emotional quality (stimulus type). Mean amplitude values of the ERPs were used as outcome measures, participants were used as random effects, and condition (2 levels: MAc and AO), stimulus type (5 levels: 100% neutral, 60-40% neutral-angry, 50-50% neutral-angry, 40-60% neutral-angry, 100% angry) and LSHS total or LSHS AVH (supplementary document A3) scores (continuous variable) were included as fixed effects, respectively, in the hypothesized models. For all models, the Gaussian distribution of model residuals and quantile-quantile plots confirmed their respective adequacy.

## **3 Results**

Following our hypotheses, we probed the interaction of sensory feedback processing (conditions: MAc and AO), self-voice quality (stimulus type: 100% neutral, 60-40%: neutral-angry; 50-50%: neutral-angry; 40-60%: neutral-angry and 100% angry), and HP for each ERP. However, we did not find a significant interaction of sensory feedback processing and self-voice quality, therefore we reduced the models to probing the interaction of sensory feedback processing and self-voice quality with HP, respectively.

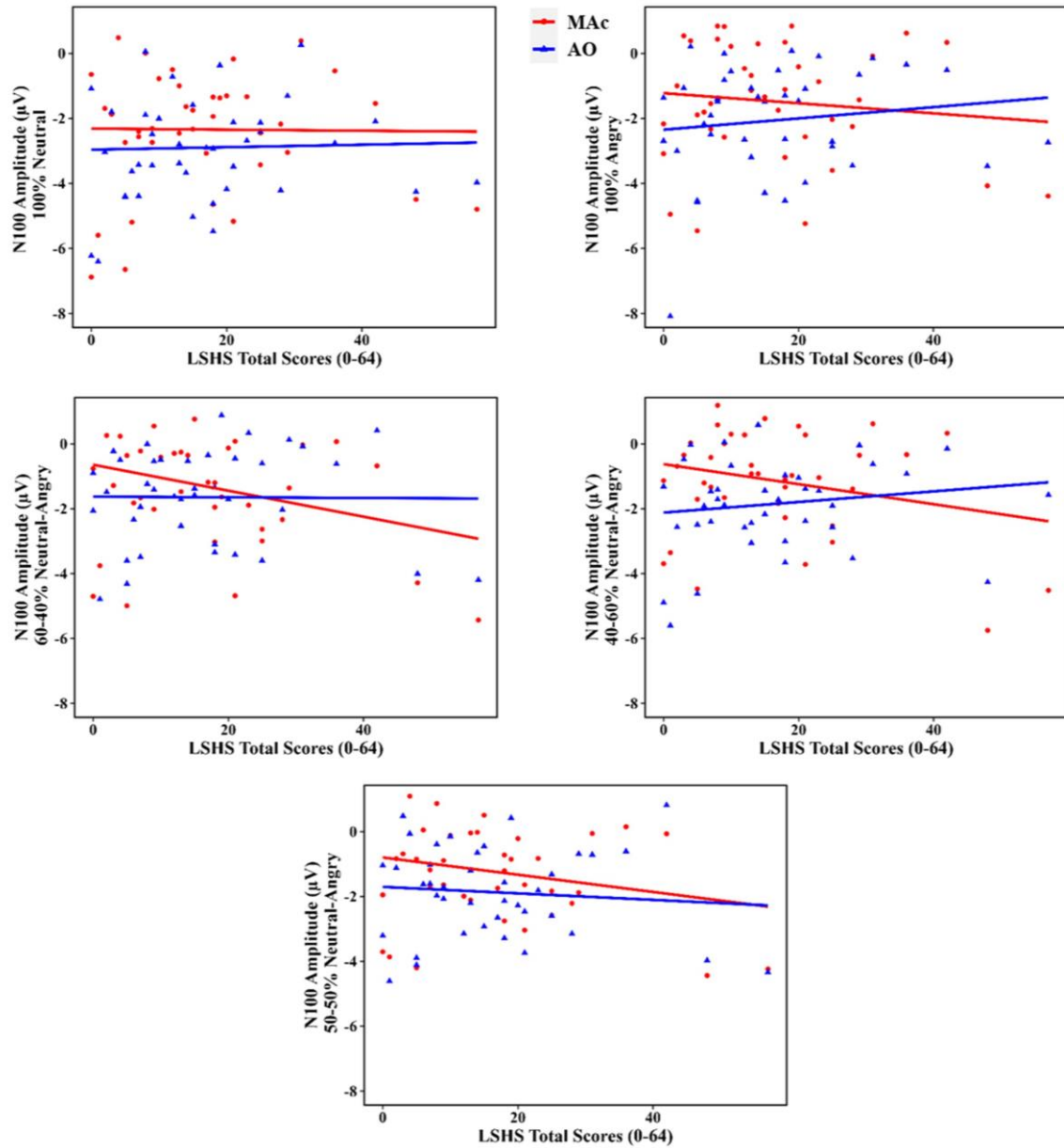
*N100 (primary outcome)*: To probe the influence of HP based on LSHS total scores on condition and stimulus type, we tested [m1\_N100 <- lmer(N100 ~ + LSHS total \* Condition + LSHS\_total\* Stimulus Type + (1|ID), data=data, REML = FALSE) ] against the null model [m0\_N100 <- lmer(N100 ~ + (1|ID), data=data, REML = FALSE); AIC = 1356.5], which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 110.5$ ,  $p = 0.000$ ; AIC = 1268.0) (table 1, figure 3). We report a significant difference between self-generated (MAc) and externally-generated (AO) self-voices, regardless of the stimulus quality and HP. Moreover, the N100 suppression effect (AO minus MAc) decreased as HP increased.

**Table 1:** The linear mixed effects model of the N100 including the effect of HP based on LSHS total scores. Abbreviation: AO = Auditory Only; SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 360.00$  (except Intercept:  $df = 64.18$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-2.121e+00	3.877e-01	-5.471	7.88e-07 ***
AO	-1.031e+00	1.603e-01	-6.433	3.98e-10 ***
LSHS total	-1.301e-02	1.847e-02	-0.704	0.483998
60N	1.502e+00	2.535e-01	5.926	7.27e-09 ***
50N	1.387e+00	2.535e-01	5.473	8.32e-08 ***

40N	1.265e+00	2.535e-01	4.992	9.32e-07 ***
Angry	8.526e-01	2.535e-01	3.364	0.000852 ***
AO*LSHS total	2.823e-02	7.640e-03	3.695	0.000254 ***
60N*LSHS total	-2.161e-02	1.208e-02	-1.789	0.074404 .
50N*LSHS total	-1.938e-02	1.208e-02	-1.605	0.109467
40N*LSHS total	-8.364e-03	1.208e-02	-0.692	0.489133
Angry*LSHS total	-2.002e-04	1.208e-02	-0.017	0.986783
<b>Groups</b>	<b>Name</b>	<b>Variance</b>	<b>SD</b>	
<b>Random Effects</b>				
Subjects	Intercept	1.6894	1.2998	
Residual		0.9714	0.9856	
Number of observations: 400, Subjects: 40				

**Figure 3:** Scatter plots depicting the change in N100 amplitudes as a function of HP based on LSHS total scores for each stimulus type. The N100 suppression effect - difference in the N100 amplitude between AO and MAc, either decreased or reversed with increase in HP scores. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected



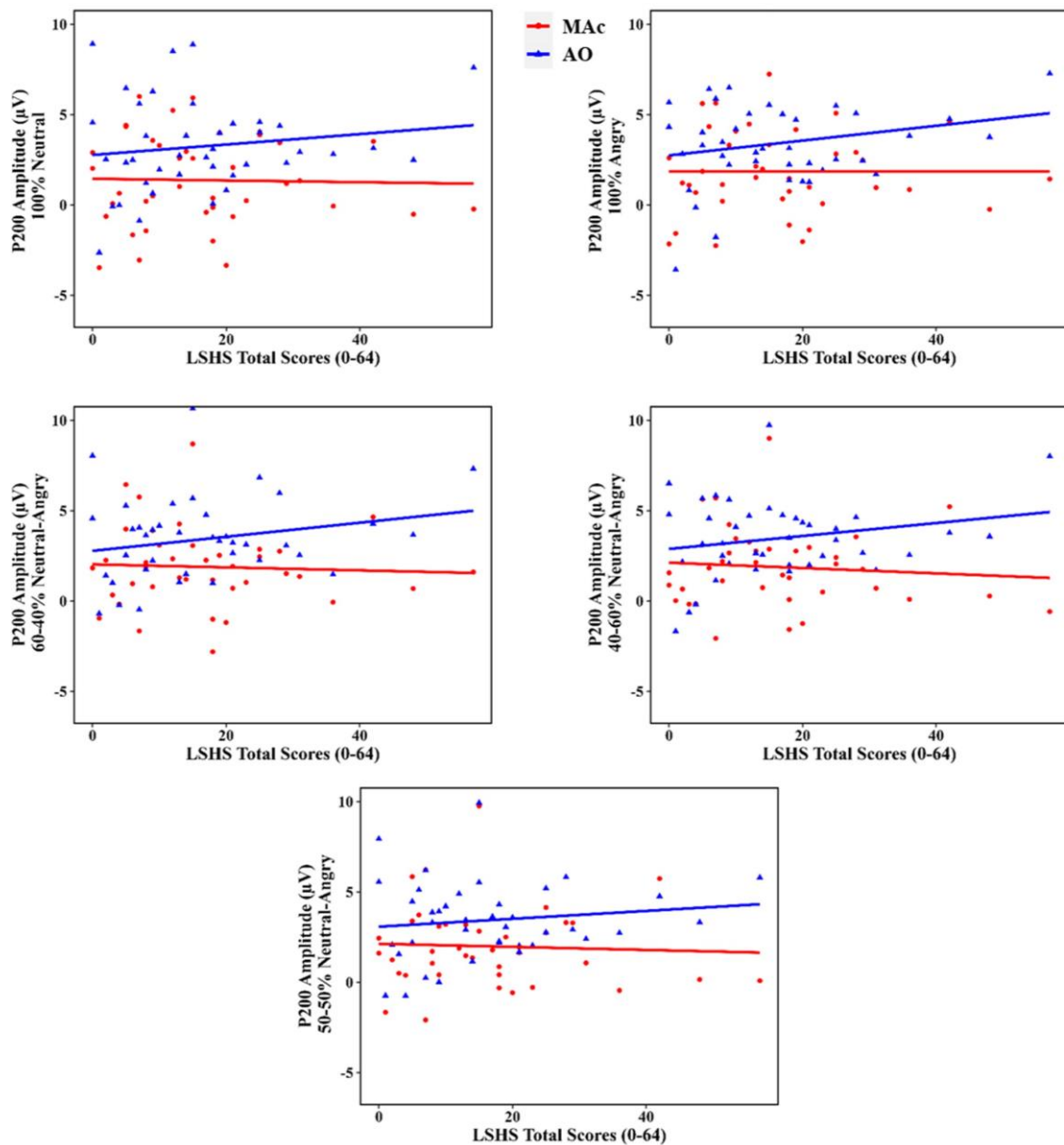
*P200 (secondary outcome):* To probe the influence of HP based on LSHS total scores on condition and stimulus type, we tested [m1\_P200 <- lmer(P200 ~ + LSHS total \* Condition + LSHS\_total\* Stimulus Type + (1|ID), data=data, REML = FALSE) ] against the null model [m0\_P200 <- lmer(P200 ~ + (1|ID), data=data, REML = FALSE); AIC = 1607.2], which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 147.82, p = 0.000$ ; AIC = 1481.3) (table 2, figure 4). We report a significant difference between self-generated (MAC) and externally-generated (AO) self-voices,

regardless of the stimulus quality and HP. Moreover, the P200 suppression effect (AO minus MAc) increased as HP levels increased.

**Table 2:** The linear mixed effects model of the P200, including the effect of HP based on LSHS total scores. Abbreviation: AO = Auditory Only; SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 360.00$  (except Intercept:  $df = 58.98$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	1.643231	0.547489	3.001	0.00394 **
AO	0.933481	0.206973	4.510	8.78e-06 ***
LSHS total	-0.008218	0.026090	-0.315	0.75389
60N	0.295346	0.327254	0.902	0.36740
50N	0.497875	0.327254	1.521	0.12904
40N	0.394437	0.327254	1.205	0.22888
Angry	0.187815	0.327254	0.574	0.56638
AO*LSHS total	0.040584	0.009863	4.115	4.81e-05 ***
60N*LSHS total	0.003236	0.015595	0.208	0.83574
50N*LSHS total	-0.005438	0.015595	-0.349	0.72753
40N*LSHS total	-0.001537	0.015595	-0.099	0.92153
Angry*LSHS total	0.008415	0.015595	0.540	0.58983
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	3.560	1.887	
Residual		1.619	1.272	
Number of observations: 400, Subjects: 40				

**Figure 4:** Scatter plots depicting the change in P200 amplitude as a function of HP based on LSHS total scores for each stimulus type. The P200 suppression effect - the difference in the amplitudes of AO and MAc, was modulated by HP such that the P200 amplitude for externally-generated (AO) voices increased with increase in HP scores. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected



*Exploratory analysis*

*P50*: To probe the influence of HP (based on LSHS total scores) on condition and stimulus type, we tested [m1\_P50 <- lmer(P50 ~ + LSHS total \* Condition + LSHS\_total\* Stimulus Type + (1|ID), data=data, REML = FALSE) ] against the null model [m0\_P50 <- lmer(P50 ~ + (1|ID), data=data, REML = FALSE); AIC = 1423.0], which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 170.4$ ,  $p = 0.000$ ; AIC = 1274.6) (table 3, figure 5). Regardless of HP and stimulus type, there is a significant difference in the P50 response between self-generated (MAc) and externally-generated (AO) conditions. Further, the P50 suppression effect increased with increase in HP.

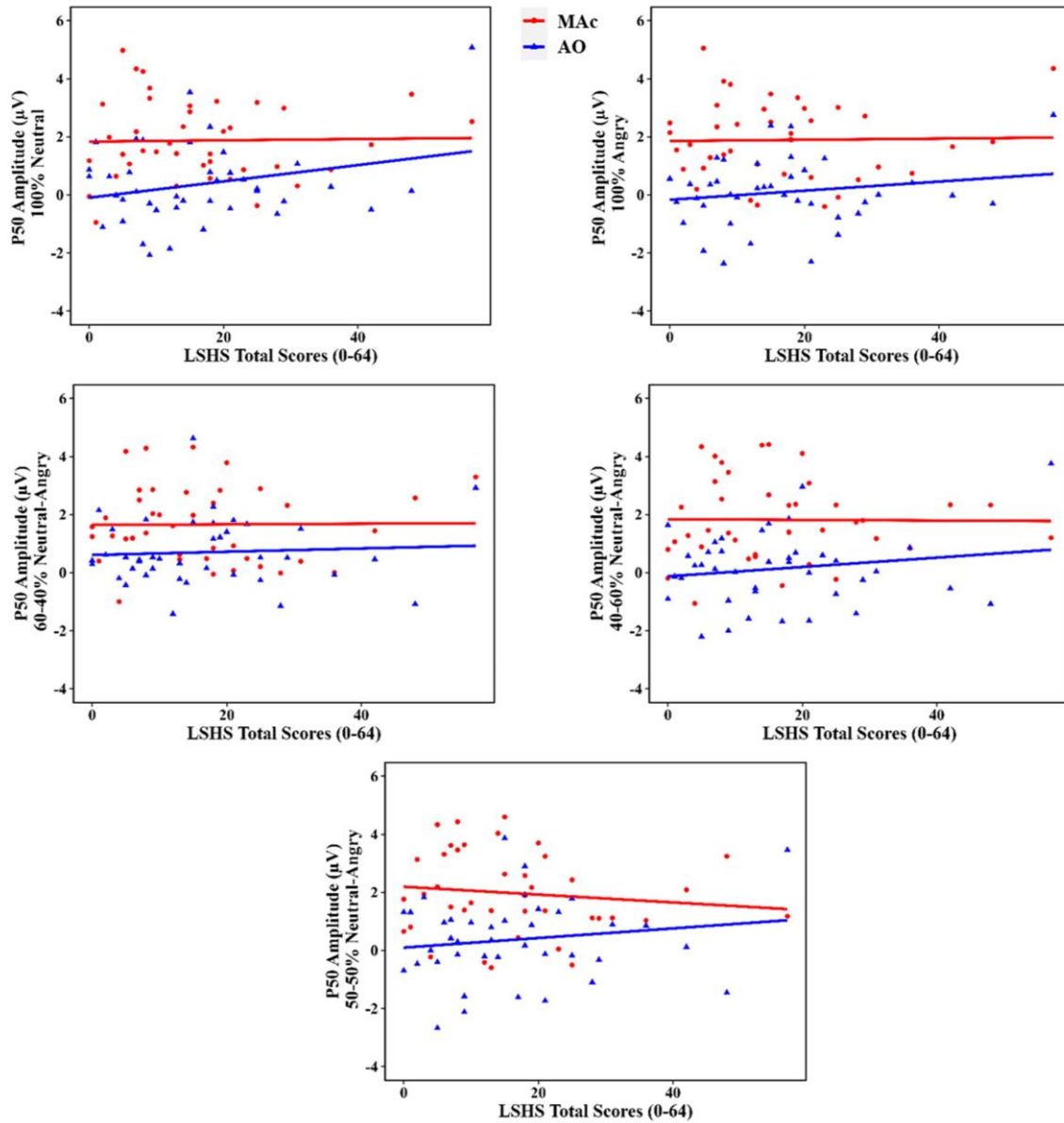
**Table 3:** The linear mixed effects model of the P50 including the effect of HP based on LSHS total scores. Abbreviations: AO = Auditory Only; SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 360.00$  (except Intercept:  $df = 116.24$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	1.774297	0.289666	6.125	1.27e-08 ***
AO	-1.810581	0.170145	-10.641	< 2e-16 ***
LSHS total	0.006003	0.013804	0.435	0.664
60N	0.256733	0.269023	0.954	0.341
50N	0.275914	0.269023	1.026	0.306
40N	-0.012501	0.269023	-0.046	0.963
Angry	-0.025393	0.269023	-0.094	0.925
AO*LSHS total	0.018250	0.008108	2.251	0.025 *
60N*LSHS total	-0.011790	0.012820	-0.920	0.358
50N*LSHS total	-0.013612	0.012820	-1.062	0.289
40N*LSHS total	-0.007643	0.012820	-0.596	0.551
Angry*LSHS total	-0.006264	0.012820	-0.489	0.625

Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	0.6121	0.7823	
Residual		1.0943	1.0461	
Number of observations: 400, Subjects: 40				

**Figure 5:** Scatter plots depicting the change in P50 as a function of HP (based on LSHS total scores) for each stimulus type. The P50 amplitude differences between AO and MAc decreased with increase in HP suggesting decrease in vigilance or sustained attention. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected





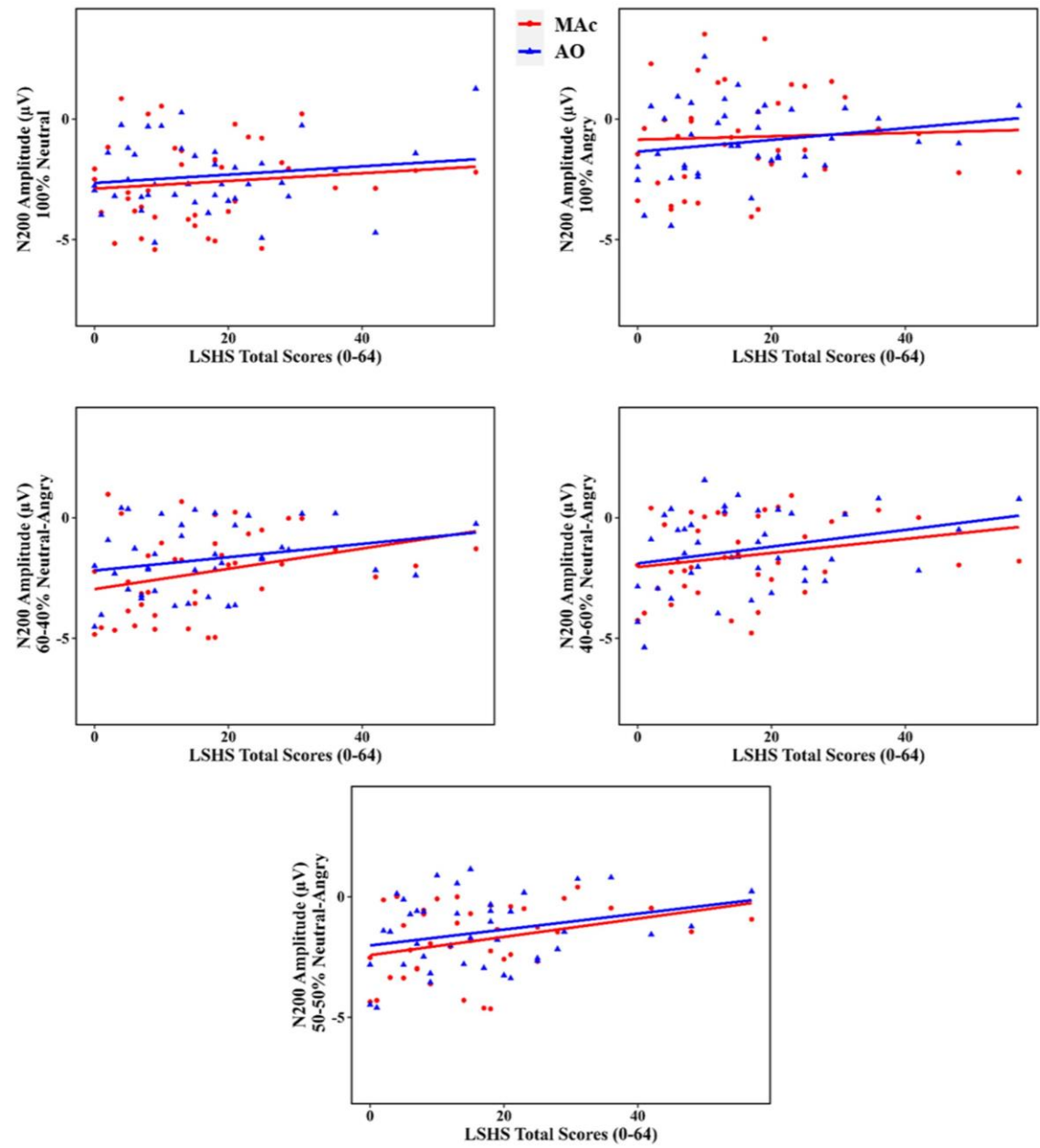
*N200*: To probe the influence of HP (based on LSHS total scores) on condition and stimulus type, we tested [m1\_N200 <- lmer(N200 ~ + LSHS total \* Condition + LSHS\_total\* Stimulus Type + (1|ID), data=data, REML = FALSE) ] against the null model [m0\_N200 <- lmer(P200 ~ + (1|ID), data=data, REML = FALSE); AIC = 1388.4], which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 106.66$ ,  $p = 0.000$ ; AIC = 1303.7) (table 4, figure 6). There was no significant interaction of HP with condition (Mac or AO) or stimulus type (5 types of self-voice) and no main effect of condition (Mac/AO). However, there was a main effect of stimulus type for 50-50% neutral-angry, 40-60% neutral-angry and 100% angry self-voice.

**Table 4:** The linear mixed effects model of the N200, including the effect of HP based on LSHS total scores. Abbreviations: AO = Auditory Only; SE = standard error; SD = standard deviation; \* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . Degrees of freedom for Fixed Effects:  $df = 360.00$  (except Intercept:  $df = 75.49$ ).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-2.874e+00	3.617e-01	-7.946	1.45e-11 ***
AO	2.116e-01	1.706e-01	1.241	0.2156
LSHS total	1.610e-02	1.724e-02	0.934	0.3531
60N	1.943e-01	2.697e-01	0.721	0.4716
50N	5.470e-01	2.697e-01	2.028	0.0433 *
40N	7.978e-01	2.697e-01	2.958	0.0033 **
Angry	1.663e+00	2.697e-01	6.165	1.89e-09 ***
AO*LSHS total	9.931e-04	8.128e-03	0.122	0.9028
60N*LSHS total	1.821e-02	1.285e-02	1.417	0.1573
50N*LSHS total	1.881e-02	1.285e-02	1.463	0.1443
40N*LSHS total	1.533e-02	1.285e-02	1.193	0.2337
Angry*LSHS total	-9.401e-04	1.285e-02	-0.073	0.9417
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	1.318	1.148	
Residual		1.100	1.049	
Number of observations: 400, Subjects: 40				

**Figure 6:** Scatter plots depicting the change in N200 amplitudes as a function of HP (based on LSHS total scores) for each stimulus type. No difference between the N200

amplitudes for AO and MAc. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected

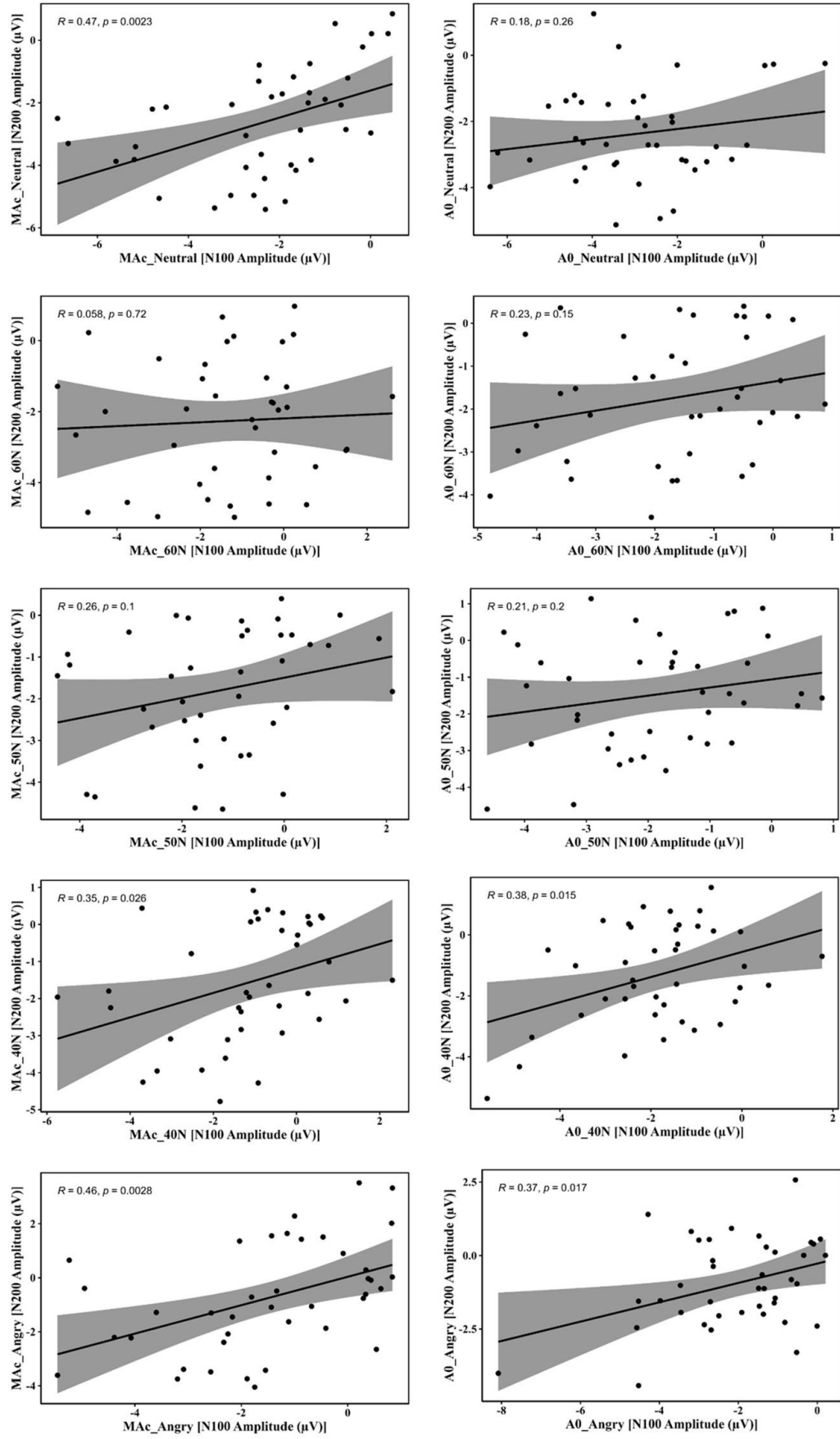


### *Correlational analysis*

If the N100 response is sensitive to discrepancies in sensory feedback processing and likely engages increased attentional resource allocation in self-voice production, we would expect corresponding changes at subsequent perceptual processing stages, like

the N200, associated with error awareness and attentional control (Dimoska, Johnstone, & Barry, 2006; Folstein & Van Petten, 2008; Hawco, Jones, Ferretti, & Keough, 2009; Kanske & Kotz, 2008). Therefore, to examine a potential relationship between N100 and N200, we correlated their amplitudes for each condition (AO and MAc) per stimulus type. Significant correlation between N100 and N200 mean amplitudes were observed for certain but not for uncertain (60-40% neutral-angry and 50-50% neutral-angry) self-voice processing.

**Figure 7:** Correlations between N100 and N200 mean amplitudes for MAc and AO per stimulus type. The significant correlations between N100 and N200 amplitudes for certain but not uncertain self-voice may suggest disengagement of, or reduced resource allocation for uncertain self-voices (60-40% neutral-angry and 50-50% neutral-angry), possibly due to perceiving them as dissimilar to one's own voice. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected



## 4 Discussion

The fundamental mechanisms that contribute to perceiving voices without any external sensory input remain inadequately understood. By varying the self-voice emotional quality, this study investigated whether changes in certainty of sensory feedback during self-voice production are linked to an individual's proneness to experience hallucinations and attentional allocation. The ERPs evoked by an individual's self- and externally-generated self-voice were examined within a classical auditory-motor paradigm (figure 1; (Pinheiro et al., 2018)). As expected, the N100 and P200 were suppressed for self- compared to externally-generated self-voices (table 1, 2; figure 2, 3, 4). Disengagement of resource allocation was observed for self-voice stimuli with ambiguous emotional quality, but not for those with unambiguous emotional quality (figure 7), suggesting that ambiguous self-voice stimuli did not recruit additional resources for further processing. HP modulated the P50, N100 and P200 suppression effects (table 1, 2, 3; figure 3, 4, 5), indicating increased error awareness and attention allocation in high HP individuals in self-voice processing likely stemming from alterations in sensory feedback processing, and/or attentional control.

### *Sensory feedback processing and attentional control*

Principally, the P50, N100 as well as P200 and N200 responses can be indicative of expected sound input albeit at different information processing stages (P50 and N100: sensory feedback processing, attention allocation (Boutros, Korzyukov, Jansen, Feingold, & Bell, 2004; Pinheiro, Schwartz, et al., 2019; Schroger et al., 2015; Schröger et al., 2015), P200: conscious distinction between self- and externally-generated voice (Knolle et al., 2012; Knolle et al., 2013a, 2013b; Knolle et al., 2019), N200: error awareness/monitoring and attentional control (Davies, Segalowitz, Dywan, & Pailing, 2001; Knolle et al., 2013b; Scheerer, Behich, Liu, & Jones, 2013b; Van Veen & Carter, 2002)). We report a more enhanced P50 response to self- than externally-generated voices, regardless of the self-voice quality or HP (table 3, figure 5). This is in contrast to previous findings that reported P50 suppression for self-generated tones (Pinheiro,

Schwartz et al., 2019). However, the P50 suppression was also associated with the decline in attention and vigilance over the course of the task (Baess et al., 2009; White & Yee, 2006). The enhanced P50 response to self-generated voices may likely reflect increased vigilance and sustained attention (White & Yee, 2006) towards the unexpected voice qualities of the self-generated voices. The self-generated condition preceded the externally-generated condition in the current task-design, which means that the novelty of unexpectedness of the self-voice remained higher in the self-generated than the passive listening condition (externally-generated voice). This interpretation is supported by studies reporting an association between the enhanced P50 and deviance detection and the encoding of irregularities in the auditory environment (Grimm & Escera, 2012). Of note, we report the opposite for the N100 response, i.e., reduced amplitudes for self- compared to the externally-generated voice, independent of the voice quality (neutral-angry) (figure 2; (Knolle et al., 2019; Pinheiro et al., 2018)). This suppression likely relates to the reduction of neural activity in the AC suggesting a match between expected and actual perceived sensory feedback of the self-voice. The explanation for the reversed P50 and N100 effects for the self-generated voice likely lies in the early time window (40-60 ms), which remains unaffected by expectation, while the later time window (100-200 ms) is impacted (Todorovic & de Lange, 2012). Further, we report a significant correlation between the N100 and N200 amplitudes for certain but not uncertain/ambiguous (60-40% neutral-angry, 50-50% neutral-angry) self-voices (figure 7). This may indicate reduced resource allocation and limited processing due to disengagement of attentional resources for non-self-relevant voices as these uncertain/ambiguous voices were not entirely perceived as either purely self-voice or entirely someone else's voice. Nonetheless, the lack of an interaction between condition and self-voice quality (based on N100 or N200 responses; table 1, table 4) and "own-ness" rating (supplementary figure 2) indicates the possibility that the differences between the uncertain/ambiguous and certain/unambiguous voices were still within the acceptable range of feasible physiological change in voice quality and thus might not have caused big enough sensory perturbations or mismatch between expected and actually perceived feedback.

*Hallucination proneness, sensory feedback processing, and attention allocation*

HP modulated P50, N100, and P200 effects. As HP (based on LSHS total scores) increased from low to high, the P50 response for self- compared to externally-generated voices decreased (table 3, figure 5). This reduction of the P50 response might reflect decreased vigilance and sustained attention due to complexity of sensory information, that is, the variation in emotional quality as well as recognition of self-voice (White & Yee, 2006). Individuals who score high in HP have been reported to show impaired filtering of information, thus might not be able to separate relevant from irrelevant stimuli, which leads to sensory overload (Clementz, Geyer, & Braff, 1998; Croft, Lee, Bertolot, & Gruzelier, 2001; Waldo, Adler, & Freedman, 1988; Yee et al., 2010). Similarly, the N100 suppression effect was reduced such that the N100 response for the self-generated voice is increased with increased HP (based on both LSHS total [table 1, figure 3] and AVH scores [see supplementary document]). This change in N100 suppression likely indicates that there is a mismatch between the expected and actual perceived sensory feedback in high HP individuals rooted in altered sensory feedback processing (Pinheiro et al., 2018; Pinheiro, Schwartz, & Kotz, 2020). The N100 response is also associated with spontaneous attention allocation (Schroger et al., 2015; Schröger et al., 2015; Schweizer et al., 2007). Attention and prediction show complementary effects on the N100 response: the N100 amplitude decreases with an increase in predictability whereas it increases when more attention needs to be allocated to an event (Schroger et al., 2015; Schröger et al., 2015). Therefore, an increased N100 amplitude in response to the self-generated voice with increased HP might also indicate altered error awareness and/or inability to suppress attention allocation to an irrelevant stimulus - one's own self-generated voice. In sum, individuals with high HP display altered sensory feedback processing for the self-voice and might misattribute attentional resources to it. Both accounts are associated with theories such as self-monitoring and salience misattribution explaining AVH (Johns & McGuire, 1999; Johns et al., 2001; Jones & Fernyhough, 2007; Kapur, 2003; Kapur, Mizrahi, et al., 2005).

The P200 response is associated with a more conscious detection of sensory feedback to a self-generated compared to an externally-generated stimulus (Behroozmand & Larson, 2011; Behroozmand, Liu, & Larson, 2011; Knolle et al., 2012; Knolle et al., 2013a, 2013b). The current findings also confirm an increase in the P200 suppression



effect with increased P200 response for externally-generated voices with an increase in HP (based on both LSHS total and AVH scores) (table 2; figure 4). The P200 response is reportedly more sensitive to temporal predictability of an auditory stimulus (Behroozmand, Sangtian, Korzyukov, & Larson, 2016; Chen et al., 2012; Pinheiro et al., 2018; Sowman et al., 2012; Timm, Schönwiesner, Schröger, & SanMiguel, 2016). Specifically, increased P200 responses were reported with longer delays between a button-press and sensory feedback, reflecting a potential decrease in the sense of agency when expectations are not matched immediately (Behroozmand et al., 2016; Chen et al., 2012; Pinheiro et al., 2018; Sowman et al., 2012). As the timing of the different self-voices were unpredictable in the externally- compared to the self-generated condition, an enhanced P200 response in high HP during passive listening to self-voice could merely reflect more mindful processing of the five types of voices with variable onsets, triggering more attentional resources than in low HP.

The current sample of participants consisted of non-voice hearers from the general population who varied in HP (N = 38) as well as 2 voice hearers with a psychotic disorder. Given the sample size calculations (supplementary document A2) and early termination of the study, the current had limited statistical power, which may additionally explain the absence of an observed three-way interaction between conditions (AO, MAc), voice quality (5 types of self-voice), and HP. Furthermore, because of the small number of voice hearers with a psychotic disorder we were unable fully to explore the putative psychosis continuum. Previous studies probing prediction of sensory feedback to the self-voice reported no N100 suppression effect in voice hearers with a psychotic disorder (Heinks-Maldonado et al., 2007; Heinks-Maldonado et al., 2006) whereas non-clinical voice hearers or high HP individuals showed a reversed N100 suppression effect (Duggirala et al., 2023; Pinheiro, Schwartze, Amorim, et al., 2020; Pinheiro et al., 2018). These studies either used group differences (Heinks-Maldonado et al., 2007) or hypothesized a linear relationship between HP and sensory feedback processing/attentional control, measured by the N100 suppression effect, using LMMs (Duggirala et al., 2023; Pinheiro et al., 2018). Future studies should consider that individuals on the postulated HP severity continuum (voice hearers with psychotic disorder, at high-risk of psychosis/prodromal and non-clinical voice hearers) may show a nonlinear

relationship with the N100 suppression effect. Future studies should also analyze the influence of HP on pre-button-press neural activity, including the readiness potential (Reznik, Simon, & Mukamel, 2018; Vercillo, O'Neil, & Jiang, 2018) and pre-stimulus alpha power in tasks manipulating emotional quality of voice feedback, which have both been related to the prediction of the sensory consequences of a self-generated neutral voice (Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018).

Using an established experimental paradigm in combination with a manipulation of the emotional quality of the self-voice, we detected associations between HP and electrophysiological indicators of sensory feedback during self-voice production. The findings contribute to a better understanding of the association between expected and actual perceived sensory feedback and its potential role in auditory hallucinations.

**Funding:** The current project was supported by internal funds.

**Data availability:** The data and analysis pipelines that support the findings of this study are available from the corresponding author upon reasonable request.

**Author Contributions:** SxD, MS, AP, DL, TvA, SK conceptualized the project. SxD, AP, MS, SK designed the experiment, SxD prepared materials, SxD and HH collected data, SxD analyzed the data, and wrote the first draft of the manuscript and SxD, HH, MS, TvA, DL, AP, SK edited and refined the manuscript. All authors have approved the final version of the manuscript.

**Competing Interests:** The authors declare that they have no competing interests.

**Acknowledgements:** The authors would like to thank our interns Alex Kalberer and Maren Cremer for their support with data collection.

## Supplementary Document

### Section A

**A1. Exclusion criteria:** Participants were excluded if they (i) had undergone any previous neurosurgery or had a neurological disorder, (ii) refused to participate in the EEG, (iii) were unable to fully comprehend the purpose of the study or to make a rational decision whether or not to participate, (iv) heard voices exclusively due to substance abuse (drug or alcohol addiction).

### **A2. Sample size calculation:**

As the primary objective was to examine N100 suppression effects (AO - MAc) as a function of hallucination proneness (HP), the total number of required participants was based on a power analysis for the correlation between the quantitative covariate (HP) and a linear contrast of the within subjects measure (e.g., N100 suppression effect for self-voice changing from fully neutral to angry in five steps) using G\*Power statistical software (3.1.9) (Faul et al., 2007) with a detectable effect size of  $(d) = 0.50$  resulting in  $\rho$  of 0.24,  $\alpha = 0.05$ , power  $(1-\beta) = 0.80$  and adjusting for confounding factors (e.g., age, education, gender, medication). Thus, the final sample size was 210 participants in total including voice hearers and participants from the general population. Based on previous literature, 7-13% of the general population experience auditory verbal hallucinations (AVH) (Beavan, Read, & Cartwright, 2011; Kråkvik et al., 2015; Linscott & van Os, 2013). Thus, 16-30 voice hearers (belonging to the three categories of non-voice hearers, non-clinical voice hearers and voice hearers with psychotic disorder) were recruited. Considering the lower prevalence of voice hearers in the general population, participants were recruited from the general population through advertisements in newspaper and social media, as well as voice hearing centers.

*t tests - Correlation: Point biserial model*

Analysis: A priori: Compute required sample size

Input: Tail(s) = Two

Effect size  $|\rho| = 0.24$

$\alpha$  err prob = 0.05

Power ( $1-\beta$  err prob) = 0.80

Output: Noncentrality parameter  $\delta = 2.8296274$

Critical t = 1.9785245

Df = 129

Total sample size = 131

Actual power = 0.8019318

After adjusting for the potentially confounding factors such as age, gender and education, the sample size is multiplied by 1.5 (variance inflation factor; VIF), 1.05 (to account for the power loss due to interim analysis) and approximately 10% drop out rate =  $(131 \times 1.5 \times 1.07) + 21 = 231$ .

The current study was concluded prematurely due to slow recruitment of voice hearers, leading to a final sample of 45 recruited participants.

### **A3. Results**

#### *HP based on LSHS AVH scores*

P50: To probe the influence of HP (based on LSHS AVH scores) on condition and stimulus type, we tested `[m1_P50 <- lmer(P50 ~ + LSHS AVH * Condition + LSHS AVH * Stimulus Type + (1|ID), data=data, REML = FALSE)]` against the null model `[m0_P50 <- lmer(P50 ~ + (1|ID), data=data, REML = FALSE); AIC = 1423.0]`, which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 167.77$ ,  $p = 0.000$ ;  $AIC = 1277.3$ ) (supplementary table 2 and figure 5).

N100: To probe the influence of HP (based on LSHS AVH scores) on condition and stimulus type, we tested `[m1_N100 <- lmer(N100 ~ + LSHS AVH * Condition + LSHS AVH * Stimulus Type + (1|ID), data=data, REML = FALSE)]` against the null model `[m0_N100 <- lmer(N100 ~ + (1|ID), data=data, REML = FALSE); AIC = 1356.5]`, which

showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 110.5$ ,  $p = 0.000$ ; AIC = 1268.0) (supplementary table 3 and figure 6).

P200: To probe the influence of HP (based on LSHS AVH scores) on condition and stimulus type, we tested [m1\_P200 <- lmer(P200 ~ + LSHS AVH \* Condition + LSHS AVH \* Stimulus Type + (1|ID), data=data, REML = FALSE) ] against the null model [m0\_P200 <- lmer(P200 ~ + (1|ID), data=data, REML = FALSE); AIC = 1607.2], which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 150.59$ ,  $p = 0.000$ ; AIC = 1478.6) (supplementary table 4 and figure 7).

N200: To probe the influence of HP (based on LSHS AVH scores) on condition and stimulus type, we tested [m1\_N200 <- lmer(N200 ~ + LSHS AVH \* Condition + LSHS\_AVH \* Stimulus Type + (1|ID), data=data, REML = FALSE) ] against the null model [m0\_N200 <- lmer(N200 ~ + (1|ID), data=data, REML = FALSE); AIC = 1388.4], which showed the best goodness of fit and yielded a significant difference ( $\chi^2(11) = 107.95$ ,  $p = 0.000$ ; AIC = 1302.5) (supplementary table 5, and figure 8).

## Section B

### Tables and table legends

**Supplementary table 1:** Neutral-angry continua with 11 voice morphs.

a) Neutral-to-angry

Emotion/ Morphs	1*	2	3	4	5*	6*	7*	8	9	10	11*
Neutral	<b>100%</b>	90%	80%	70%	<b>60%</b>	<b>50%</b>	<b>40%</b>	30%	20%	10%	<b>0%</b>
Angry	<b>0%</b>	10%	20%	30%	<b>40%</b>	<b>50%</b>	<b>60%</b>	70%	80%	90%	<b>100%</b>

\*final auditory stimuli

**Supplementary table 2:** Linear mixed effects model of P50 amplitude including the effect of HP based on LSHS AVH scores. Abbreviations: SE = standard error; SD = standard deviation; AO = auditory only; \*p < 0.05; \*\*p < 0.01; \*\*\*p < 0.001. Degrees of freedom for Fixed Effects: df = 360.00 (except Intercept: df = 118.89).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	1.786494	0.227496	7.853	2.01e-12 ***
AO	-1.637872	0.134772	-12.153	< 2e-16 ***
LSHS AVH	0.034515	0.056391	0.612	0.542
60N	0.137511	0.213094	0.645	0.519
50N	0.108717	0.213094	0.510	0.610
40N	-0.124078	0.213094	-0.582	0.561
Angry	-0.133966	0.213094	-0.629	0.530
AO*LSHS total	0.051218	0.033407	1.533	0.126
60N*LSHS total	-0.030061	0.052821	-0.569	0.570
50N*LSHS total	-0.023005	0.052821	-0.436	0.663

40N*LSHS total	-0.005906	0.052821	-0.112	0.911
Angry*LSHS total	0.001943	0.052821	0.037	0.971
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	0.5963	0.7722	
Residual		1.1048	1.0511	
Number of observations: 400, Subjects: 40				

**Supplementary table 3:** Linear mixed effects model of N100 amplitude including the effect of HP based on LSHS AVH scores. Abbreviations: SE = standard error; SD = standard deviation, AO = auditory only; \*p < 0.05; \*\*p < 0.01; \*\*\*p < 0.001. Degrees of freedom for Fixed Effects: df = 360.00 (except Intercept: df = 65.29).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-2.000372	0.302946	-6.603	8.52e-09 ***
AO	-0.842473	0.127317	-6.617	1.33e-10 ***
LSHS AVH	-0.133046	0.075094	-1.772	0.081107 .
60N	1.215397	0.201306	6.038	3.90e-09 ***
50N	1.083033	0.201306	5.380	1.34e-07 ***
40N	1.122067	0.201306	5.574	4.89e-08 ***
Angry	0.761286	0.201306	3.782	0.000182 ***
AO*LSHS AVH	0.110205	0.031559	3.492	0.000539 ***
60N*LSHS AVH	-0.028115	0.049900	-0.563	0.573486
50N*LSHS AVH	-0.006568	0.049900	-0.132	0.895359
40N*LSHS AVH	0.001936	0.049900	0.039	0.969067
Angry*LSHS AVH	0.034853	0.049900	0.698	0.485341

Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	1.641	1.281	
Residual		0.986	0.993	
Number of observations: 400, Subjects: 40				

**Supplementary table 4:** Linear mixed effects model of P200 amplitude including the effect of HP based on LSHS AVH scores. Abbreviations: SE = standard error; SD = standard deviation, AO = auditory only; \*p < 0.05; \*\*p < 0.01; \*\*\*p < 0.001. Degrees of freedom for Fixed Effects: df = 360.00 (except Intercept: df = 58.65).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	1.81249	0.43259	4.190	9.52e-05 ***
AO	1.16573	0.16246	7.175	4.15e-12 ***
LSHS AVH	-0.12090	0.10723	-1.127	0.264
60N	0.26002	0.25688	1.012	0.312
50N	0.32058	0.25688	1.248	0.213
40N	0.30127	0.25688	1.173	0.242
Angry	0.08711	0.25688	0.339	0.735
AO*LSHS AVH	0.17403	0.04027	4.321	2.01e-05 ***
60N*LSHS AVH	0.03520	0.06367	0.553	0.581
50N*LSHS AVH	0.03457	0.06367	0.543	0.587
40N*LSHS AVH	0.02682	0.06367	0.421	0.674
Angry*LSHS AVH	0.09504	0.06367	1.493	0.136
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	3.590	1.895	



Residual		1.605	1.267	
Number of observations: 400, Subjects: 40				

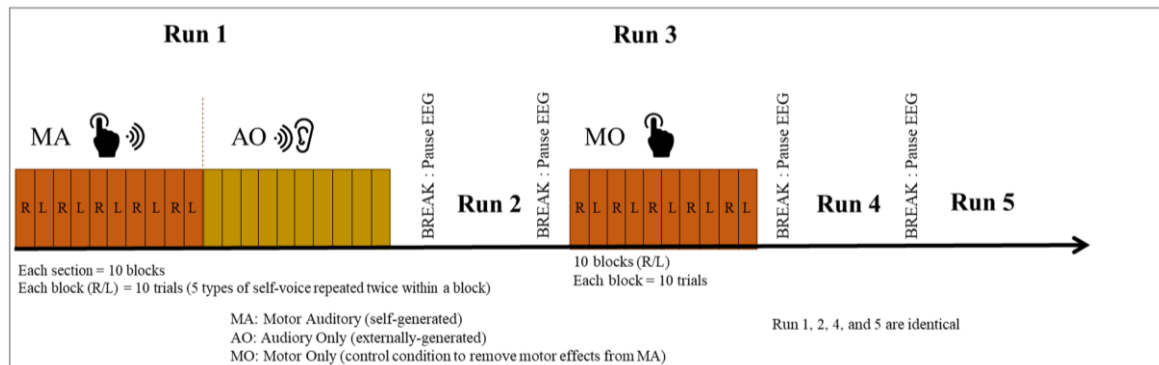
**Supplementary table 5:** Linear mixed effects model of N200 amplitude including the effect of HP based on LSHS AVH scores. Abbreviations: SE = standard error; SD = standard deviation, AO = auditory only; \*p < 0.05; \*\*p < 0.01; \*\*\*p < 0.001. Degrees of freedom for Fixed Effects: df = 360.00 (except Intercept: df = 72.86).

Variable	Estimate	SE	t value	Pr(> t )
<b>Fixed Effects</b>				
Intercept	-2.54473	0.29093	-8.747	5.64e-13 ***
AO	0.08674	0.13378	0.648	0.51718
LSHS AVH	-0.02490	0.07211	-0.345	0.73091
60N	0.24894	0.21153	1.177	0.24004
50N	0.63322	0.21153	2.994	0.00295 **
40N	0.87151	0.21153	4.120	4.70e-05 ***
Angry	1.60508	0.21153	7.588	2.81e-13 ***
AO*LSHS AVH	0.05596	0.03316	1.688	0.09237 .
60N*LSHS AVH	0.09777	0.05243	1.865	0.06306 .
50N*LSHS AVH	0.08913	0.05243	1.700	0.09003 .
40N*LSHS AVH	0.07129	0.05243	1.360	0.17481
Angry*LSHS AVH	0.01670	0.05243	0.318	0.75031
Groups	Name	Variance	SD	
<b>Random Effects</b>				
Subjects	Intercept	1.406	1.186	
Residual		1.089	1.043	
Number of observations: 400, Subjects: 40				

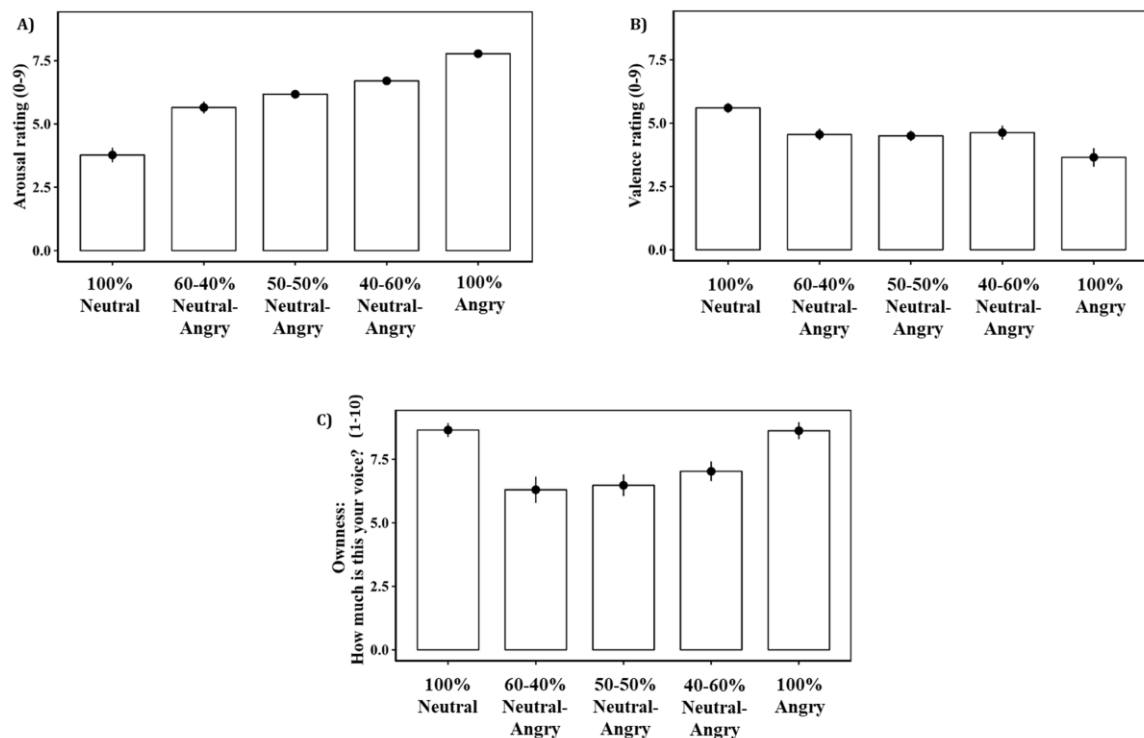
## Section C

### Figures and figure legends

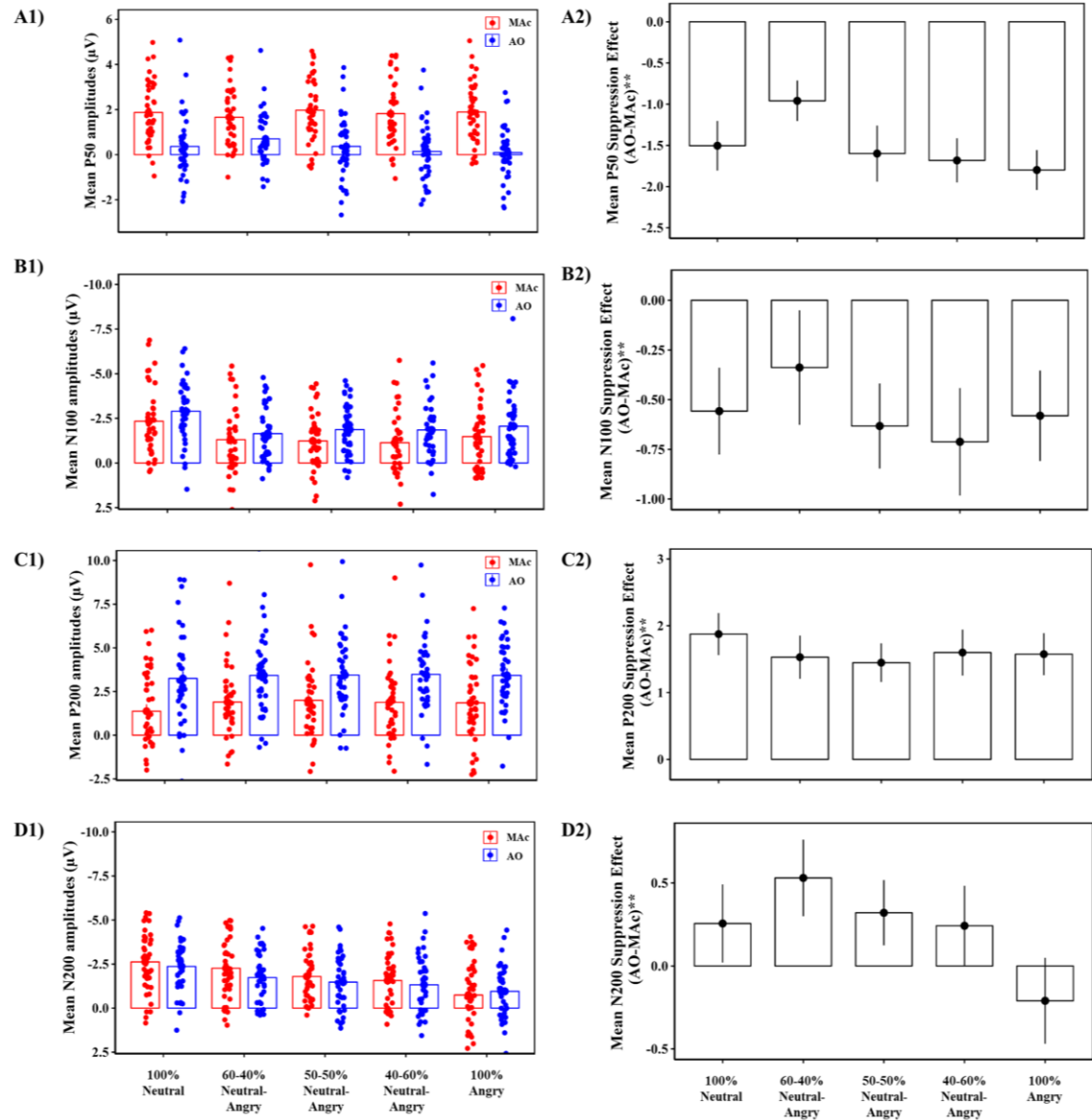
**Supplementary figure 1:** Schematic representing the paradigm design.



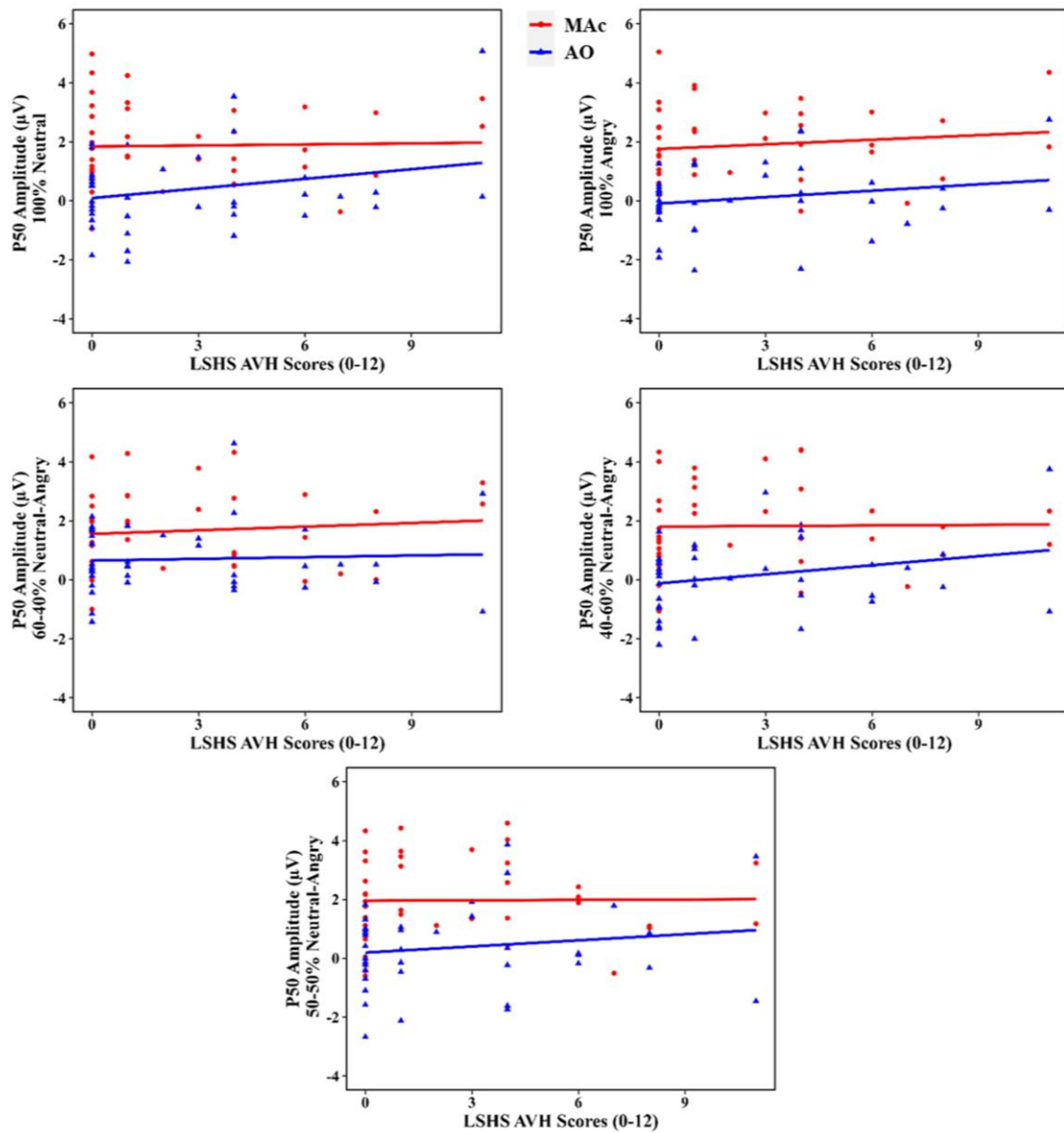
**Supplementary figure 2:** Post experiment stimuli rating. A) Arousal rating on a scale of 0-9 for each voice stimulus. B) Valence rating on a scale of 0-9 for each voice stimulus. C) Ownness rating on a scale of 1-10 for each voice stimulus.



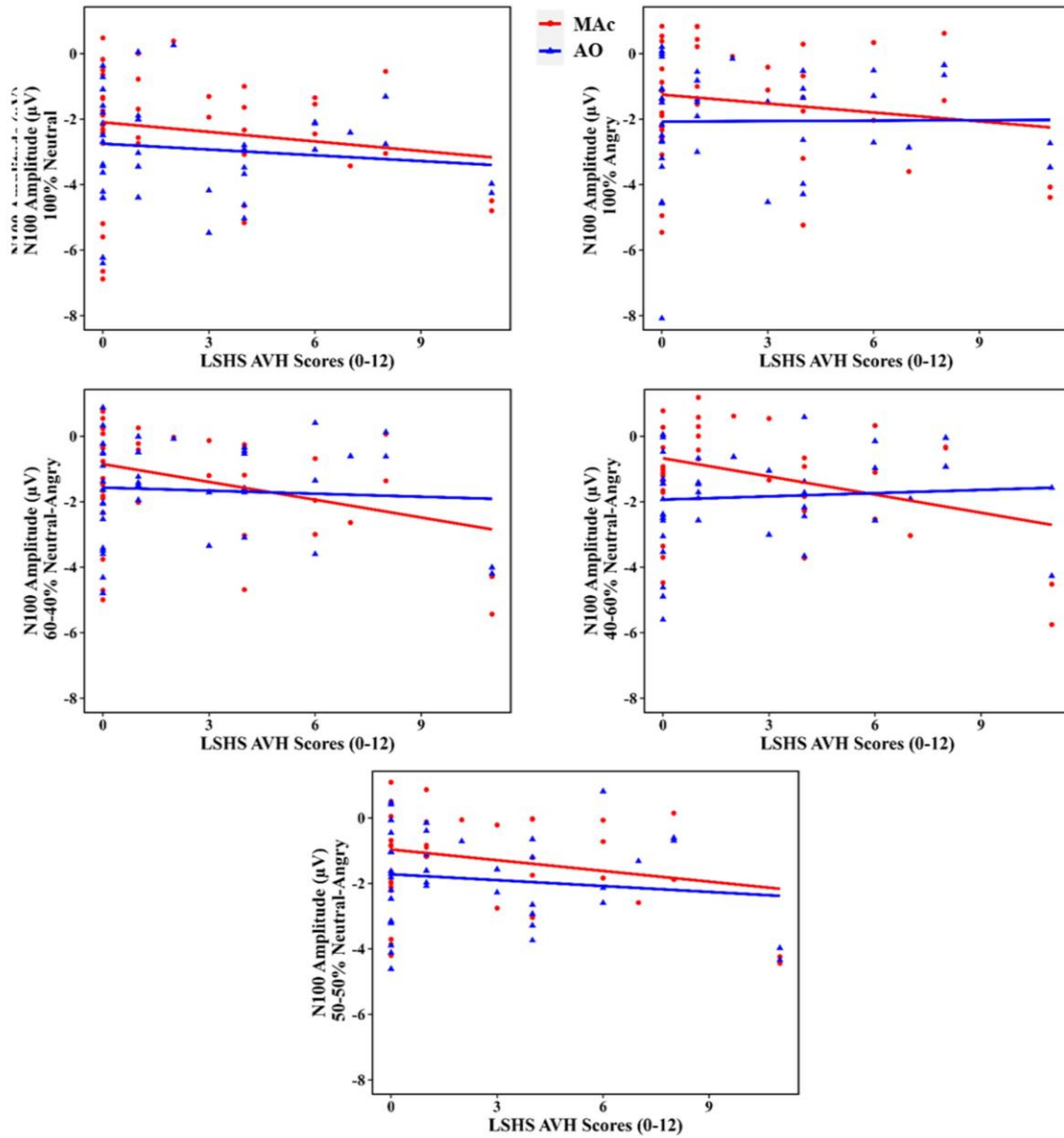
**Supplementary figure 3:** Mean ERP amplitudes for MAc and AO and suppression effects (AO - MAc) per stimulus. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected.



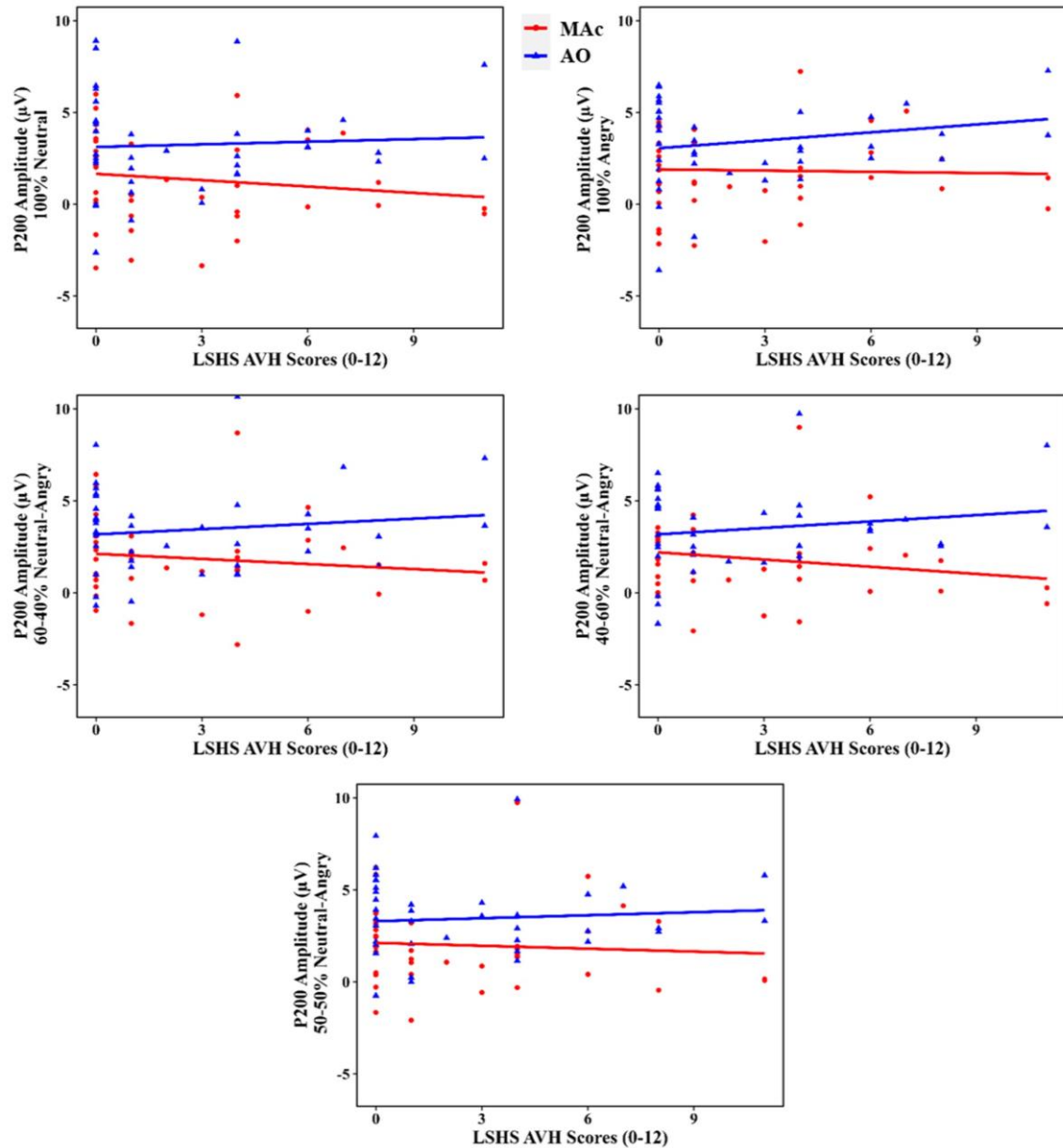
**Supplementary figure 4:** Scatter plots depicting the change in P50 amplitudes as a function of HP based on LSHS AVH scores for each stimulus type. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected.



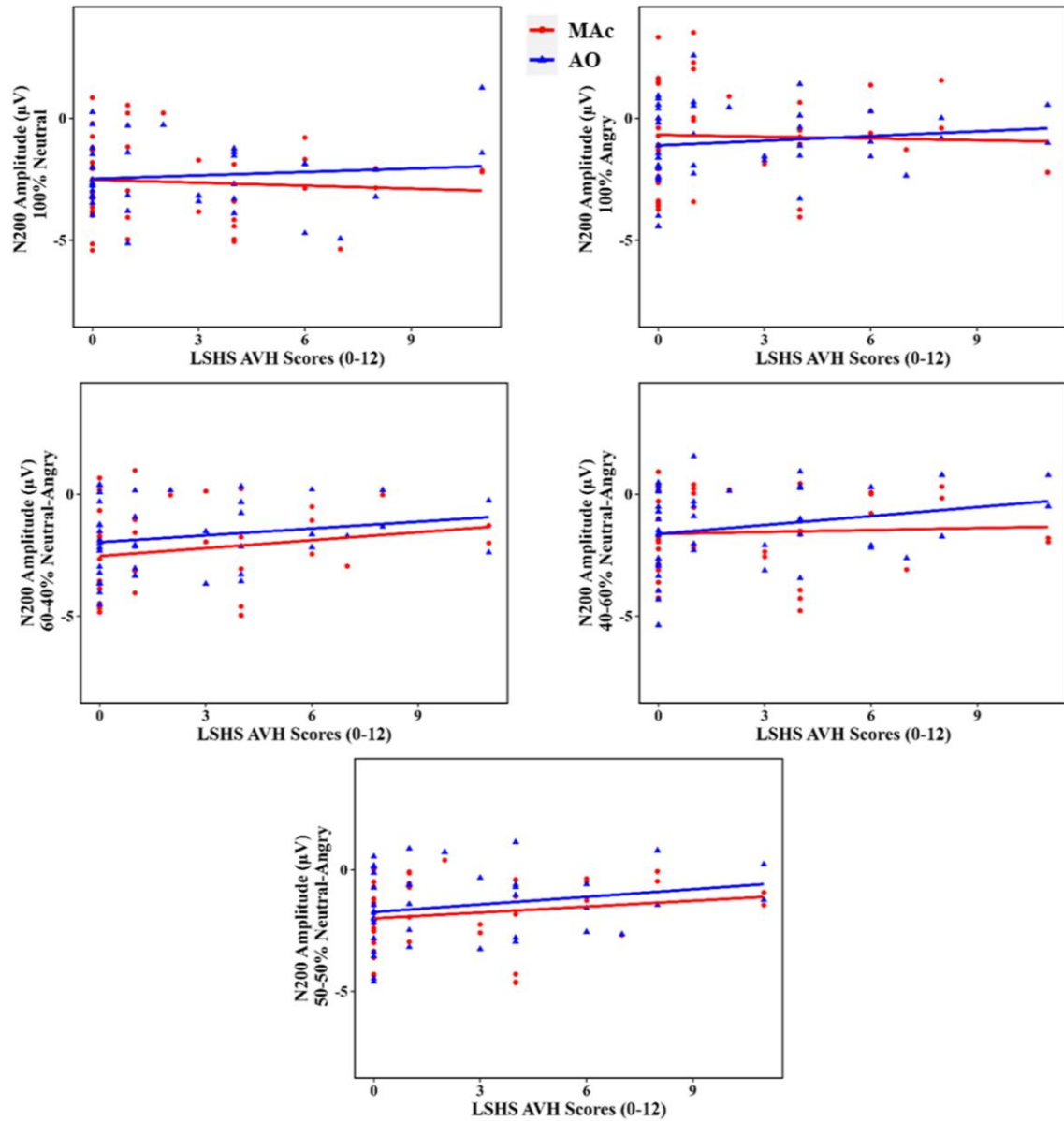
**Supplementary figure 5:** Scatter plots depicting the change in N100 amplitudes as a function of HP based on LSHS AVH scores for each stimulus type. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected.



**Supplementary figure 6:** Scatter plots depicting the change in P200 amplitudes as a function of HP based on LSHS AVH scores for each stimulus type. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected.



**Supplementary figure 7:** Scatter plots depicting the change in N200 amplitudes as a function of HP based on LSHS AVH scores for each stimulus type. Abbreviations: AO = Auditory Only; MAc = Motor Auditory corrected.







# **Chapter 7**

## **Summary and General Discussion**

Through our ability to predict upcoming events, we can continuously adapt to the dynamic nature of the world, even if what we expect and what we actually perceive does not always match. It is important to understand whether these potential mismatches stem from changes in our prior knowledge that generate predictions and shape expectations, comparisons of these expectations with actual sensory input, or noisy sensory input. In the former two cases, adapting and updating predictions is necessary when prior expectations are not met. Attention not only facilitates an estimation of the mismatch between prior expectations and actual input but also modulates its precision and reliability. Any changes in the generation of predictions or the control of attention allocation can lead to imprecise or aberrant prediction error signaling, which in turn may lead to auditory phantom perceptions such as voice hearing and AVH. This dissertation systematically explored the role of prediction and attention in auditory phantom perceptions focusing on voice hearing. The first empirical chapter investigated how attention is modulated by different types of emotions (neutral, positive, negative; chapter 2), and as a function of HP (chapter 3) based on neuroimaging evidence. These chapters were followed by investigations into the interplay of sensory prediction processes and attentional control in the generation and perception of vocalizations as a function of HP (chapter 4-6) using EEG. The subsequent section provides a summary and discussion of the findings presented in the dissertation.

## 1. Summary

Modulations of the interplay of sensory prediction and attentional control are often associated with the experience of hearing voices (Ford, Gray, et al., 2007; Griffin & Fletcher, 2017; Heinks-Maldonado et al., 2007; Hugdahl et al., 2008; Nelson et al., 2014a, 2014b). Unlike non-clinical voice hearers, voice hearers with a diagnosis of psychotic disorder more often perceive derogatory voices, thus further accentuating an individual's attentional bias towards negative emotions (Daalman, Boks, et al., 2011; Johns et al., 2014; Nelson et al., 2014a). This negative attentional bias might also lead to imprecise or aberrant predictions of negative cues and result in misattribution of negative meaning (threat) to a neutral stimulus (Alba-Ferrara et al., 2013; Nelson et al., 2014a). Similarly, imprecise or aberrant predictive processing might contribute to the inability to differentiate internally- from externally-generated events (Griffin & Fletcher, 2017; Nelson et al., 2014b). However, it is still unclear how modulations of predictive processing and attentional control might influence the transition from non-clinical to clinically relevant AVH. Chapter 2 elucidated if and how negative and positive emotional stimuli control attention allocation to task-relevant and task-irrelevant aspects in a conflict scenario. Specifically, it explored whether distinct behavioral and neural responses arise from these emotions or if they simply mark salience. An adapted version of the flanker task was combined with fMRI to investigate valence-specific emotion effects on attentional control in conflict processing. Slower behavioral responses were observed for high (incongruent) compared to low (congruent) conflict scenarios. However, negative and positive emotions did not evoke distinct responses. Neural activity in the dorsal ACC pointed toward its general role in monitoring and assessing conflict as well as in selecting appropriate responses regardless of the stimulus quality. Findings from this study thus confirmed that negative and positive emotional stimuli mark salience in both low (congruent) and high (incongruent) conflict scenarios. This suggests that, regardless of the conflict level, emotional stimuli attract more attentional resources in goal-directed behavior than neutral stimuli. The lack of significant differences between negative and positive emotional stimuli could be attributed to switching between trials of different congruence, arousal, and valence, which might have created an experimental context that required higher cognitive effort to sustain attentional control. These switching

costs also might have diluted valence-specific effects on conflict processing. Overall, the findings underscore the potential of attentional control to mitigate the impact of emotional contexts in both high and low conflict situations, thereby facilitating the attainment of overall task goals.

Behavioral and neural changes in emotion processing and attentional control are often described in non-clinical voice hearers and those with a diagnosis of a psychotic disorder (Alba-Ferrara et al., 2013; Amminger, Schafer, Papageorgiou, et al., 2012; Amorim et al., 2022; Pinheiro & Niznikiewicz, 2019; Pinheiro et al., 2014; Pinheiro et al., 2017). Chapter 3 discussed and integrated fMRI evidence pertaining to the attentional control of emotion along a postulated psychosis continuum. Over-sensitivity towards attentional control of negative compared to positive emotions was associated with the severity of positive and negative symptoms in voice hearers. Further, the interaction between emotion and context-sensitive attentional control was altered and differentially influenced by factors such as arousal, motivation, and reward. This might contribute to altered interpersonal communication and real-life skills in individuals with psychosis. Lastly, increased neural activity in subcortical brain regions that mediate the coupling of emotion and cognitive control (e.g., basal ganglia, thalamus, and angular gyrus) was associated with increased effort in emotion-attentional control tasks in psychosis, reflecting the role and influence of context and individual differences on the interplay of emotion processing and attentional control (chapter 3). However, further research is necessary to specify how these findings might contribute to the understanding of transitions from non-clinical to clinically relevant positive symptoms.

To acquire a more thorough understanding of the contributions of attentional control, emotion processing, and sensory predictive processing in voice hearing, the emotional quality of the self-voice was systematically altered (chapter 4, 6 – neutral-angry; chapter 5 – neutral-pleasure) to manipulate the degree of certainty about sensory feedback in self-voice production. ERPs evoked by an individual's self- and externally-generated self-voice (unambiguous/certain [100% neutral, 100% emotional] and ambiguous/uncertain [60-40% neutral- emotional, 50-50% neutral- emotional, 40-60% neutral- emotional]) were examined as a function of HP (based on LSHS scores) in a classical auditory-motor button-press paradigm. The N100 (chapter 4, 6) and

P200 (chapter 6) suppression effects were replicated such that their amplitudes were suppressed for self- compared to externally-generated self-voice, regardless of their emotional quality or the degree of HP. The N100 suppression effect is related to reduced activity in the auditory cortex when expected sensations match the actual sensory feedback of one's own voice compared to when it is passively listened to (Ford & Mathalon, 2004; Ford, Mathalon, et al., 2001b; Ford et al., 2013; Pinheiro et al., 2018). Similarly, the P200 suppression likely reflects the conscious distinction between the self- and externally-generated self-voice (Knolle et al., 2012; Sowman et al., 2012). Conversely, increased N100/P200 responses to the externally- compared to self-generated voice can also be interpreted as an increase in attention allocation to the unpredictable onset of the self-voice. The P50 response (chapter 6) was higher for self- compared to externally-generated self-voices, likely indicating higher levels of alertness associated with anticipation of the novelty and unexpectedness of different voices/voice feedback presented in the self-generated condition, which preceded the externally-generated condition in the blocked task design (Bramon, Rabe-Hesketh, Sham, Murray, & Frangou, 2004; Grimm & Escera, 2012; Patterson, Hetrick, Boutros, Jin, Sandman, Stern, Potkin, & Bunney, 2008; Thaker, 2008). To examine whether changes in the certainty of sensory feedback to self-voices recruit additional attentional resources, the N100 and N200 responses of both self-generated and externally-generated voices were correlated. The N100 and the N200 responses for self-generated certain/unambiguous but not for uncertain/ambiguous self-voice correlated significantly (see chapter 6: figure 7). This significant correlation might reflect engagement of additional resource allocation for self-relevant unambiguous self-voices (100% neutral and 100% angry self-voice), whereas the lack of significant correlation might reflect disengagement of resource allocation for further perceptual processing of non-self-relevant ambiguous voices (60-40% neutral-angry and 50-50% neutral-angry self-voice).

HP modulated the P50 (chapter 6), N100 (chapter 4, 6) and P200 (chapter 6) suppression effects such that the N100 suppression effect decreased ( $MAc > AO$ ), whereas the P50 and P200 suppression effects increased ( $AO > MAc$ ) with higher HP scores. The decreased or reversed N100 suppression effect, i.e., an increased response to the self-generated voice in high HP individuals, reflects altered sensory feedback

processing and suggests a discrepancy in the matching of the expected and actual sensory feedback of the self-voice (Ford & Mathalon, 2004; Ford, Mathalon, et al., 2001b; Ford et al., 2013; Pinheiro et al., 2018). Alternatively, it may indicate increased error awareness and/or attention allocation to the self-generated own voice in high HP individuals. The larger P50 suppression effect, specifically increased responses during the externally-generated condition in high HP, may reflect larger effort to remain alert and process an overload of sensory information, i.e., the variation in emotional voice quality as well as in the recognition of the self-voice (Bramon et al., 2004; Grimm & Escera, 2012; Patterson, Hetrick, Boutros, Jin, Sandman, Stern, Potkin, & Bunney, 2008; Thaker, 2008). Similarly, increased P200 responses to externally-generated self-voices in high HP may also indicate increased attentional allocation and more conscious processing of the self-voice differing in emotional quality. Of note is that the effects of changes in HP on sensory feedback processing and attentional control were only observed in the context of the neutral-angry emotion spectrum in self-voice production (chapter 4, 6), but not in the neutral-pleasure emotional context (chapter 5). This might be due to lower perceptual discriminability among the five types of voices varying in pleasure content (i.e., similar acoustic properties), which may have resulted in an ambiguous context. This low perceptual discriminability and ambiguous context may have resulted in the lack of discernible differences in certainty about the sensory feedback to self-voices and consequently, a lack of differences in attentional engagement.

To conclude, the combined findings suggest that (i) efficient attentional control counteracts the distracting influence of emotional stimuli on attaining task goals (chapter 2); (ii) alterations in the context-sensitive attentional control of emotion are associated with increased HP (chapter 3); (iii) increased HP is associated with (neurophysiological) changes in sensory prediction and feedback processing as well as attentional control in self-voice production (chapter 4, 6). These findings complement the psychosis continuum hypothesis, identifying alterations in the processing of sensory consequences of one's own actions as well as attentional control in individuals with high HP, which were previously reported in individuals with psychotic disorder (chapter 4, 6). Overall, these findings critically advance the understanding of the neural dynamics of voice hearing and AVH.

## **2. General Discussion**

Our brain processes a constant flux of information from the environment. Predictive processing utilizes internal models constructed from past experiences to predict upcoming events (Corlett et al., 2019; Powers et al., 2016). These processes enhance information filtering capacity by employing attentional control to amplify relevant information, or information that is incompatible with predictions, ultimately optimizing perception (Corlett et al., 2019; Powers et al., 2016). Any mismatch between the expected and actual sensation can lead to an error response. Based on the magnitude of this mismatch, expectations are either updated or in specific cases may lead to phantom perceptions such as AVH. This dissertation sought to systematically examine the role of predictive processing and attentional control in phantom perceptions, in particular AVH. Using neuroimaging, the influence of emotion on attentional control was examined (chapter 2), followed by a review of neuroimaging evidence on the interaction of emotion and attentional control as a function of HP (chapter 3). Next, the interplay of sensory predictive processing and attentional control in self-voice production and perception was examined as a function of HP using EEG (chapter 4-6). The following sections reflect on the outcome of these chapters. Finally, an outlook and potential future research directions are discussed.

### **Sensory suppression**

Self-monitoring plays a fundamental role in cognitive functions and involves the planning, regulation, and anticipation of the outcome of motor actions (Jeannerod, 1997). It helps to prevent the brain from becoming overwhelmed by constant input from internal sensations, such as one's own thoughts. By suppressing or attenuating self-generated sensations, the brain can allocate its resources more efficiently to process external stimuli and maintain a balanced perception of the environment. Prediction plays a crucial role in self-monitoring, enabling the computation of expected and actual perceived consequences/outcomes of an action (Sperry, 1950; Von Holst & Mittelstaedt, 1950). This predictive process aids the distinction of sensations as self-generated or externally-generated, and enables individuals to perceive physically identical sensations differently based on the concepts of agency (origination from oneself or externally) and volition (self-controlled and willful). For

example, we feel less ticklish when we tickle ourselves than when someone else is tickling us (Blakemore et al., 2000). Sensations resulting from our own actions and volition are typically predictable, less salient, and elicit a suppressed neural response compared to the ones generated externally (Wolpert et al., 1995). This sensory self-suppression mechanism allows to enhance salience of, and allocate attention to sensations that arise from external sources (e.g., listening to one's own recorded voice or someone else speaking, compared to when one is speaking) or those that conflict with the predictions (e.g., one's own voice when one has a cold or post vocal strain while singing high notes or yelling at a sports event (Baess et al., 2011; Palmer, Davare, & Kilner, 2016; Wolpert et al., 1995)).

#### *Prediction and attention: two sides of the same coin?*

Previous studies (Baess et al., 2011; Ford & Mathalon, 2004; Hughes et al., 2013) that used an motor-auditory paradigm have focused on the N100 suppression in response to self-generated auditory stimuli, which seems to represent the outcome of predictive processing. This predictive processing may also subserve other cognitive functions such as controlling attention allocation, allowing reactions to unexpected events, and differentiating self- from externally-generated events (Schroger et al., 2015; Schröger et al., 2015). The following provides a brief discussion of the role of prediction and attention in self-suppression mechanism within the context of the motor-auditory paradigm.

Relying on the internal forward model framework (Blakemore et al., 2000; Wolpert et al., 1995), the most widely accepted explanation for the N100 suppression in the motor-auditory paradigm is that it represents the mismatch between the expected and actual perceived sensory consequence of a self-generated action/stimulus or cancellation of the auditory re-afference by a corollary discharge/efference copy-mechanism (Sperry, 1950; Von Holst & Mittelstaedt, 1950). Support for these explanations has come from studies showing a reduced N100 suppression effect in schizophrenia patients (Ford & Mathalon, 2004; Ford, Mathalon, et al., 2001a, 2001b; Ford et al., 2013; Ford, Roach, et al., 2007) as well as individuals with high HP ((Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018); this thesis, chapters



4, 6), suggesting alterations in the forward model. Likewise, a greater lateralization of the readiness potential seems to indicate more robust N100 suppression, implying that intentional/voluntary initiation of action leads to a more pronounced cancellation of the auditory reafference (Ford, Palzes, Roach, & Mathalon, 2014; Pinheiro, Schwartz, Amorim, et al., 2020). In addition to studies replicating the N100 suppression effect using the motor-auditory paradigm (see review Hughes et al., 2013), the forward model has received further support from studies that used the coincidence paradigm (Horvath, 2013a, 2013b, 2014; Horvath, Maess, Baess, & Toth, 2012). In this experimental paradigm, participants generate a series of motor actions (e.g., button-presses) at a specific pace. Simultaneously but independently, a sequence of sounds with randomly spaced intervals is introduced, establishing conditions of motor-only, sound-only, and motor-action-sound based on the probability distribution of the actions and sounds. These studies yielded N100 and P200 suppression effects similar to the motor-auditory paradigms.

Suppression effects might also stem from variations in attention allocation towards auditory stimuli in passive listening and active self-generation conditions. In the motor-auditory paradigm, attention allocation might depend on the (i) task-design – event-related/blocked/mixed, (ii) conditions – active button press to elicit auditory stimulus or passive listening to the same, and/or (iii) task-instructions – whether participants are instructed to pay attention to the auditory stimuli presented or not. Within a blocked design (chapter 6 – figure 1, supplementary figure 1; (Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018)), a self-generated condition block precedes the externally-generated condition block. In the self-generated condition, attention might be distributed between the motor action (e.g., pressing the button) and generated auditory stimulus; whereas in the externally-generated condition, it is assumed that participants maintain sustained attention and vigilance to attend to auditory stimuli presented to them. These differences in attentional resource division might potentially result in an enhanced N100 response during externally- compared to self-generated conditions (chapter 6; (Horvath, 2015; Horvath et al., 2012)). Further, the externally-generated condition was used in active and passive tasks, where participants had to detect specific sounds in the active but not in the passive version of the externally-generated condition (Saupe, Widmann,

Trujillo-Barreto, & Schröger, 2013). The N100 suppression effect, i.e., the difference between self- and externally-generated conditions, was larger in the active than in the passive listening/externally-generated condition (Saupe et al., 2013), suggesting that attentional differences might have contributed to the suppression effect. Presenting the motor-auditory paradigm within an event-related design (this dissertation, chapters 4-6; (Emmendorfer, Bonte, Jansma, & Kotz, 2023)) might influence attentional processes differently. In this version of the paradigm, the Motor-Auditory (MA), Motor-Only (MO), and Auditory-Only (AO) conditions are presented in a randomly mixed fashion. Consequently, a visual cue, represented by a horizontal dash instructing to press a button or a vertical dash indicating not to press a button, was introduced to inform participants when to and when not to press a button. Participants were assumed to be in a heightened state of vigilance or alertness, due to the allocation of attention across various task aspects such as visual cues, button-press, and different auditory stimuli. Additionally, there were likely switching costs associated with shifting attention between processing visual cues, decision-making regarding button presses, and processing different types of auditory stimuli (chapter 4, 5; (Emmendorfer et al., 2023)). These studies reported either a lower magnitude of, or an absence of the N100 suppression effect. Alternatively, other studies (Bass et al., 2008; Knolle et al., 2013a) using a mixed design such that the externally-generated condition was introduced between the self-generated condition trials, reported a larger magnitude of the N100 suppression effect compared to the blocked design. In these studies, there were no visual cues presented and the onset of the externally-generated sounds were unpredictable, leading to higher attention allocation and consequently a higher N100 response. Attention allocation might therefore depend on the intricacies of the task-design.

Nevertheless, some studies provided an alternative viewpoint. Attention allocation was manipulated by directing participant's attention to task-relevant aspects in three conditions; for example, asking them to count (i) the sounds (in both self- and externally-generated conditions), (ii) the extended fixation crosses, and (iii) the button-presses (Timm et al., 2013). The N100 suppression was observed in all three attention allocation conditions. However, differential effects of prediction and attention were observed in different N100 subcomponents. While attention enhanced

all three sub-components of the N100 response (N100a, N100b, and N100c), N100 suppression only showed in the N100b and N100c components. Similarly, the scalp distribution of the lateral N100 subcomponent showed different frontocentral scalp distributions for the attention and the suppression effect (Saupe et al., 2013). These findings imply that sensory prediction and voluntary attention could operate in tandem, exhibiting distinct electrophysiological correlates.

In chapter 4, the N100 suppression effect ( $AO > MAc$ ) for self-voices ('ah' and 'oh' vocalizations varying in anger expression on the neutral-angry continuum) was reported within a mixed event-related design of the motor-auditory task (table 1, figure 3 of chapter 4). The suppressed N100 response for the self-generated own voice, albeit via a button press, showcased that this voice is predictable and therefore, less salient and consequently requires less attentional resources compared to the externally-generated self-voice. This finding was replicated in chapter 6, wherein participants self-generated and passively listened to self-voices ('ah' vocalizations varying in anger expression on the neutral-angry continuum) presented within a blocked design of the motor-auditory task. Apart from the differences in the task-design (blocked vs. mixed) and type of self-vocalization ('ah' and 'oh' in chapter 4 vs. only 'ah' in chapter 6), chapter 6 included a larger sample of 40 participants (versus 25 participants in chapter 4, 5), including two voice hearers. Both chapters also reported a reduced N100 suppression effect with an increase in HP (table 1, figure 2 of chapter 4; table 1, figure 3 Chapter 6). Specifically, the N100 response for the self-generated self-voice increased with an increase in HP scores. This finding can be interpreted in two ways. First, this may be indicative of alterations in predictive processing. These alterations could be associated with the generation of the efference copy/corollary discharge or the comparison of the expected and actual sensory feedback of the self-generated voice (Ford & Mathalon, 2004, 2005; Ford, Mathalon, et al., 2001b; Ford et al., 2013). Alternatively, the increased N100 response to the self-generated voice might be an index of increased attention allocation to a typically predictable stimulus. For high HP individuals, this might reflect their inability to inhibit attention to their own voice when self-generated, perceiving it and deliberating as if it were an error ("Is this my voice?"). In this context, prediction and attention might be considered two sides of the same coin.

The N100 suppression in response to self-generated auditory stimuli is frequently coupled with the P200 suppression (chapter 6, (Chen et al., 2012; Knolle et al., 2012; Knolle et al., 2013b; Sowman et al., 2012)). In Chapter 4, there was no P200 suppression effect, whereas in Chapter 6, a P200 suppression effect was observed (table 2 and figure 4 of Chapter 6). While the P200 response within the context of the motor-auditory paradigm is associated with sensory prediction and feedback processing (Knolle et al., 2012; Knolle et al., 2013a; Knolle et al., 2019; Pinheiro et al., 2018), studies have reported a dissociation in the functional interpretations of the two ERPs. The P200 (but not N100) response was enhanced when the timing of a self-generated stimulus presentation was unpredictable and suppressed when it was predictable (Chen et al., 2012). This might suggest that the P200 component is more susceptible to manipulations of expectations when stimuli are self-generated. Further, the P200 (but not N100) suppression was unaffected in participants with cerebellar lesions, suggesting that the P200 suppression might reflect conscious detection of a self-initiated sound, whereas the N100 suppression might indicate the outcome of comparing expected and actual sensory consequences of a motor action (Knolle et al., 2013a). There is also evidence that the N100 and P200 suppression can be differentially affected by the type of effector (hand vs. foot movement ;(van Elk, Salomon, Kannape, & Blanke, 2014)).

The P200 suppression effect became larger, specifically the P200 response for the externally-generated increased with an increase in HP scores (table 2, figure 4 of Chapter 6). This increase in the P200 response might reflect increased alertness and allocation of attention to the five self-voice types with variable onsets, indicating more conscious processing of these stimuli. This is supported by a study that showed the P200 response to be sensitive to unpredictable auditory stimulus onsets albeit in the self-generated condition (Chen et al., 2012). Therefore, a further increase of the P200 response in high HP individuals might indicate increased alertness and effort to process and categorize different types of self-voices. Previous studies using the same paradigm or the talk-listen paradigm found the same pattern of the P200 suppression effect (AO > MAc) but did not report changes in the P200 suppression effect as a function of HP (Pinheiro et al., 2018), or differences in schizophrenia (Ford et al., 2014) or cerebellar patients (Knolle et al., 2013a) compared with controls, respectively.

The P50 response was enhanced for the self- compared to the externally-generated self-voices, regardless of the voice quality or HP (table 3, figure 5 of chapter 6). Previous studies using a similar paradigm with voices or tones (Knolle et al., 2012; Knolle et al., 2013a, 2013b; Knolle et al., 2019; Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018) did not report this P50 pattern. The enhanced P50 response to self-generated voices might reflect self-induced sensory consequences to different types of self-relevant voices. Further, the self-generated condition block preceded the externally-generated one. Consequently, the self-generated condition might have maintained a higher level of novelty and unexpectedness (“oh, is this my voice?” or “this is indeed my voice”) to the different voice stimuli generated via a button-press, compared to when the same stimuli were presented within the externally-generated condition block. HP also modulated the P50 responses such that the P50 response for the externally-generated voices increased with an increase in HP. This increase in the P50 response might indicate an increased effort and increased attentional focus to process different types of self-relevant voices presented. An abnormal sensory gating is distinctly linked with the decreased suppression of the P50 response in schizophrenia (Bramon et al., 2004; Patterson, Hetrick, Boutros, Jin, Sandman, Stern, Potkin, & Bunney, 2008; Thaker, 2008), indicating that schizophrenia patients cannot inhibit irrelevant sensory input, leading to an overload of sensory information, consequently resulting in perception and attention deficits (Nelson et al., 2014a). The increased P50 response in high HP for externally-generated voices (chapter 6) goes along with this interpretation of the alterations in the attentional focus due to sensory overload of information.

In summary, while attentional processes alone cannot fully explain the suppression mechanism, they likely play a complementary role in it. In light of the findings presented in chapters 4 and 6, the question whether prediction and attention operate differentially in the contexts investigated in this dissertation remains open.

### *Manipulations of voice quality*

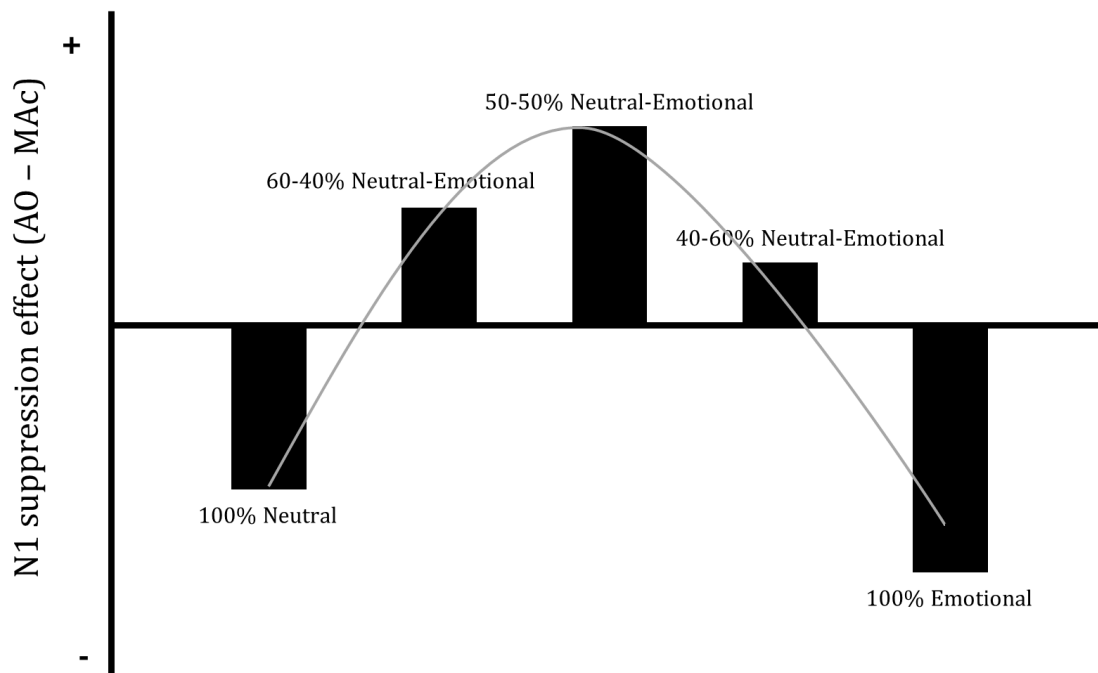
In human auditory perception, voices stand out as one of the most socially relevant stimulus. Most humans spend considerable time listening to voices every day, and our

ability to understand and interpret what voices tell us is vital in social interactions. Voices not only carry verbal speech content but also non-verbal information about identity, emotional and motivational states of the speaker, directly influenced by aspects such as age, gender, mood, context, social background, accent (Amorim et al., 2022; Banse & Scherer, 1996; Paulmann, Bleichner, & Kotz, 2013; Pell, Monetta, Paulmann, & Kotz, 2009; Pell, Paulmann, Dara, Allasseri, & Kotz, 2009; Pinheiro et al., 2015). Similarly, vocalizations that lack verbal content still convey non-verbal cues. Being able to perceive and comprehend these signals can be crucial for survival (Rendall, Owren, & Rodman, 1998; Rendall, Rodman, & Emond, 1996). For instance, different sounds like a baby's cry, a cough, a painful 'ah' when hurting oneself, an angry 'ah' when frustrated, a pleasurable 'ah/oh' during a massage, or an 'oh' when one is surprised all reveal important details about a person's identity and emotional state. One's own voice in particular is the most frequently encountered and emotionally significant auditory stimulus, leading to its prioritization in perception (Conde, Goncalves, & Pinheiro, 2015; Pinheiro, Sarzedas, Roberto, & Kotz, 2023). Processing one's self-voice relies on self-monitoring, which includes the ability to distinguish self from externally-generated voices. Discrepancies in this ability are related to experiencing auditory phantom perceptions such as voice hearing and AVH (Brebion, Gorman, Amador, Malaspina, & Sharif, 2002; Brebion et al., 2016; Ditman & Kuperberg, 2005; Griffin & Fletcher, 2017; Jones & Fernyhough, 2007; Larøi, Collignon, & Van der Linden, 2005). These discrepancies have been reported in electrophysiological research, showing altered N100 suppression for self-generated voices in non-clinical voice hearers as well as voice hearers with a psychotic disorder (Ford, Gray, et al., 2007; Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford et al., 2013).

Hallucinated voices are often indistinguishable from real voices (Larøi, 2012; McCarthy-Jones et al., 2014; Nayani & David, 1996) and typically are of prominent emotional quality (Baumeister et al., 2017; McCarthy-Jones et al., 2014; Nayani & David, 1996; Waters, Allen, et al., 2012; Waters & Fernyhough, 2017). The emotional quality of hallucinated voices is often threatening and derogatory in voice hearers with a psychotic disorder, whereas in non-clinical voice hearers it is more often neutral and/or positive (Daalman, Boks, et al., 2011; Johns et al., 2002; Johns et al., 2014; Larøi, 2012). Additionally, alterations in vocal emotion processing have been associated with

AVH severity such that voice hearers have shown deficits in recognizing vocal emotions and emotional prosody (Alba-Ferrara et al., 2013; Phillips & Seidman, 2008; Rossell & Boundy, 2005; Shea et al., 2007; Tseng et al., 2013). Likewise, the aberrant salience theory (Kapur, 2003, 2004) posits that voice hearers assign salience to irrelevant stimuli, which can manifest as misattributing negative emotional meaning to neutral stimuli (Allott et al., 2014), failure to inhibit attention to irrelevant information (Alba-Ferrara et al., 2013), or perceiving meaningful content in noise (Alderson-Day et al., 2017; Barkus, Stirling, Hopkins, Mckie, & Lewis, 2007b; Powers et al., 2017; Vercammen et al., 2008). Likewise, voice hearers with a psychotic disorder typically exhibit diminished levels of positive affect, as well as a reduced capacity to express and recognize pleasure (Cohen & Minor, 2010; Horan et al., 2008; Kring & Moran, 2008; Li et al., 2019; Watson & Naragon-Gainey, 2010). This impaired ability to engage with positive emotions is closely connected to negative symptoms, such as social withdrawal and constricted affect (Watson & Naragon-Gainey, 2010). Specifically, in the case of non-clinical voice hearers, who frequently encounter positive and neutral voices, they may have an enhanced ability to discern positive content within neutral stimuli. Taken together, investigating both the negative and positive emotional quality of voices and introducing ambiguity into the self-voice processing in a motor-auditory paradigm might identify the transitions from non-clinical to pathological instances of voice hearing. In this dissertation (chapter 4-6), ‘ah’ and ‘oh’ self-vocalizations were used in a motor-auditory EEG paradigm. The emotional quality of these self-vocalizations was manipulated (unambiguous/certain: 100% neutral, ambiguous/uncertain: 60-40% neutral-emotional, ambiguous/uncertain: 50-50% neutral-emotional, ambiguous/uncertain: 40-60% neutral-emotional and unambiguous/certain: 100% emotional) to vary the level of certainty about the sensory consequences in self-voice production as well as to probe changes in attention allocation, as a function of HP. The hypothesis posited that ambiguity in self-voice sensory feedback processing should alter the certainty in perception and affect the authenticity of one’s own voice — “Is this really my voice?”. This effect was anticipated to manifest in a reversed N100/P200 suppression pattern (MAc > AO; figure 1).

**Figure 1:** Hypothetical N100 suppression effect for each voice stimulus.



The conducted studies (chapters 4-6) did not reveal interactions between sensory feedback processing (measured by N100 suppression effect) and voice quality (5 types of self-voice). Based on the ownness ratings obtained from the voice stimuli after each EEG experiment, it is probable that the perceptual differences between ambiguous and unambiguous self-voices (see chapter 4 - supplementary figure 1, chapter 5 - supplementary figure 1, chapter 6 - supplementary figure 1) fell within an acceptable range of feasible physiological voice quality changes. Therefore, these differences might not have induced significant sensory perturbations or a mismatch between expected and actually perceived feedback. Another explanation for the absence of an interaction of voice quality and sensory feedback processing stems from the adaptive nature of predictive processing (Griffin & Fletcher, 2017; Heinks-Maldonado et al., 2007). Perceptions can be characterized as a process in which incoming information integrates with internal predictions generated by a model based on past experiences. These predictions enable stable and unambiguous perceptions, even in situations where sensory signals are erratic or ambiguous. Given the adaptive nature of predictive processing, when the sensory input deviates only slightly from the expected sensory feedback, the internal models would adjust and update to even a minor



discrepancy (prediction error) accordingly. This adaptive nature of predictive processing was reflected in the trend observation of a N100 suppression effect (see chapter 4 - supplementary figure 2, chapter 6 - supplementary figure 2). In both chapters, the differences among the N100 suppression effects for five types of self-voices were not statistically significant, although the N100 suppression effect aligned with the anticipated inverted U shape trend. Of note is that, among the five voice stimuli, both the emotional quality (anger or pleasure content) and the ambiguity of sensory feedback to self-voice varied (chapter 4-6). These voice stimuli were presented in a mixed and random fashion, either within a block of condition (AO or MAc in Chapter 6) or mixed conditions (AO, MO, MAc in Chapters 4 and 5) in an event-related design. The lack of a discernible impact of these voice stimuli on the N100/P200 suppression effect may be ascribed to the incurred switching costs stemming from the presentation of stimuli with varying degrees of emotionality and ambiguity in a mixed fashion. Specifically, the switching among self-voices with varying degrees of emotionality and ambiguity in a mixed manner may have led to a dilution of attentional effects, meaning that none of the stimuli could command sufficient attentional resources to distinguish itself prominently from the others.

Further, the N200 response for the 60-40% neutral-angry self-voice, regardless of the condition (AO or MAc), decreased with an increase in HP (table 2, figure 3 of chapter 4). Based on pilot studies, it is known that anger in 'ah' vocalization was identifiable in the initial morphing steps, i.e., 70-30% neutral-angry voice on the neutral-angry continuum. Hence, the 60-40% distribution of neutral-angry self-voice among the five presented voice types may represent a notable transition from perceiving neutrality to discerning anger in the voice, introducing an element of uncertainty in self-perception. Consequently, this particular self-generated voice may have produced the most ambiguous outcome concerning any perceptual uncertainty of the self-voice. Although not statistically significant, this claim is also supported by the numerically decreased N100 suppression effect for the 60-40% neutral-angry compared to other self-voices (chapter 4 - supplementary figure 2). As the N200 response is associated with error awareness, attentional control and conscious processing of perceptual novelty (Folstein & Van Petten, 2008; Folstein et al., 2008), the decreased N200

response in high HP individuals might likely reflect alterations in processing ambiguity or decreased error awareness while processing an ambiguous self-voice.

In chapter 5, contrary to expectation, the ERP responses showed neither a global N100/P200 suppression effect nor an effect of voice quality on the N100/P200 suppression effects. Further, HP did not influence N100, P200, or N200 responses for self- or externally-generated voices. Multiple factors may have contributed to these null findings. Participants showed reduced ownership (“my voice or someone else’s voice”) of 100% pleasure compared to 100% angry self-voice (see supplementary material of chapter 4, 5; (Herbert et al., 2011; Yoshie & Haggard, 2013)). Moreover, studies have shown a low sense of agency (“feeling associated with the sensory outcome of one’s voluntary action” (Haggard & Chambon, 2012)) associated with pleasure compared to other positive emotions such as pride or elation (Sauter, 2017). Studies have also shown that pleasure can be confused with other positive emotions such as relief and contentment (Sauter, 2017; Sauter et al., 2010). These emotions are similar in their arousal and valence ratings as well as their physical properties, however, they are different in their meaning (Sauter, 2017; Sauter et al., 2010). Given the potential ambiguity in perceiving pleasure emotions and the study’s modulation of voices along the neutral-pleasure continuum, it is possible that these factors collectively created an ambiguous context. This ambiguous context, in addition to low sense of ownership and agency associated with the pleasure emotion, may have led to the lack of distinction in certainty of sensory feedback to self-voices and consequently, lack of differences in attentional engagement.

In sum, to discern the effects of uncertainty of sensory consequence in self-voice production, subsequent studies might explore 100% neutral, 100% emotional, and the most ambiguous neutral-emotional voices in distinct task blocks to disentangle their respective influences on sensory feedback processing as well as attentional control.

### **The continuum perspective**

Traditional psychiatric diagnostic tools use categorical methods to identify clinically relevant psychotic symptoms. Yet, psychotic-like experiences, such as hallucinations, also occur in otherwise healthy individuals (Daalman, Boks, et al., 2011; Johns et al.,

2002; Johns et al., 2014; Larøi, 2012). Cross-sectional studies reported a shared phenomenology and neural mechanisms of voice hearing in those with and without diagnosed psychotic disorders (Ford & Mathalon, 2004; Ford, Mathalon, et al., 2001a, 2001b; Ford et al., 2013; Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018). Given this evidence and hallucination prevalence, a dimensional approach suggests that psychosis forms a continuous phenotype in the general population, with hallucinations (and other psychotic-like experiences) spanning a continuum of normative human experiences (Baumeister et al., 2017; Oestreich et al., 2015; van Os, 2003; Verdoux & van Os, 2002). The continuum perspective of psychosis has become a well-accepted tenet in the last couple of decades. Electrophysiological studies (chapter 4-6) in the current dissertation used a novel approach to examine the changes in sensory prediction and feedback processing, and attentional control in self-voice production as a function of hallucination proneness using sophisticated linear mixed modeling. Scores from the LSHS were used to measure an individual's proneness to hallucinate, which is rooted in the theoretical perspective of the psychosis continuum (Badcock et al., 2008; Castiajo & Pinheiro, 2017; van Os, 2003; van Os et al., 2000). The findings from these studies (chapter 4, 6) support a continuity perspective — increased proneness to hallucinations was associated with changes in sensory feedback processing and increased attention allocation to the self-generated voice based on a reversed/reduced N100 (chapter 4, 6) and increased P200 suppression effect (chapter 6). While these findings support a continuity perspective and enhance our understanding of the fundamental mechanisms underlying AVH through the lens of the forward model theory, it is important to acknowledge certain constraints associated with these findings. These aspects are elaborated below.

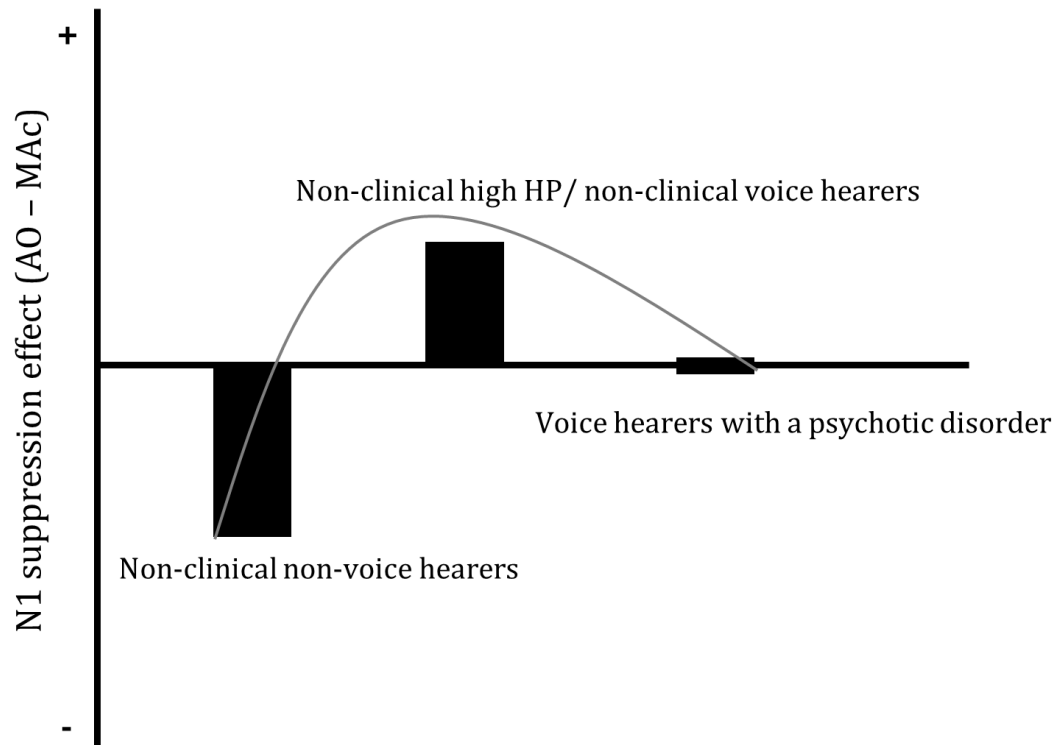
The empirical studies (chapter 4-6) primarily engaged participants from the student community, consisting of undergraduate psychology students affiliated with the faculty of psychology and neuroscience at Maastricht University. Despite the proximity of their age range (chapter 4-5: 18-37 years; chapter 6: 20-44 years) to those undergoing first episode of psychosis or individuals at risk for psychosis (Dean & Murray, 2022; O'Donoghue et al., 2015), the participant pool used in the empirical chapters present a homogeneous portrayal of a restricted population subset. It, therefore, lacks representation of the broader population. Homogeneity within this

participant pool originates from demographic attributes like gender (low male representation), city dwellers, non-immigrant status, and a predominantly Caucasian ethnicity. These demographic factors are acknowledged risk indicators for psychotic-like experiences in the general population and mirror those for clinical psychosis (Belbasis et al., 2018; Cornblatt et al., 2012; Dean & Murray, 2022; Johns, 2005; Van Os, Jones, Sham, Bebbington, & Murray, 1998). As a result, this (non-clinical) participant cohort presented minimal risk for experiencing chronic hallucinations, despite the presence of variability (chapter 4-5: LSHS total scores = 3-42; chapter 6: LSHS total scores [excluding two patients] = 0-42) in their proneness to such experiences based on the LSHS scores. An accurate portrayal of the psychosis continuum entails representation from distinct subsections linked to varying frequencies or severities of hallucinatory experiences, encompassing non-clinical non-voice hearers, non-clinical voice hearers, subclinical voice hearers, and clinical voice hearers. Further, psychosis-like experiences encompass cognitive, motivational, psychosocial and developmental challenges that manifest prior to the emergence of positive symptoms (e.g., hallucinations and delusions), exhibiting partial causal overlap (Kaymaz & van Os, 2010; Murray et al., 2004; Rössler et al., 2015; Tandon, Keshavan, & Nasrallah, 2008a, 2008b). A comprehensive understanding of voice hearing mechanisms would be better achieved by incorporating a multidimensional continuum that includes information on personality traits, emotional states, cognitive attributes and psychosocial risk factors to complement the neurophysiological evidence. This approach would offer a more well-rounded explanation of the voice hearing phenomenon.

Sensory self-suppression implies that sensations from our own actions are usually less intense and provoke a weaker neural response than sensations arising from external sources (Wolpert et al., 1995; Wolpert & Kawato, 1998). In healthy individuals, the sensory consequence of a self-generated action is predicted and attenuated, allowing enhancement of sensations generated from external sources or those that violate predictions (Baess et al., 2011; Palmer et al., 2016; Wolpert et al., 1995). The N100-suppression effect, a measure of the neural suppression of the self- compared to an externally-generated stimulus (e.g., voice) was reduced in individuals with a psychotic disorder compared to controls (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al.,

2001; Ford, Mathalon, et al., 2001b; Ford et al., 2013) and was reversed in high HP individuals compared to individuals with low HP ((Pinheiro et al., 2018), chapter 4, 6). In light of these findings, it was expected that the N100 suppression effect would exhibit a non-linear change with an increase in the severity of hallucination proneness. This could manifest as a normal N100 suppression effect (N100 response for self- > externally-generated) for non-voice hearers or individuals with low HP, followed by an inverted N100 suppression effect for non-clinical voice hearers, and a diminished N100 suppression effect for clinical voice hearers (figure 2). Consequently, drawing from the present findings (chapter 4, 6; (Pinheiro, Schwartz, Amorim, et al., 2020; Pinheiro et al., 2018)), evidence supports the identification of a neuropsychological continuum involving alterations in sensory predictive and feedback processing observed in both highly hallucination-prone non-clinical individuals, as previously observed in (non-clinical and psychotic) voice hearers. Upcoming investigations should take into account the potential for an inverted U-shaped alteration in the N100-suppression effect with varying levels of HP across the full spectrum of the psychosis continuum.

**Figure 2:** Hypothetical N100 suppression effect as a function of hallucination proneness (Ford et al., 2001, 2004, 2013; Pinheiro et al, 2018; Chapters 4, 6).



Furthermore, the N100 suppression was found to be similar in control participants and in schizophrenia patients when a delay of 50 ms was introduced between the button press and a tone delivery (Whitford et al., 2011). Similarly, a delay of 25 ms between the button-press and tones resulted in normal N100 suppression in non-clinical high schizotypal individuals whereas they exhibited subnormal N100 suppression to undelayed self-generated tones (Oestreich et al., 2015). The authors speculated that the processes that generate predictions are still functioning, albeit at a lower capacity and perhaps they are influenced by slower signal transmission. Further, the delay in timing of sensory feedback to self-generated auditory stimulus influences distinct and functionally dissociable processes that affect the forward model (Pinheiro, Schwartz, et al., 2019). Delays outside a certain temporal integration window (~100 ms) are consciously detected as altered sensory feedback (Pinheiro, Schwartz, et al., 2019). Further, the N100 suppression differences between immediate and delayed tone presentation when self-generated were eliminated by

behavioral training, suggesting participants' neural expectations regarding the anticipated timing of self-generated sensations can be modified with training (Elijah, Le Pelley, & Whitford, 2016). These findings bear relevance for potentially improving the diminished sensory suppression mechanism observed in individuals with schizophrenia through training.

### **Forward model, AVH and sensory suppression**

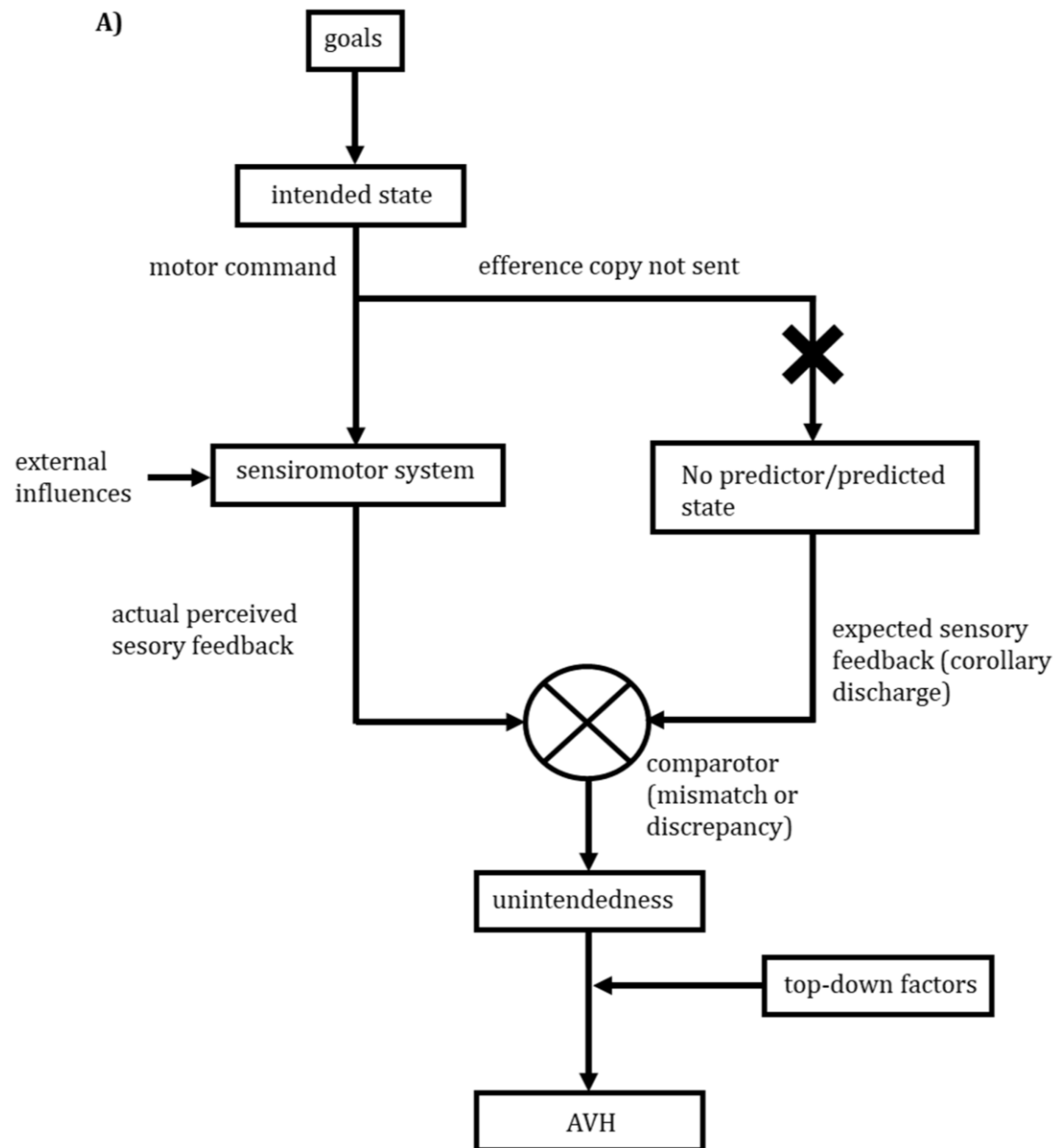
In this dissertation, sensory self-suppression was investigated by examining the N100 (and P200) suppression effect(s) (chapter 4-6) based on the internal forward model framework of sensorimotor control (Blakemore, Rees, et al., 1998; Blakemore et al., 2000; Wolpert et al., 1995; Wolpert et al., 1998). According to this model, the magnitude of the mismatch between predicted and actual sensations is positively correlated with the intensity with which the self-generated action/sensation is perceived. Sensory suppression aids in preserving a sense of agency and self-awareness by enabling recognition of actions, sensations, and thoughts as belonging to ourselves (Blakemore & Frith, 2003).

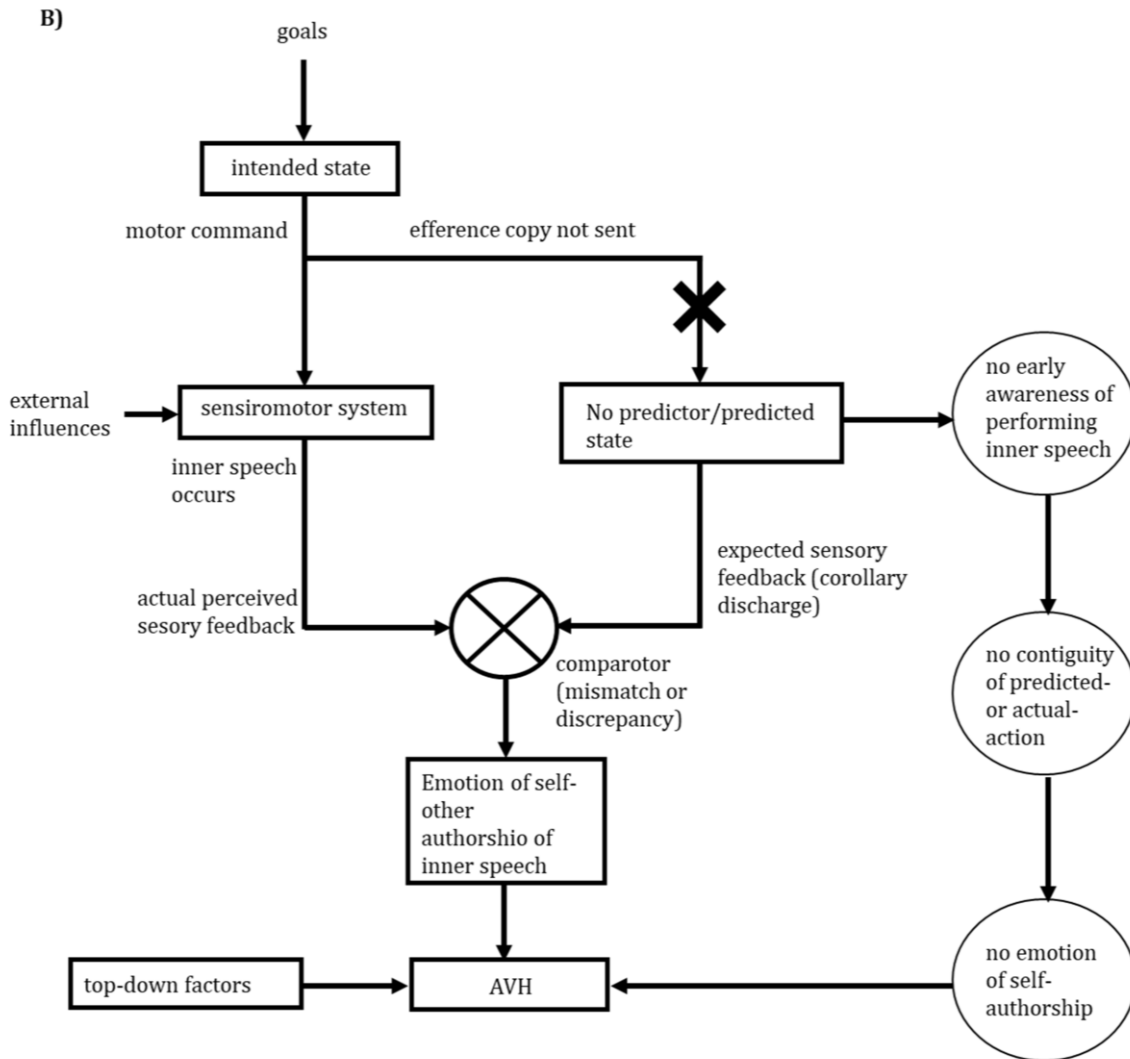
In individuals with psychotic disorders, sensory suppression has been studied in the tactile (e.g., with force-matching tasks (Shergill et al., 2005; Shergill et al., 2013)), visual (e.g., visual displacement tasks, (Sperry, 1950)) and auditory domain (e.g., talk-listen or auditory-motor tasks, (Feinberg, 1978; Ford & Mathalon, 2004)). These studies have shown that self-generated sensations are not suppressed in schizophrenic patients, suggesting a dysfunction of their ability to predict the sensory consequences of their own actions (Ford & Mathalon, 2004; Frith et al., 2000; Martinelli, Rigoli, & Shergill, 2017; Shergill et al., 2005). In voice production, the frontal lobes' voice production areas send predictions to the auditory cortex through an efference copy that generates a corollary discharge. This predictive mechanism helps us recognize the voice as internally generated (Feinberg, 1978; Ford & Mathalon, 2004; Ford, Roach, et al., 2007). Disruptions of this mechanism are considered a potential cause of AVH in individuals with a psychotic disorder (Ford & Mathalon, 2004, 2005; Frith et al., 2000; Martinelli et al., 2017; Shergill et al., 2005). In voice production, voice hearers with a psychotic disorder display increased activity in the auditory cortex (Frith et al., 1992) as well as reduced functional connectivity between

the frontal and temporal auditory regions (Ford et al., 2002). This reduction might contribute to the attribution of inner thoughts to external voices in schizophrenia. These findings conform to principles of an internal forward model in sensory suppression and likely suggest that the efference copy/corollary discharge that originates from frontal cortex fails to signal the temporal regions about the intention to speak.



**Figure 3:** A) Adapted from Seal et al.'s (2004) application of the forward model to AVH.  
 B) Adapted from Jones and Ferynhough's (2004) application of the forward model to AVH.





Although the forward model is effective in elucidating passive experiences like delusions of control, its use in directly investigating voice hearing and AVH where there is no overt behavior has led to criticism (Brown, Adams, Parees, Edwards, & Friston, 2013a; Gallagher, 2004; Randeniya, Oestreich, & Garrido, 2018). The delusional sense of control or the loss of sense of agency is ascribed to the disruption of the efference copy, which is a preparatory action aspect of the motor system. This disruption of the efference copy mechanism is similar to involuntary actions whereby the intent of the action is absent and, therefore, there is no pre-action preparation leading to problems with a sense of agency ('I am not the one moving my hand') but not ownership ('I am moving') (see Gallagher 2004). Similarly, the loss of sense of agency comes from the mismatch of the predicted and actual sensory feedback leads

to increased activity in the parietal cortex (Blakemore & Frith, 2003), which may contribute to the feeling that willed actions are externally controlled (Spence et al., 1997). In the following, a brief discussion on the forward model and its explanation of AVH is provided.

Building upon the mechanisms of inner speech as a basis for AVH, other researchers (Seal et al., 2004) have employed the forward model in the context of AVH, emphasizing unintendedness as a crucial characteristic in these experiences. The involvement of inner speech in AVH was supported by empirical research documenting the measurement of sub-vocalizations using a throat microphone during hallucinatory experiences (Gould, 1950), supporting the notion of an involvement of inner speech in AVH. Additionally, findings indicate that the cessation of vocalizations, and consequently the termination of hallucinations occurred when individuals hearing voices opened their mouths widely (Bick & Kinsbourne, 1987). As per the authors' explanation (Seal et al., 2004), a trigger event instigates the generation of AVH, concomitantly prompting the generation of motor commands to produce inner speech, viewed as a form of action. A mismatch between the distorted or absent predictions and actual sensation gives rise to unintended inner speech, subsequently resolved into self or other authorship guided by top-down attributional biases ((Seal et al., 2004); figure 3A). Others (figure 3B; (Jones & Fernyhough, 2007)) posit that it is the distortion in the generated predictions that causes the absence of self-authorship even before the comparison between predicted and actual sensations occur. They explain that the awareness of performing inner speech cannot occur if there are no predictions generated, leading to no self-authorship (Libet et al., 1983; Wegner, 2002). They additionally add that the model does not require the feeling of unintendedness for the lack of self-authorship (Jones & Fernyhough, 2007).

Gallagher (2004) further suggests that the comparator mechanism in the forward model fails to explain why all thoughts are not perceived as hallucinations and that there are no comparisons between predicted and actual inner speech associated with AVH/voice hearing. In particular, the assumption that thoughts are a form of action is criticized (Gallagher, 2004). As all thoughts are internally generated (Frith & Done, 1988), we are never confronted with the idea of sorting out our own thoughts from

someone else's thoughts (Stephens & Graham, 2000). This is different from differentiating self-generated action from externally-generated ones. Similarly, sometimes 'unbidden' thoughts can unexpectedly appear in our mind without us feeling like we initiated them, however, we do not attribute these thoughts to someone else as someone with psychosis may do (Gallagher, 2004). However, there is no empirical evidence to confirm that these spontaneous, uninvited thoughts actually manifest as AVH (Fernyhough, 2004; Jones & Fernyhough, 2007). According to Jones and Fernyhough, 2007's model (figure 3b), predictions associated with these thoughts may still be generated, and therefore these thoughts are less likely to be attributed to someone else. These thoughts, therefore, may be akin to AVH-like experiences in non-clinical but not in voice hearers with a fully expressed psychotic disorder (Jones and Fernyhough, 2007). Integrating empirical research with individual interviews asking when, how, and under what circumstances voice hearers experience unexpected thoughts could offer valuable insight into how predictions/predicted states influence the ownership of inner speech. Furthermore, if the predicted state or predictions are altered, why are all thoughts not perceived alien in voice hearers (Gallagher, 2004)? If this were the case, where every thought is seen as alien, it would suggest that predictions are consistently malfunctioning, which is not the case. Predicted states/predictions are likely only intermittently altered, hence, it is relevant to understand if these alterations are caused by external stressors, or inability to process complex and ambiguous perceptual cues, or proneness to suggestions. A deeper understanding of the phenomenology of inner speech in voice hearing continuum will pinpoint the association between voice hearing and altered predictive processing.

Brown et al. (2013) point out other issues with the forward model. The forward model links the magnitude of prediction error to the strength of a percept, and the rationale behind this correlation remains unclear (Brown et al., 2013). The prediction error is used for adjusting predictions, but it does not represent the prediction or the perception itself. The author proposes that the percepts are built based on prior beliefs and sensory evidence which is conveyed by the prediction errors (Brown, Adams, Parees, Edwards, & Friston, 2013b; Friston, 2005b; Rao & Ballard, 1999), stating that while prediction errors are crucial for perception, they are not the percept itself. Further, the author notes that the model maps any mismatch between the predicted

and sensory input onto a single variable i.e., the prediction error and lacks an explanation for how the amplitude of the prediction error can integrate information aggregated across all sensory channels (Brown et al., 2013). For example, parametrically delaying the sensation after movement execution alters the degree of mismatch (i.e., the magnitude of the prediction error). However, it is not clear if and how the amplitude of the prediction error correlates with the magnitude of the sensory suppression. For example, sensory suppression was reported even when the temporal predictability of the self-generated sensation was delayed (Bass et al., 2008; Bays, Wolpert, & Flanagan, 2005); suggesting that despite the generation of prediction errors, self-generated delayed sensation can cause sensory suppression. Further, studies have shown that sensory suppression occurs also for stimuli presented (400 ms) before the onset of the movement (Bays et al., 2005; Voss, Ingram, Wolpert, & Haggard, 2008). These studies suggest (i) that the magnitude of prediction error does not correlate with the magnitude of sensory suppression and (ii) that the mechanism of sensory suppression is broader than what is explained by the forward model framework (Brown et al., 2013).

### 3. Outlook and parting thoughts

The findings reported in this dissertation confirm previous findings and offer new insights into the interplay of sensory predictive processing and attentional control in voice hearing. Rooted in the forward model framework, the findings also hint at new venues for future investigation. This concluding section will delve into matters that warrant careful consideration, including some caveats, in forthcoming research endeavors.

*Components of the forward model:* Dysfunction of different components of the forward model (e.g., generation and/or transmission of efference copy and corollary discharge or comparison of expected and actual sensory feedback) may be linked to AVH and voice hearing in multiple ways (Pinheiro, Schwartz, & Kotz, 2020). The present dissertation focused on changes in the suppression of self-generated sensations using self-voices with varying emotional quality and ambiguity levels. These changes affected the certainty of sensory feedback to an individual's voice production as indicated by ERP component modulation discussed in chapters 4-6. ERP components served as indices for changes in the sensory feedback processing within the forward model. Nevertheless, the specific brain regions involved in distinct components of the forward model during self-voice processing and their association with voice hearing remain unspecified. Numerous task-based and resting state fMRI investigations have documented changes in neural activity linked to voice hearing within brain regions and neural circuits associated with speech production, perception, and self-monitoring (Pinheiro, Schwartz, & Kotz, 2020). These regions encompass the superior and middle temporal gyrus, insula, anterior cingulate gyrus, basal ganglia, and inferior and middle frontal gyrus (see review by Pinheiro et al., 2020). Likewise, support for the forward model explanation of voice hearing has emerged from both EEG and fMRI studies (Ford & Mathalon, 2004; Ford, Mathalon, et al., 2001b; Ford et al., 2013; Ford et al., 2002; Heinks-Maldonado et al., 2005; Heinks-Maldonado et al., 2007). Specifically, it has been hypothesized that disruptions in different connections within the cerebellar-thalamo-cortical neural circuitry, which represent distinct components of the forward model, may play a role in the occurrence of voice hearing (Pinheiro, Schwartz, & Kotz, 2020). However, further studies are required to untangle

the relevant brain connectivity, particularly in identifying which specific component of the forward model is altered and its association with particular brain regions or neural networks. Future research may utilize a combination of fMRI and EEG techniques to test spatio-temporal dynamics of different components of the forward model in the context of voice hearing. This would not only serve the purpose of identifying the specific brain regions linked to these forward model processes, but also help examine how their interactions evolve over time in individuals varying in HP.

*Emotional states and emotional quality of voices:* Voices, whether real or hallucinated, encompass a wide array of perceptible features, including the speaker's identity and emotional state (Belin, Bestelmeyer, Latinus, & Watson, 2011). The manner in which voices are perceived is intricately tied to the emotional and psychological state of the listener. Voice hearing in individuals with psychotic disorder is frequently intertwined with negative emotional states such as depression, anxiety, fatigue, and stress (Luhmann et al., 2019; Ratcliffe & Wilkinson, 2016). Similarly, these emotional states are also commonly encountered by individuals without psychotic disorders in the general population and present themselves as negative symptoms. Therefore, it becomes imperative to incorporate assessments of emotional states when studying voice production and perception as a function of HP. This approach would help to disentangle the extent to which perception is influenced by HP and how much can be attributed to an individual's emotional state or negative symptoms. Recognizing the intertwined nature of emotional states and hallucinatory experiences may help in gaining a more comprehensive understanding of individual differences.

*Hearing loss and auditory hallucinations:* Studies indicate that auditory hallucinations in individuals with hearing impairment exhibit greater similarities to those observed in psychiatric diagnoses than previously assumed (Waters et al., 2012). Potential factors contributing to this connection include feelings of isolation as a result of decreased ability to form correct representations of the world (van der Werf, van Winkel, van Boxtel, & van Os, 2010), alterations in source monitoring and dysfunctional top-down processing of information (Linszen, Brouwer, Heringa, & Sommer, 2016) as well as biological links associated with striatal dopamine (Gevonden et al., 2014). Hearing impairment, through its subtle modulation of the

perception of socially significant cues, can affect one's capacity to accurately navigate complex social situations. Additionally, it promotes conversational ambiguity and reduces the ability to effectively discern and evaluate contextual cues (van der Werf et al., 2010). Inadequate filtering abilities and heightened social ambiguity might lead to misattribution of salience to irrelevant information in complex social situations (Kapur, 2003; Weiser et al., 2007) and facilitating hallucinatory experiences. Likewise, source monitoring relies on sensory input, and in situations where external input is diminished, there is an increased risk of misattributing an experience to either external or internal origins, leading to increased disposition to hallucinatory experiences (Johnson et al., 1993; Linszen et al., 2016). Similarly, perception must rely on predictions formed from past experiences and internal representations when bottom-up sensory input is distorted. However, these predictions are often flawed due to difficulties in processing complex social environments, resulting in erroneous top-down predictions and ultimately leading to hallucinatory experiences ((Waters, Allen, et al., 2012)- auditory hallucinations in SZ; (Linszen et al., 2016)). Despite the presence of numerous theories attempting to elucidate the occurrence of hallucinatory experiences in hearing impairment, a comprehensive body of empirical evidence is still lacking to comprehensively grasp the underlying neural mechanisms and the pathophysiological progression.

In a recent study (Linszen et al., 2019), it was revealed that nearly half of individuals with hearing impairment who reported hallucinatory experiences described hearing voices. Additionally, music and tinnitus were also noted as auditory hallucinations in this population. These findings give rise to a few pertinent questions. What distinctions exist in the underlying neural mechanisms between individuals with hearing impairment experiencing auditory hallucinations and those experiencing auditory hallucinations associated with psychosis but without hearing impairment? Do the various types of auditory hallucinatory experiences among individuals with hearing impairment signify distinct phenomena along a shared spectrum, possibly sharing etiological mechanisms like deafferentation? A recent study compared psychotic auditory (verbal) hallucinations and tinnitus (mild and severe hearing loss), all within the framework of predictive processing using EEG and talk-listen paradigm (Ahn et al., 2022). Lack of N100 suppression was observed in both, tinnitus patients



with severe (but not mild) hearing loss and patients with psychosis but not in tinnitus without hearing loss, suggesting a common dysfunction in predictive processing following severe sensory deprivation in tinnitus and psychotic hallucinations. Further, contrary to patients with a psychotic disorder, functional connectivity within the auditory attention network associated with self-generation of a stimulus was well preserved in tinnitus with severe hearing loss. These differences were associated with dysfunctional monitoring of the sensory consequence rooted in sensory loss in tinnitus with severe hearing loss whereas fundamental deficits in sensory feedback processing were associated with psychotic hallucinatory experiences. This suggests that different components of the forward model framework are altered in tinnitus (with hearing loss) and in psychotic disorder. These findings offer new insight into the neurobiological facets of atypical auditory perception stemming from deficiencies in predictive processing and motivate further exploration of interconnectedness of hearing loss, auditory hallucinations (tone or voice) and predictive processing.

*Drug-induced hallucinations:* Hallucinations extend beyond associations with psychotic disorders and can also be induced by substances like psychostimulants, psychedelics, and dissociative anesthetics (Leptourgos et al., 2020; Rolland et al., 2014; Waters, 2023). Examining the controlled hallucinogenic drug usage not only holds potential for gaining insights into intriguing changes in cognition, perception, emotion, and creativity but also discerning similarities and differences in the phenomenology, pharmacology, and neural mechanisms related to psychotic and drug-induced hallucinations (for detailed comparisons see Leptourgos et al., 2020). In terms of neuroimaging markers, drug-induced hallucinations tend to intensify activity in primary sensory cortices, while psychotic hallucinations typically involve the overactivation of subcortical associative brain circuits, particularly thalamo-cortical networks responsible for regulating and modulating sensory information overflow (Leptourgos et al., 2020). Moreover, a dissociation between the default mode network and central executive network has been linked to changes in self-monitoring in both schizophrenia (Alderson-Day et al., 2016; Williamson, 2007) and psychedelic usage (Carhart-Harris, Brugger, Nutt, & Stone, 2013). These hallucinatory experiences may share a common foundation grounded in the same neural processes that underpin our individual perspectives and cognitive operations, specifically through alterations in

predictive processing (Leptourgos et al., 2020). Notably, a study (Corlett, Frith, & Fletcher, 2009) suggested that the atypical perceptions associated with psychedelics stem from an excessive reliance on prior knowledge-based predictions, echoing a similar underlying mechanism seen in psychotic hallucinations (Corlett et al., 2019; Powers et al., 2017). Given these findings, it is important to consider the extent to which the different types of hallucinations exhibit clinical and neurobiological similarity. Research that directly compares drug-induced hallucinations and non-drug-induced hallucinations in non-clinical individuals is still scarce. Studying drug-induced hallucinations in a controlled and supervised way with emotionally stable individuals and comparing them with non-clinical voice hearers can help us better understand the origins and the mechanisms underlying hallucinations (Waters, 2023). Future research should examine the evolution of perceptual awareness with time, discern factors influencing perceptual content, emotional affect, semantic significance, and cognitive appraisals during hallucinatory experiences. Such investigations hold the potential to inform the development of innovative therapeutic and pharmacological methodologies (Waters, 2023).

In closing, the empirical findings in this dissertation showed that sensory predictive processes and attentional control mechanisms are interrelated in the context of sensory suppression during self-voice production and perception. Moreover, HP influenced the sensory suppression of self-voice, indicating that individuals prone to hallucinate voices exhibit abnormalities in sensory predictive processes (inability to predict the consequences of one's actions) and attentional control (misattribution of attention to irrelevant stimuli). For a comprehensive understanding within the entire continuum of psychosis, upcoming studies should include broader representation from various segments of the psychosis continuum. This representation should encompass non-clinical non-voice hearers, non-clinical voice hearers, individuals hearing voices who are clinically at a high-risk of psychosis, and voice hearers with psychotic disorders.





# Appendix



## Impact Paragraph

Investigating the changes in sensory predictive processing, attentional control, and emotion processing as a function of hallucination proneness (HP), with self-voice production as an example, advances our understanding of the neural mechanisms underlying voice hearing. The findings subsumed in this dissertation provide a differentiated theoretical account and give insights that are relevant for future empirical inquiries. Specifically, these findings provide empirical evidence for the existence of a neurophysiological continuum of HP by confirming that individuals with high proneness to hallucinations exhibit alterations in the N100-suppression effect to self-generated own vocalizations, previously shown in voice hearers with a psychotic disorder (Ford & Mathalon, 2004; Ford, Mathalon, Heinks, et al., 2001; Ford, Mathalon, et al., 2001b; Ford et al., 2013; Pinheiro et al., 2018). This may imply that the N100-suppression effect could be a neurophysiological marker that may allow predicting transitions from non-clinical to clinically relevant voice hearing (Oestreich et al., 2015).

By specifically targeting non-clinical individuals who are highly prone to hallucinations, researchers can gain valuable insights into the mechanisms underlying positive symptoms of psychosis. Drawing from the neurophysiological continuum evidence, future research could pinpoint alterations in specific brain regions, neural circuits, and neurotransmitter functioning in non-clinical high HP individuals to establish a basis for the development and the refinement of targeted pharmacological interventions. Furthermore, the advantage of the continuum perspective is that it provides the rationale to study individuals along this continuum who are not diagnosed with a psychotic disorder but exhibit potential precursors in terms of similar symptoms or characteristics. Potentially confounding factors such as medication, illness onset and awareness of illness are not found in these individuals. The acceptance and credibility of the continuum perspective of psychosis rely on empirical evidence. This evidence serves to destigmatize the concept of hearing voices and encourages the acknowledgment that varying degrees of vulnerability are inherent in all individuals (Bentall, 2003; Kessler, 2002).

The research conducted in this dissertation has been disseminated through a variety of channels, including science communication platforms (e.g., FEM-Female



Empowerment at University of Maastricht), international scientific conferences (e.g., The Federation of European Neuroscience Societies (FENS) and Congress of the Schizophrenia International Research Society (SIRS)), and engagement with the general public (e.g., Pint of Science). The findings presented here are openly accessible through publication in journals that adhere to an open access policy. This facilitates the transfer of knowledge and supports the potential for replication of the research. Beyond the scientific community, efforts have been made to make aspects of this research accessible to the general public in the Netherlands. This outreach has included explaining the research in layman language without using scientific jargon during events like "Pint of Science." Additionally, opportunities have been taken to introduce Psychology bachelor students from Maastricht University and Bangaluru City University, India to research methodologies and to the neurofunctional mechanisms underlying voice hearing. Online events have been utilized to showcase the outcome of the empirical studies in seminars discussing the contributions of women in science at Maastricht University.



## **Curriculum Vitae**

## About the author

Suvarnalata Xanthate Duggirala (Xan) was born in Guntur, a city in the south of India on the 2nd of January 1988. She pursued a Bachelor of Engineering degree in Electronic Instrumentation and Control Engineering (2009) followed by Master of Technology degree in Bio-Medical Engineering (2011) from India. Her interest in understanding human behavior and motivation from her father, led her to enroll in the distance learning Master's program in Psychology alongside her regular Master's degree. Her absolute fascination with brain sciences started when she came across the 'The Man Who Mistook His Wife for a Hat' by Oliver Sacks, a book that opened her engineer's eye to the most sophisticated of electronic circuits, the most efficient of signal processing algorithms-the human brain. And she understood how the key to unravelling the bizarrest of behavioural and cognitive disorders was held in understanding the intricate functioning of this mysterious three-pound clump of grey matter. On completion of her Master's, she joined the deemed National Brain Research Centre as an R&D Engineer and later was promoted to Neuropsychologist. She learned the fundamentals of neuroscience including conducting empirical neuroimaging studies, extensive training in neuropsychological assessment as well as publishing her first first-authored empirical paper. To further understand the intricacies of the human brain, she enrolled in the Clinical and Cognitive Neuroscience Research Master's program of Maastricht University, The Netherlands. She explored the interplay of attentional control and emotion processing for her Master thesis, which paved her way to the subsequent PhD projects. After completion of her second Masters, she began her PhD in October 2017 under the supervision of Sonja Kotz, David Linden, Ana Pinheiro and Michael Schwartz. She is currently working as a researcher and university teacher at Maastricht University.

## List of publications

### *As **part** of this dissertation*

- Duggirala, S. X., Honcamp, H., Schwartz, M., Amelsvoort, T., Pinheiro, A., Linden, D., & Kotz, S. A. (under review). Exploring Neural Dynamics in Self-Voice Processing and Perception: Implications for Hallucination Proneness. *bioRxiv*, 2023-09. <https://doi.org/10.1101/2023.09.21.558843>
- Duggirala, S.X., Schwartz, M., Goller, L.K., Linden, D.E.J., Pinheiro, A.P., & Kotz, S.A. (to be submitted). Not enough pleasure? Influence of hallucination proneness on sensory feedback processing of positive self-voice.
- Duggirala, S. X., Schwartz, M., Goller, L. K., Linden, D. E., Pinheiro, A., & Kotz, S. A. (under review). Hallucination proneness alters sensory feedback processing in self-voice production. *bioRxiv*, 2023-07.. <https://doi.org/10.1101/2023.07.28.550971>
- Duggirala, S. X., Belyk, M., Schwartz, M., Kanske, P., & Kotz, S.A. (2022). Emotional salience but not valence impacts anterior cingulate cortex conflict processing. *Cognitive, Affective, & Behavioral Neuroscience*, 22(6), 1250-1263. <https://doi.org/10.3758/s13415-022-01025-9>
- Duggirala, S.X., Schwartz, M., Pinheiro, A.P., & Kotz, S.A. (2020). Interaction of emotion and cognitive control along the psychosis continuum: A critical review. *International Journal of Psychophysiology*, 147, 156-175. <https://doi.org/10.1016/j.ijpsycho.2019.11.004>

### ***Not part** of this dissertation*

- Duggirala, S.X., Honcamp, H., Schwartz, M., Linden, D.E.J., Pinheiro, A.P., & Kotz, S.A. (in preparation). Functional deficits in sensory predictive processing of simple and complex auditory stimulus as a function of hallucination proneness: An EEG replication study.
- Duggirala, S.X., Schwartz, M., Linden, D.E.J., Pinheiro, A.P., & Kotz, S.A. (in preparation). The influence of hallucination proneness on auditory emotion perception.
- Duggirala, S.X., Schwartz, M., Pinheiro, A.P., & Kotz, S.A. (in preparation). Cultural differences in positive and negative vocal emotion perception.
- Contreras-Ruston, F., Wingbermühle, J., Duggirala, S.X., Navarra, J., & Kotz, S. A. (to be submitted). How does Parkinson's disease affect the production and perception of one's own voice?
- Honcamp, H., Goller, L.K., Amorim, M., Duggirala, S.X., Johnson, J.F., Schwartz, M., Pinheiro, A.P., & Kotz, S.A. (to be submitted). Multidimensionality of Hallucination-like Experiences: A Factor Structure Refinement of the Launay-Slade Hallucination Scale.

- Honcamp, H., Duggirala, S. X., Rodino Climent, J., Astudillo, A., Trujillo-Barreto, N. J., Schwartz, M., ... & Kotz, S. A. (accepted). EEG resting state alpha dynamics predict individual proneness to auditory hallucinations. *bioRxiv*, 2023-05. <https://doi.org/10.1101/2023.05.22.541696>
- Korb, S., Mikus, N., Massaccesi, C., Grey, J., Duggirala, S. X., Kotz, S. A., & Mehu, M. (2022). EMOSEX–emotion prevails over sex during implicit judgment of faces. <https://doi.org/10.1037/emo0001089>
- Duggirala, S.X., Saharan, S., Raghunathan, P., Mandal, P.K. (2016). Stimulus-dependent modulation of working memory for identity monitoring: A functional MRI study. *Brain and Cognition*, 102, 55-64. <https://doi.org/10.1016/j.bandc.2015.12.006>

## **Presentations and Outreach**

### *Conferences*

- Duggirala, S.X., Honcamp, H., Schwartz, M., Linden, D.E.J., Pinheiro, A.P., & Kotz, S.A. (July 2022). Does hallucination proneness alter sensory feedback in emotional self-voice perception? Federation of European Society of Neuroscience (FENS), Paris
- Honcamp, H., Duggirala, S.X., Schwartz, M., Linden, D.E.J., Pinheiro, A.P., & Kotz, S.A. (July 2022). Unpacking Hidden Resting State Dynamics in Hallucination-Prone Individuals using a Hidden Semi-Markov Model. FENS, Paris
- Duggirala, S.X., Honcamp, H., Schwartz, M., Linden, D.E.J., Pinheiro, A.P., & Kotz, S.A. (April 2021). Assessing Brain State Dynamics in Hallucination-Prone Individuals with Hidden Semi-Markov Models Schizophrenia International Research Society (SIRS)
- Duggirala, S.X., Schwartz, M., Linden, D.E.J., Pinheiro, A.P., & Kotz, S.A. (April 2020). Self-voice processing and its relationship with hallucination proneness; SIRS, Florence, Italy (note: poster was accepted but conference was canceled due to COVID)
- Duggirala, S.X., Belyk, Philipp, K., & Kotz, S.A. (April 2019). Valence-specific influence of emotion on cognitive control. Research Day, Faculty of Psychology and Neuroscience, Maastricht University, Maastricht.

### *Invited talks*

- Heard but not present (May 2022), Public talk at Pint of Science 2022, Maastricht, Netherlands
- Self-voice perception and Auditory Verbal Hallucinations (April 2022), Online pitch presentation at Female Empowerment Maastricht, Maastricht University, Netherlands

- Hallucination proneness and its neurophysiological correlates (May 2022). Pitch talk at NP&PP Research Day, Faculty of Psychology and Neuroscience, Maastricht University, Maastricht.
- Voices in the head, underlying mechanisms of auditory verbal hallucinations (October 2021), Lecture at International e-Conference on Emerging Research in Cognitive Neuroscience (eCERCN), Department of Psychology, Mount Carmel College (Autonomous), Bengaluru, India
- Why do some people hear voices? (June 2021), Pitch talk at Psychology Bachelors Conference, Maastricht University, Netherlands
- Emotional self-voice processing and its relationship with hallucinatory proneness: preliminary ERP evidence (October 2019). Pitch talk at MHeNs Research Day. Faculty of Health, Medicine and Life Sciences. Maastricht University, Maastricht.
- Sensory gating and executive attention: two sides of the same coin in auditory verbal hallucinations (AVH)? (April 2018). Pitch talk at NP&PP Research Day, Faculty of Psychology and Neuroscience, Maastricht University, Maastricht.

### **Certifications and Scholarship**

- Basic course for clinical investigators (BROK), certified by the examination Board EMWO, NFU BROK committee, Netherlands, Valid from June 2021 until June 2024
- University Teaching Qualification (UTQ), Maastricht University, Netherlands, certificate expected November 2023
- BIAL Scholarship (August 2018 – July 2019), One-year competitive fellowship (€12K) by BIAL foundation, Portugal





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