A comprehensive study on the cognitive mechanisms and neural substrates of hallucination proneness

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Dissertation

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Chapter 1

General Introduction

Since antiquity, rationalists and empiricists alike have asked the question: Do my perceptions reflect reality? Plato first proposed that the true forms of reality are not accessible to our senses (2000/370 B.C.E). Instead, what we truly perceive is determined by comparing our sensations to prototypical object qualities grasped only by the mind. Contemporary formulations expand this concept by describing perception as part of an active process of hypothesis testing by which the most likely object qualities are learned (Gregory, 1980). These hypotheses are key to the interpolation of sensory causes when information is ambiguous and to extrapolation when future conditions are predictable. Already In the 19th century, von Helmholtz argued that perception is additionally shaped by our interaction with the environment (Koenigsberger, 1906). A common aspect underlying these notions is a process of unconscious inference, marked by the development of a division between how self- and externally-generated sensations contribute to perceptions. Consequently, this bestows an understanding of how willed-action and environmental stimuli may interact and influence each other. At a basic level, this property is present across the animal kingdom, allowing for adaptable volitional and reflexive actions to promote survival in a changing environment (Bruineberg & Rietveld, 2014; Crapse & Sommer, 2008). Moreover, increasing complexity in these mechanisms is hypothesized to underlie emergent properties in humans, including mental imagery such as inner speech (Jones & Fernyhough, 2007a; Perrone-Bertolotti et al., 2014; Tian & Poeppel, 2012), visual imagery and imagination (Dijkstra et al., 2022; Gershman, 2019; Thakkar et al., 2021; Williams, 2021), a sense of self (Fotopoulou & Tsakiris, 2017; Haggard, 2017; Moore, 2016; Picard & Friston, 2014), and even consciousness (Clark, 2013; Clark et al., 2019; Haggard, 2005; Hobson & Friston, 2014; Seth et al., 2012; Vallortigara et al., 2021; Vilas et al., 2021).

Unsurprisingly, variability in the function of mechanisms fundamental to perception is linked to both anomalous perceptual experiences and distinctions between self and other (or internal and external) attribution. For example, distortions of reality such as

hallucinations are externalized false perceptions in the absence of corresponding sensory input (Bentall, 1990; Brookwell et al., 2013). Hallucinations are transdiagnostic phenomena most often reported as a key psychotic symptom in schizophrenia, a disorder marked by abnormal perceptual and self-related processing (Bauer et al., 2011; Lim et al., 2016; McCarthy-Jones et al., 2017). However, these experiences have been conceptualized as a part of a psychosis-continuum across the population as opposed to a clinical dichotomy (Baumeister et al., 2017; Healy & Cannon, 2020; Johns & van Os, 2001; Johns, 2005; Myin-Germeys et al., 2003; van Os, 2003). For example, a spectrum of proneness is reported within the general population, with 8-15% of otherwise healthy people reporting to have experienced hallucinations (Beavan et al., 2011; Linscott & van Os, 2013; McGrath et al., 2015). Nevertheless, most of the research into these perceptual distortions of reality has been conducted within samples of individuals diagnosed with schizophrenia. It has been suggested that, due to the observed continuum and common risk factors associated with the presence of hallucinations in both clinical and non-clinical samples, a common aetiology may be present (Johns & van Os, 2001; Johns, 2005; Myin-Germeys et al., 2003).

Due to the inherent confounds introduced by clinical samples (e.g., duration of illness, effects of medication, and hospitalization), investigations in healthy samples have been suggested as crucial for uncovering functional and structural factors of the proneness to experience hallucinations (Kelleher et al., 2010; Kelleher & Cannon, 2011; Verdoux & van Os, 2002). In psychotic disorders, hallucinations occur in combination with a range of other symptoms (Breier & Berg, 1999; Peralta & Cuestra, 1999). This results, for example, in a vastly heterogenous presentation of symptom profiles within schizophrenia (e.g., Grube et al., 1998; Lehoux et al., 2009; Shafer, 2005; Shafer & Dazzi, 2019; Smith et al., 1998; Wallwork et al., 2012), describing multiple subtypes with distinctive phenomenological and pathophysiological states (see reviews in Grover et al., 2018; Picardi et al., 2012). Although neurochemical theories have provided evidence for an underlying dysregulation of dopamine – and interaction with glutamate, gamma-aminobutyric acid, and serotonin pathways – associated with schizophrenia, these explanations are not pathognomonic and can only explain the presence of positive symptoms such as hallucinations (Brisch et al., 2014; Howes et al., 2017; Stahl, 2018). Therefore, it is reasonable to conduct hallucination research independent of

other symptoms of schizophrenia, for example, in terms of cognitive mechanisms which may specifically associate with the phenomenology of hearing voices (Waters et al., 2012a).

Throughout this dissertation, the putative neural substrates and cognitive mechanisms associated with hallucination proneness were investigated in healthy samples. The respective research set off with a focus on three particular aspects: 1) sensorimotor feedback processing, as arguably the most prominent theory of 'voice hearing', 2) the cerebellum, as a structure critical to processing errors in predicted sensory feedback, and 3) voice processing regions of the brain, as the most common form of hallucinatory experience is in the form of hearing voices. Numerous other explanatory models for the emergence and phenomenology of hallucinations are acknowledged, including spontaneous or hyperactive neural responses, intrusive memory or thought, as well as underlying fundamental cognitive mechanisms such attention or inhibition impairments (reviewed in Rollins et al., 2019).

1.1 Perceptual inference

1.1.1 Systems built to predict and how they may err

Over a century after von Helmholtz proposed that perception is the product of unconscious inference, neuroscience began to make strides in developing a formal biologically plausible theory; offering hypotheses for the emergence of false perceptions. Between 1950 and 1990 multiple schemes of brain function and organization were put forth, describing perception as the result of an optimization process where one models the world by comparing actual and expected sensory input (reviewed in Friston, 2010, 2012a). During this era, the foundations for contemporary theories of perceptual systems in the biological brain can be credited to the reciprocal interaction between the fields of machine learning and computational neuroscience. In recent years, this relationship has been reignited due to technological advances in both computer and brain sciences, demonstrating the effectiveness of machine learning models of perception in accomplishing complex cognitive tasks as well as presenting feasible neural correlates (Lillicrap et al., 2020; Song et al., 2020; Whittington & Bogacz, 2019; Yang & Wang, 2020). Moreover, by integrating computational models with

findings from experimental neuroscience, it is possible to build comprehensive hypotheses incorporating the neural substrates, underlying mechanisms, and abnormal structure and function observed in psychiatric illness such as schizophrenia (reviewed in Valton et al., 2017). Although the direct comparison of neural correlates with computational models is outside the scope of the current dissertation, a general understanding of the putative network and components underlying perception is invaluable for the interpretation of hallucinatory experience.

The first attempts to model neural networks of perception were limited to machines akin to single neurons. In these single-layer feedforward neural networks, unilateral external inputs (sensory information) are propagated to output nodes (perceptual belief) which activate based on the sum of the weighted inputs (Rosenblatt, 1958). Decades later, a breakthrough was achieved by creating multi-layer feedforward neural networks where nested levels of sensory input could adjust the weight of connections in subsequent layers (Rumelhart et al., 1986a). Within this model, hidden layers throughout the multiple level framework encode an internal state based on previous experience to predict later inputs to the system. When faced with errors in predicted output, backpropagation of signals can adjust weighting at each previous layer to minimize the chance of future discrepancies, forming a recurrent neural network (Rumelhart et al., 1986b). Ultimately, in 1995, the Helmholtz machine based on the statistical inference of perception outlined the hierarchical system of interacting high- and low-level inputs necessary for building internal generative models capable of predicting the sensorium of the external world (Dayan et al., 1995). These findings provided an initial basis for reciprocal hierarchical cortico-cortical connection models of the brain as a recurrent neural network in the emergence of perception (i.e., Mumford, 1992). Notably, cerebellar circuitry frameworks proposed around 1970 by Marr (1969), Ito (1970) and Albus (1971) provide an analogous explanation for a subcortical system capable of linking expected sensory input with action (i.e., motor learning and adaptation via prediction error) which are now understood to represent hierarchical internal models (reviewed in Kawato et al., 2021). The presence of hierarchical sensory prediction systems in both cortical processing of passive stimuli and subcortical processing of self-generated stimuli has created a long-standing discussion regarding the nature of false perceptions (Behrendt, 1998).

Decades prior to modern formulations of false perceptions, theories of schizophrenia symptoms had attributed auditory verbal hallucinations (AVH) to abnormal prediction of self-generated sensory input (Feinberg, 1978). Conversely, at around the same time, hallucinations were proposed to emerge from faulty hypothesis testing of prior expectations in environmental sensory input against new sensory evidence (Frith, 1979). Nazimek et al. summarize a broad range of possible defective sensory prediction mechanisms attached to AVH (2012). These include dysfunction or hyperactivity of the auditory cortex, impaired bottom-up signalling of external input, abnormal modulation of the thalamus, as well as aberrant connectivity or neurotransmission. The authors present an expectation-perception hypothesis that outlines hallucinations as most likely driven by overly strong predictions that are not adjusted due to the absence or weakening of prediction error transmission (Nazimek et al., 2012). However, theories ascribing hallucinations to abnormal processing of self-generated input view false perceptions as the result of weak predictive signalling resulting in misattribution of sensory feedback to an external source (Ford & Mathalon, 2005; Heinks-Maldonado et al., 2007; van Lutterveld et al., 2011; Whitford et al., 2012). These dissonant propositions have resulted in a debate regarding the characterization of abnormal predictive processing associated with hallucinations (Brower et al., 2019; Corlett et al., 2019; Ford & Mathalon, 2019; Sterzer et al., 2018).

1.1.2 Learning and predicting our effect on the external world

The Bayesian Brain Hypothesis describes an adaptive self-organizing system driven to minimize uncertainty by encoding a probabilistic model of the environment (Bogacz, 2017; Friston et al., 2006; Friston & Stephan, 2007). This requires the functional integration of disparate cortical regions subserving hierarchical components of passive perceptual inference and has been supported by advancing electrophysiological and neuroimaging methods (Friston, 2002, 2005a, 2012a). Through an intricate arrangement of neurons across the cortex, inference and representational learning are recruited to optimize a generative model to aptly predict the causes of sensory inputs (Friston, 2003, 2005a). However, inferring the nature of our perceptions is not limited to exteroceptive (afferent) input. Predicting the source and quality of stimuli that build perception is also fundamental to how the brain processes self-generated (reafferent)

sensory feedback (Friston, 2003; Miall & Wolpert, 1996). Akin to passive perceptual inference, this active process is compatible with a unifying free energy principle by which all organisms and systems are driven to minimize entropy (Friston et al., 2007). In mobile organisms, this drive encourages selective sampling of the environment, such as curiosity motivated behaviour, to actively seek out salient and novel sensory input that may aid in reducing uncertainty (Friston et al., 2017). Notably, divergent theories are proposed by afferent and reafferent sensory prediction models for the presence of false perceptions such as hallucinations. Contrary to the putative role of overweighted top-down priors in passive inference models of perception, reafferent models ascribe hallucinatory experience to weakened or absent top-down signalling of predicted sensory input during action (Ćurčić-Blake et al., 2017a; Horga et al., 2019; Nazimek et al., 2012). Not only does this mark a theoretical divide in how false perceptions emerge, but it puts to question what hallucinations are at their core; are hallucinations just meaning imposed onto otherwise meaningless noise, the externalization of one's own feedback, or a combination of both? Although hallucination research is often conducted from either theoretical framework, there appears to be no definitive reason for these concepts to be mutually exclusive.

From a Helmholtzian perspective, action unto the external world is a process of self-evidencing. Generative models of the environment are learned primarily through observation, while interaction with the environment provides the secondary ability to test hypotheses and minimize prediction error (Hohwy, 2014, 2016, 2017). An alternative proposal claims that action is not simply a tool used in tuning internal models of the outside world. Instead, action and perception together form a single cyclic process to construct the generative model (Bruineberg et al., 2018a). Hence, an organism does not merely model its environment, but instead models an interactive coupling between organism and environment. In such a way, the drive to act is not to provide evidence for hypothesis testing, but to reduce misalignment with the environment, a necessary function in driving the planning and navigation of mobile creatures (Bruineberg & Rietveld, 2014; Crapse & Sommer, 2008; Kaplan & Friston, 2018). As the causal structure between organism and environment is bidirectional, free-energy may also be reduced via manipulating one’s action or the environment itself, for example, in reacting to a danger or constructing a secure niche (Bruineberg

et al., 2018b; Clark, 2013). The general theory of the brain as a system that regulates and optimizes the interaction between organism and environment has been present for much of the past century (e.g., Conant & Ashby, 1970; Craik, 1943; Kawato, 1999). Importantly, a system capable of this task must physically change itself to match the environment in which it regulates (Parr & Friston, 2018). Through cycles of action and perception, the structure and function of the brain becomes aligned with the external conditions, fitting the classical description of a self-organizing system (Ashby, 1947; Banzhaf, 2009; Bruineberg & Rietveld, 2014; Sengupta et al., 2016). Moreover, the structure and function of internal sensorimotor models to predict causal organism-environment interaction has been instrumental for motor control research (e.g., Kawato et al., 1987; Jordan and Rumelhart, 1992; Jordan, 1995; Miall and Wolpert, 1996; Wolpert et al., 1995, 2001; Wolpert, 1997). Notably, accounts of action-observation in motor control diverge computationally from active inference models of cortical prediction coding. Under this self-monitoring theory, one can assess a network of sensorimotor processing to understand how false perceptions emerge in those who hallucinate as well as related functions that may contribute to the phenomenology of those experiences.

The sensorimotor system directs two pathways of action, namely, feedback and feedforward control (Frith 1987; Goldberg, 1985; Wolpert et al., 2011). Through interaction with the environment, a process of sensorimotor integration binds what one does and what one senses (Wolpert et al., 1995). As a result, a sensorimotor transformation is mapped which aligns a causal relationship between action and sensation and is crucial to any biological organism or artificial system that possesses the ability to react to the environment (Pouget & Snyder, 2000). Simultaneous to this mapping, internalized models of the transformations are learned (Jordan, 1995; Kawato et al., 1987; Miall & Wolpert, 1996; Wolpert et al., 2001). An internal model can be applied to feedforward control by determining a required motor command to generate a desired stimulus (forward model) or for feedback control by determining which motor command is necessary in response to a specific sensation (inverse model) (Jordan & Rumelhart, 1992; Miall & Wolpert, 1996). The predictive forward model is of particular importance as it subserves multiple functions integral to motor control: to cancel or suppress cortical responses to expected reafferent sensations, to estimate the current

state or predict future ones, to maintain efficient action in the presence of natural feedback delay, to learn from errors in prediction, and to mentally simulate action and feedback (Frith et al., 2000; Miall & Wolpert, 1996; Wolpert et al., 1995). Additionally, via the monitoring of congruence between predicted and observed sensory input during action, an abstract sense of agency or distinction between self- and other-generated stimuli develops (e.g., David et al., 2008; Griffin & Fletcher, 2017; Jeannerod & Pacherie, 2004). Due to the dynamic nature of the state of the environment, forward models are continuously engaged with the goal of constantly reducing incongruence between intended and actual feedback (Desmurget & Grafton, 2000; Jordan & Rumelhart, 1992). This form of self-monitoring can be applied to feedback from proprioceptive (Miall & Wolpert, 1996; Wolpert & Kawato, 1998), visual (Leube et al., 2003), auditory (Hashimoto & Sakai, 2003), and tactile modalities (Blakemore et al., 1998). Indeed, schizophrenia patients – a clinical group most often marked by AVH – have displayed impairments in motor-induced suppression (e.g., Ford et al., 2014; Whitford et al., 2019), and in attribution between self- and other-generated sensation (Blakemore et al., 2002; Frith et al., 2000; van Der Weiden et al., 2015). Therefore, it has been proposed that a proneness to hallucinate is the product of defective forward model processing - a central tenet to hypotheses across this dissertation (Jones & Fernyhough, 2007a).

1.1.3 The cerebellum: Internal models and error signal processing

Although learning sensorimotor associations requires a network of specialized regions throughout the brain, the cerebellum plays the critical role in compensation or adaptation to unexpected changes in feedback (Caligiore et al., 2017; Doya, 2000, Doya et al., 2001; Bastian, 2006; Doyon et al., 2009; Halsband & Lange, 2006; Hardwick et al., 2013; Krakauer et al., 2019; Schmuelof & Krakaeur, 2011; Schwartze & Kotz, 2013, 2016). During action, descending motor fibres send signals to the effectors (e.g., muscles) while parallel projections representing an efference copy are sent to the pons. Mossy fibres project from the pons into the cerebellum to innervate granule cells which extend long dendritic branches along the outer layer of the cerebellar cortex as parallel fibres. To determine if a predicted outcome has been achieved at the expected time, ascending feedback signals are compared to expected feedback in medullary nuclei and enter the cerebellum as climbing fibres where the error is processed (reviewed in Popa & Ebner, 2019). The resulting signal is propagated back to the cerebral cortex to

facilitate the updating or adaptation of motor commands and internal models. However, the cerebellum has been suggested as more than just a tool for producing smooth successful movement, instead acting as a universal prediction system (Ramnani, 2006). Three properties of this structure make this possible: generalizability, specificity, and sheer processing power.

First, in order to serve various functions, the cerebellum must be simplistic enough to offer a common computational mechanism. It has been hypothesized that this requires a hierarchically arranged architecture of repeated modules (Grafton & Hamilton, 2007; Haruno et al., 2003), each with paired internal (controller) and forward (predictor) components (Gomi & Kawato, 1993; Haruno et al., 2001; Wolpert & Kawato, 1998; Wolpert & Flanagan, 2001). The uniform structure of the cerebellum is congruent with such computational schemes of sensorimotor learning and inference (Dean et al., 2009; Haruno et al., 2003; Ito, 2000, 2006; Kawato & Gomi, 1992; Miall et al., 1993; Porrill et al., 2004; Raymond et al., 1996). Further evidence is provided in the field of robotics, where algorithms inspired by cerebellar microcircuitry have produced successful results in motor adaptation and learning (e.g., Asadi-Eydivand et al., 2015; Broucke, 2021; Carrillo et al., 2008; Casellato et al., 2014, 2015; Coenen et al., 2001; Floreano et al., 2014; Hofstötter et al., 2002; Kalidindi et al., 2019; Urbain et al., 2020). The maintenance of accurate predictions requires two opposing states of cerebellar activity during feedback processing. A) Expected feedback (successful prediction): simple spike Purkinje cell (PC) activity as a result of parallel fibre (PF) input and normal spontaneous firing patterns. Here, PFs which innervate the PC are active in the absence of climbing fibre inputs, allowing long-term potentiation (LTP) of the PF-PC synapse to persist. B) Unexpected feedback (unsuccessful prediction) feedback: complex spike PC activity as a result of climbing fibre input. In this case, the PC and PF will be coactive, leading to long-term depression (LTD) of the PF-PC synapse essential for adaptive processing (detailed description of cerebellar circuits in Fujita, 2022; Ishikawa et al., 2016; Kawato et al., 2021; Manto et al., 2011; Zang & De Schutter, 2019). Therefore, bidirectional PF-PC LTP/LTD plasticity allows for the system to continuously update, for example, to achieve the most reliable motor command-sensory feedback relationship.

Second, in order to influence the activity in numerous specialized regions of the cerebral cortex, the cerebellum must have specific connections to diverse areas of the brain. Separate functional regions within the repeated structure of the cerebellar cortex receive inputs from motor, cognitive, and sensory regions of the cerebral cortex (Buckner, 2013; Glickstein et al., 2011; Guell & Schmahmann, 2020; Ito, 2008; Koziol et al., 2013; Stoodley & Schmahmann, 2010, 2018). The functional divisions of the cerebellar cortex share both functional connectivity to corresponding regions of the cerebral cortex (Allen et al., 2005a; Bernard et al., 2012; Bucker et al., 2011; Habas et al., 2009; Krienen & Bucker, 2009; O'Reilly et al., 2009; Sang et al., 2012), as well as structural connectivity by forming closed loops (or processing units) which operate using the same computational structure (Kelly & Strick, 2003; Witter & De Zeeuw, 2015). Within the sensorimotor domain, there is also strong evidence for somatotopic mapping of the cerebellar cortex of different body parts similar to the primary motor cortex (Boillat et al., 2020; Grodd et al., 2001; Manni & Petrosini, 2004; Mottolese et al., 2013; Nitschke et al., 1996; Rijntjes et al., 1999; van der Zwaag et al., 2013; Wiestler et al., 2011). Although it may appear that one-to-one relationships between functional regions of cerebral and cerebellar cortices create an excess of processing units, the nature of the PF-PC synapse allows for an extraordinary number of combinations to increase efficiency. For example, each PC acts as a convergence point receiving synapses from 100,000 PFs, yet is innervated by only a single climbing fibre (Hoxha et al., 2016). This allows each climbing fibre error signal to manipulate the plasticity of many different PFs, which themselves receive inputs (via mossy fibres) from different regions of the cerebral cortex. Moreover, each PF innervates multiple PCs either directly or via inhibitory interneurons (detailed review of cerebellar interneurons in Kim et al., 2021; Palacios et al., 2021; Schilling et al., 2008; Yamazaki & Lennon, 2019). The result is a mixture of overlapping functional zones of the cerebellar cortex, and conversely, contributions from disparate functional zones to a single sensorimotor process.

Third, in order to serve various tasks involving different motor, sensory, and cognitive regions of the brain, the cerebellum requires a substantial level of processing power. Although the cerebellum only makes up roughly 10% of total brain volume, it holds more than 50% of the neurons in the brain (roughly 50-100 billion neurons in comparison to the 10-20 billion of the cerebral cortex) (Von Bartheld et al., 2016).

Granule cells (including PF dendritic branches) account for the majority of cerebellar neurons at 50-70 billion, while PCs have been reported at a far smaller number of about 20 million. The densely packed repeating structure of the cerebellar cortex, composed of only four inhibitory (Purkinje, Golgi, basket, and stellate) and two excitatory neurons (granule and brush), therefore provides the simple yet high power universal computation machine required for complex tasks of learning and inference in the brain (Ito, 2006; Paulin, 2005; Raymond et al., 1996).

Across the studies in this dissertation, the second topic of focus was on the role of the cerebellum in the externalization of self-generated feedback. This structure - essential to monitoring and processing prediction error - has shown abnormal cerebellar activity associated with hallucinations in schizophrenia and self-monitoring (Picard et al., 2008; Pinheiro et al., 2021; Moberget & Ivry, 2019). Although the role of the cerebellum in sensorimotor feedback processing and theories of self-monitoring abnormalities in false perception are well established, there is still no full consensus on exactly what component is responsible for the emergence of hallucinations. For example, abnormal function within the cortico-cerebello-cortical loops may exist at the level of top-down predictive signalling, integration of those signals, comparison with actual feedback, or signalling from the cerebellum to the cortex (Pinheiro et al., 2020). Therefore, the cerebellum and its connections are prime candidates for future research into the role of sensorimotor processing in hallucinations.

1.2 Voice signals and speaker identity

The majority of hallucinations are in the form of “hearing voices” in the absence of a corresponding speaker. This pattern is reported for patients, most notably as the hallmark symptom of schizophrenia (Bauer et al., 2011; McCarthy-Jones et al., 2017; Waters et al., 2014). Although psychosis-like experiences in non-clinical samples are reported in multiple forms (Healy et al., 2019; Kelleher et al., 2012; Linscott & van Os, 2013, van Os et al., 2009), one of the most prevalent findings is voice hearing whereby perceptual aberrations lie on a continuum with pathological presentation of symptoms (Beavan et al., 2011; de Leede-Smith & Barkus, 2013; Johns, 2005; Johns et al., 2014; Larøi et al., 2012; Maijer et al., 2018; Zhuo et al., 2019). Although most people who do not meet the criteria for diagnosis report only attenuated forms of abnormal

experiences related to voice hearing, a specific group known as *healthy voice hearers* shows a phenomenology and severity similar, but not equal, to those in schizophrenia (Baumeister et al., 2017; de Leede-Smith & Barkus, 2013; Johns et al., 2014; Larøi et al., 2012; Zhuo et al., 2019). This underscores the presence of a dimensionality in general cognitive mechanisms subserving perception as opposed to schizophrenia-specific pathological brain states as the cause of hallucinatory experience. For this reason, the third focus of the dissertation centres on how the brain responds to voice signals in non-clinical samples and how the involved regions are associated with one's proneness to hallucinate. Voice signal processing, in theory and functional organization of the brain, has much to do with the concepts of perceptual inference and self-monitoring discussed above. Therefore, a brief description is provided for how the same underlying mechanisms have afforded the ability to recognize, understand, and communicate with other actors in the world, and the relationship of these functions to hallucinations of voice. Finally, a specialized network in the brain required to serve voice processing is described, including how and where abnormal functioning associated with hallucinations may occur.

1.2.1 Communication signaling: Aligning with actors in the external world

Predictive action-perception models develop based on the distinct physiological attributes and environmental niche of a species. Driven by the hierarchically mechanistic mind, the motor system directs an organism to low entropy states particular to their phenotype, increasing the chance of survival (Badcock et al., 2019a,b). This innate drive may rely on both the phylogenetic contribution of heritable adaptive priors and ontogenetic adaptations over the lifespan which may be passed onto future generations. Through the pressures of natural selection, as a Bayesian model of free-energy minimization (Campbell, 2016), species-typical patterns of cognition and behaviour develop (Friston, 2011a). This implies that conspecific creatures applying active inference in a shared environment may evolve the ability to: 1) find common meaning in stimuli (Constant et al., 2019), 2) form a social cognition to attribute the mental states of other actors (Brown & Brüne, 2012; de Bruin & Michael, 2021; Teufel et al., 2010), and/or 3) regulate one's action and inner states via the observed behaviour of other organisms (Tison & Poirier, 2021a). Moreover, intentional

cooperative communication emerges. This may be driven by an innate pressure to minimize prediction error by aligning with other actors within the species (Vasil et al., 2020), or alternatively to constrain affordances to the mutual goal of a collaborative action (Tison & Poirier, 2021b). Functionally, the alignment of internal states between actors relies on an intrinsic link between action and perception (i.e., James, 1890/1950), evidenced by the activation of motor programmes when imagining or observing the corresponding action being performed by another actor (Jeannerod, 1994; Prinz, 1997). Through the collective feeling of agency during joint action (Loehr, 2022; Zapparoli et al., 2022), shared action-perception representations are formed to predict the actions of the other actors (Bolt & Loehr, 2021; Sebanz et al., 2006; Wilson & Knoblich, 2005). By synchronizing internal brain states, a communicative dialogue may form allowing one to infer the state of mind necessary for the action of the other agent (Friston et al., 2011; Friston & Frith, 2015; Sebanz et al., 2006). Self-monitoring theories of motor control offer a mechanistic model for joint action and communication resulting from the alignment of internal states (Brown & Brüne, 2011; Pesquita et al., 2018; Vesper et al., 2010; Wolpert et al., 2003). As proposed by Wolpert, Doya, and Kawato (2003), the hierarchical modular architecture underlying sensorimotor transformations extends to 'social interaction loops', whereby communication (i.e., motor signaling) can tune models of action-perception between actors and the reliability of the intended meaning of internal states being conveyed. Notably, hallucinations are most often misperceived as communicative signals (i.e., voices) in the absence of an external agent (Baumeister et al., 2017; Larøi et al., 2012; Stephane et al., 2003; Waters & Fernyhough, 2017; Zhuo et al., 2019). Although auditory verbal hallucinations (AVH) may appear as inside or outside of the head, they can be dissociated from inner speech and be commenting, commanding, or even conversational in nature (e.g., Alderson-Day et al., 2021; Toh et al., 2020a,b; Woods et al., 2015).

Multiple functional neuroimaging studies have demonstrated abnormal localized and distributed activations across a cortical network underlying vocal communication in people who experience hallucinations (Alderson-Day, 2015, 2016; Bohlken et al., 2017; Ćurčić-Blake et al., 2017a; Hugdahl, 2017; Northoff, 2014). Additionally, there is evidence for both speech production and speech perception regions to become active

when experiencing AVH (Allen et al., 2008; Bohlken et al., 2017; Jardri & Sommer, 2013; Upthegrove et al., 2016). However, not all reports of AVH symptom capture during scanning report involvement of speech production regions, leaving outstanding questions regarding the necessity of premotor and prefrontal area activation for the emergence of hallucinatory voice hearing. Moreover, interpreting studies that do observe speech production region activity during AVH is further complicated, as theories of motor system involvement in passive voice perception have been presented, contested, and refined for decades (reviewed in Devlin & Aydelott, 2009; Diehl et al., 2004; Galantucci et al., 2006; Massaro & Chen, 2008; Samuel, 2011; Schwartz et al., 2012; Scott et al., 2009; Stokes et al., 2019). In particular, the last 20 years have included controversial accounts of "mirror neuron" activity during passive voice hearing (Grafton & Hamilton, 2007; Kilner et al., 2007; Rizzolatti & Craighero, 2004), and in doing so have re-ignited debate (e.g., Hickok et al., 2009; Lotto et al., 2009; Wilson, 2009). The original (contentious) motor theory of speech perception posits the primary mechanism of speech perception as the decoding and recognizing of stored motor programmes associated with phonetic representations (Liberman, 1957; Liberman et al., 1967; Liberman & Whalen, 2000). However, as evident from aphasia research, a lack of speech production region involvement does not eliminate the ability to perceive speech sounds (reviewed in Hickok et al., 2011; Lotto et al., 2009; Venezia & Hickok, 2009). It is, therefore, clear that speech production regions are not essential for perception. Conversely, it has been suggested that speech production regions serve a secondary role to speech perception, engaged under specific contexts. For example, motor regions engage during ambiguous, noisy, or degraded stimuli to assist in decoding of phonetic representations (Adank, 2012; Liebenthal & Möttönen, 2018; Wilson, 2009; Wu et al., 2014), and to guide turn-taking or conversational smoothness by predicting the other speaker's speech to avoid processing delays (Pickering & Garrod, 2007; Scott et al., 2009). Additionally, a prominent motor control theory of speech describes the action-perception mapping as necessary for successful acquisitions of speech sounds and articulation (Guenther, 1994, 1995; Guenther & Vladuisch, 2012; Kearney & Guenther, 2019; Tourville & Guenther, 2011). This model outlines an adaptive neural network which integrates specialized cortical and subcortical components, such as speech sound maps in the left inferior frontal cortex

and ventral premotor cortex, auditory state and target/error maps in the left superior temporal gyrus, somatosensory state maps in the ventral somatosensory cortex, and - in line with self-monitoring theories - cerebellar modules serving a feedforward control subsystem (see schematics in Golfinopoulos et al., 2010; Guenther, 2006; Tourville & Guenter, 2011).

The involvement of the motor system when 'hearing voices' in the absence of another speaker is most often attributed to self-monitoring theories, i.e., served by the aforementioned speech production-perception network (Jones, 2010). An assumption central to this theory posits AVH as the result of abnormal top-down predictive signaling (in contrast to theories of abnormal bottom-up sensory processing). Essentially, the propensity to externalize self-generated stimuli is extended to inner speech. Predictive signaling from speech productions regions to auditory target/error maps in the planum temporale are insufficient, resulting in a lack of suppression of sensory cortices allowing activation thresholds to be surpassed under conditions of minimal noise (Bentall, 1990; Frith & Done, 1988; Frith et al., 2000; Jones & Fernyhough, 2007b; Weiss & Heckers, 1999). These accounts may provide a sound explanation for why AVH are the most common form of hallucination, as there are no direct analogues for the speech action-perception network within other sensory modalities. Evidence for the self-monitoring of (inner) speech theory of AVH is primarily demonstrated in electrophysiological responses where motor-induced suppression is reduced for those with schizophrenia, schizotypy, or clinical high-risk for psychosis (e.g., Ford et al., 2001a, 2001b, 2007, 2013; Ford & Mathalon, 2004; Mathalon & Ford, 2008; Oestreich et al., 2015; Perez et al., 2012; Pinheiro et al., 2017, 2020b; Rosburg et al., 2008; van Lutterveld et al., 2011; Whitford, 2019). Furthermore, the pathway connecting prefrontal speech production and temporal speech perception regions assumed to subserve a direct route for motor predictions to target/error predictions maps has indicated structural abnormalities in patients with schizophrenia who experience AVH (Catani et al., 2011; Chawla et al., 2019; Ćurčić-Blake et al., 2015; De Weijer et al., 2011, 2013; Hubl et al., 2004; Leroux et al., 2017; McCarthy-Jones et al., 2015; Psomiades et al., 2016; Rotarska-Jagiela et al., 2009). Additionally, atypical processing of left-lateralized speech perception in those with AVH has been linked to variability in the connectivity of pathways connecting the cerebral hemispheres (Hugdahl, 2009). The

interhemispheric miscommunication theory is supported by both structural and functional connectivity evidence which may be influenced by progression into psychotic illness or severity of symptoms such as AVH (Ćurčić-Blake et al., 2017a; Steinmann et al., 2014). Like unconscious inference accounts of false perceptions, this theory integrates the imbalance of excitatory/inhibitory inputs in top-down signaling, ascribed to an increase in the excitatory neurotransmitter glutamate affecting interhemispheric pathways involved in the perception of speech signals (Ćurčić-Blake et al., 2017b; Hugdahl, 2009; Hugdahl et al., 2015; Steinmann et al., 2019).

The self-monitoring theory provides a compelling explanation for the emergence and phenomenology of AVH. However, observations in neuroimaging research indicate the involvement of more distributed, interacting, brain networks which may indicate a broader cognitive mechanism (Alderson-Day et al., 2015, 2016; Ćurčić-Blake et al., 2017a; Northoff, 2014). For example, activation during hallucinations is not limited to left-hemisphere speech and language processing regions, and includes a larger network including bilateral frontal, temporal, and parietal regions of the cortex (see meta-analyses in Jardri et al., 2011; Kompus et al., 2011; Kühn & Gallinat, 2012; van Lutterveld et al., 2013; Zmigrod et al., 2016). Likewise, abnormalities in various white matter pathways additional to those serving speech have been reported in schizophrenia patients and healthy individuals prone to hallucinate (Bopp et al., 2017; Ćurčić-Blake et al., 2015; de Weijer et al., 2011, 2013; Filippi et al., 2014; Hubl et al., 2004; Jung et al., 2020). Perhaps most importantly, functional connectivity studies provide evidence for abnormal switching between default-mode and active sensory processing (including speech and language) as the result of inappropriate attribution of salience to irrelevant stimuli (Menon et al., 2011; Palaniyappan & Liddle, 2012; Schmidt et al., 2015).

1.2.2 Voice signal processing: Identifying other actors in the external world

To explore neural substrates associated with AVH, brain activity across a cortical voice perception network is assessed. A summary of the information carried in voice relevant to AVH (e.g., identity) is provided, and candidate regions for abnormal hypersalient cortical responses as well as errors in self-other identity attribution. In general, most living organisms possess a capacity to produce and understand signs necessary for

successful interaction with the outside world (reviewed in Sebeok, 2001). Detecting signals from the environment or conspecifics conveying affective meaning will trigger an automatic behavioural response. However, for species who have evolved intentional communication of meaning, more complex abilities are endowed such as the ability to learn about the agency of actors and to engage in cooperative information sharing (Tomasello et al., 2012). Although this can be signalled via gesture, touch, or even scent, vocal communication is particularly efficient. It allows for information transfer over long distances, and when articulated through speech, conveys increasingly complex messages which peak at the uniqueness of human language (Christiansen & Kirby, 2003). In humans, the emergence of this complex vocal communication system relied on an interwoven evolution of multiple capacities, such as voluntary control of the vocal apparatus, a flexible set of vocal sounds, and the capacity for vocal learning to add to this repertoire (reviewed in Belyk & Brown, 2017). Seminal biosemiotic and biolinguistic theories present different explanations on why language emerged in humans, yet find agreement in that linguistic communication may be an exaptation made possible from the adaptive traits evolved for vocal signaling in the human social environment (Barbieri, 2010; Hauser et al., 2002; Sebeok, 2001). Foreseeably, an evolutionary history shaped by social interaction has produced additional value to vocal signaling irrespective of language, in the form of discriminating or recognizing the identity of other individuals. In such a way, voice itself may operate as an "auditory face" by which the specific affective and identity information of an actor may be ascertained (Belin et al.,2004; Belin et al., 2011; Schirmer, 2018). Accordingly, human voice processing imports two functions: to identify what a speaker is communicating and to identify information about the speaker themselves. To extract these features from complex human voice signals, the brain relies on a broad network of temporal and extra-temporal regions.

Human voice processing spans many cortical and subcortical regions of the brain, with divergent functional systems recruited for the analysis of speech-, affective, and identity-related features. Although language may be unique to humans, vocal communication is not, and therefore early processing of information wrapped in a voice signal is likely constrained to basic encoding of dynamic, rapid, acoustic changes phylogenetically related to non-human primate models (Davis & Johnsrude, 2007; Fitch

et al., 1997; Ghazanfar & Hauser, 1999; Rauschecker, 1998; Rauschecker & Scott, 2009; Scott & Johnsrude, 2003). In humans, cortical processing begins in the primary auditory cortex (A1) with structural encoding of input from the ascending auditory pathway (Belin et al., 2004). The bilateral posterior superior temporal gyri (STG) are recruited for the identification of relevant features signifying the presence of a communicative voice signal requiring further processing. These regions are involved in the extraction of spectrotemporal templates for different classes of complex sounds, including the voice (Griffiths & Warren, 2004; Warren et al., 2005a,b). Although the posterior superior temporal gyri are not specialized for voice per se, activation in these regions is preferential for complex voice signals in comparison to other environmental sounds (Pernet et al., 2007). At this point, speech processing pathways diverge into a left hemisphere-lateralized dorsal ("what") stream engaged in sound-to-action mapping and a ventral ("where/how") stream engaged in sound-to-meaning mapping and localization involving both left and right hemispheres (Hickok & Poeppel, 2004, 2007; Rauschecker, 2011, 2018). The dorsal stream converges with the aforementioned substrates of speech action-perception underlying self-monitoring theories of AVH. However, further analysis into the involvement of sound-to-meaning processing in voice signals may provide more insight into the phenomenological presentation of hallucinated voice (e.g., speech content, affective state, or identity of the perceived speaker).

An important distinction when assessing the distributed voice processing network, is to isolate subsystems or components of interest (e.g., serving speech, language, or speaker evaluation). Within sound-to-meaning (auditory object) voice processing, we can further distinguish between speech-related linguistic ("what") and speaker-related paralinguistic ("who") features (Belin et al., 2000; Formisano et al., 2008). Although these processes may interact, they are predominantly biased to specialized hemispheric pathways. The left hemisphere is more involved in linguistic information and the right in paralinguistic analysis such as speaker identity, and emotion. This asymmetrical processing may be the result of general hemispheric specialization, delineating a left-side advantage in temporal resolution processing and a ride-side in spectral, benefitting speech and tonal information processing, respectively (Belin & Zatorre, 2000; Zatorre et al., 2002; Zatorre & Gadour, 2008; see rebuttal in McGettigan

& Scott, 2012). Furthermore, for both linguistic and paralinguistic subsystems, processing of increasingly complex information appears to be processed hierarchically. In the right-hemisphere paralinguistic pathway, hierarchical processing proceeds along a posterior to anterior gradient (Belin & Zatorre, 2003, Nakamura et al., 2001; von Kriegstein et al., 2003, von Kriegstein and Giraud, 2004). Low-level acoustic components containing paralinguistic information are identified, allowing the posterior STG to detect a change in voice source (Belin et al., 1999; Warren et al., 2003, Warren et al., 2005a). For voice identity processing, a multi-stage process outlines the evaluation of low-level acoustic features relevant to speaker identification in the posterior STG, followed by prototype comparison and deviance detection in the middle STG, and finally identity recognition in the anterior STG (Maguinness et al., 2018). Importantly, this hierarchy may not be serial, as cross-modal information, or the interaction of top-down and bottom-up signals may modulate the activity of the system (Warren et al., 2006). For example, attribution of identity may be influenced by visual information such as faces (Blank et al., 2011, 2014; von Kriegstein et al., 2005; Watson et al., 2014a; Yovel & Belin, 2013), an interaction between auditory, visual, and affective cues (Davies-Thompson et al., 2019; Ethofer et al., 2013; Stevenage & Neil, 2014; Watson et al., 2014b), or selective attention to task-relevant voice cues (Bonte et al., 2014).

It has been suggested that voice-related activity most accurately represents a general processing of dynamic auditory information such as picking out innately salient signals from a noisy environment (reviewed in Grandjean, 2021). The theory of abnormal salience attribution in the emergence of false perceptions (i.e., over-weighted priors in perceptual inference) is congruent with functional connectivity findings indicating an abnormal switching to active sensory processing during irrelevant stimuli (Menon et al., 2011; Palaniyappan & Liddle, 2012; Schmidt et al., 2015). It is possible that an initial salience attribution issue jump starts prefrontal top-down signaling from speech production, memory, and emotion systems to drive the phenomenology of AVH perception. Voice stimuli from the environment are considered highly salient, and therefore may serve as the ideal trigger for the abnormal engagement of the voice processing network leading to the emergence of AVH. Although this theory appears to reject self-monitoring theories of AVH which describe the impetus of the false

perception as weak predictive signals, it indeed does not stand in full opposition. For example, the hallucinatory percept may emerge through hypersalient cortical responses, yet the perception must still be guided phenomenologically by voice production regions as well as misattributed to an external source, indicating deficient self-monitoring. This dissertation provides an exploration into an association between this putative early-stage hypersalient voice processing as one's subjective proneness for hallucinatory experience.

2 Aims and methods of the dissertation

The goal of this dissertation is to provide new insight into the cognitive mechanisms and neural correlates underlying the proneness to hallucinate. This is realized through a comprehensive research project that combines functional neuroimaging meta-analyses, established and novel functional tasks, and structural analyses on the pattern of diffusion and connectivity of white matter tracts of the brain. Multiple theories have been put forward that attempt to describe the emergence of hallucinations and emphasize the key role of different regions across the brain. This research is approached primarily from the perspective that hallucinations are a consequence of incorrect prediction or monitoring of sensorimotor feedback with an emphasis on the role of cerebellar pathways. Alternatively, theories of cortical neural coding suggest a general difference in balancing top-down and bottom-up inputs leading to the presence or propensity to experience false perceptions. Additional conceptualizations of self-monitoring have prominently described auditory hallucinations as an erroneous processing of prefrontal speech planning and production signals by the temporal cortex as the voice of another person. Although all these explanations are sufficient in describing the overactivity of sensory cortices during hallucinations – both as the result of external noise or the misattribution of internal processes – it is unclear if these processes are mutually exclusive or comprise a common dysfunction affecting multiple pathways. The individual experiments within this dissertation have been successively arranged and conducted to describe how putative hallucination-related substrates compare to a subjective proneness for hallucinatory experience. Furthermore, the dissertation aims to present these findings within samples of the general population, to

avoid the confounds of previous hallucination research in clinical samples often limited to schizophrenia.

2.1 Measure of hallucination proneness

In two of the four original studies presented in the dissertation (**chapter 3** and **chapter 5**), the subjective level of hallucination proneness (HP) of each participant was determined. This measure can be acquired using the adapted Launay-Slade Hallucination Scale (LSHS) revised scale (Larøi & Van Der Linden, 2005a). In contrast to metrics which assess the severity of positive symptoms in psychotic disorders (e.g., Scale for the Assessment of Positive Symptoms: SAPS, the Positive and Negative Syndrome Scale: PANSS, or the Psychotic Ratings Symptom Scale: PSYRATS), the LSHS is particularly effective in providing quantifiable indications of the level of susceptibility one has for transient false or abnormal perceptual experience. For this reason, the LSHS is a reliable tool applicable to the general population and can provide a measure of proneness along a continuum ranging from overt hallucinatory experience to normal states (Waters et al., 2003).

Originally developed in 1981 to investigate the link between aggressive-paranoid tendencies and HP in prison inmates (Launay & Slade), the LSHS originally consisted of 12 true or false items related to intrusive and vivid thoughts, daydreams, as well as auditory and visual hallucinations. In the three following decades, the tool has been revised and adapted multiple times, including multiple principal component and validation analyses. In 1985 (Bentall & Slade), the scale was first revised with the introduction of a 5-point Likert scale rating system. The application of the revised LSHS (LSHS-R) was influential in hallucination research, as principal component analyses determined that this tool was capable of both separating subclinical and clinical types of auditory hallucinations (Levitan et al., 1996), and supported HP as a multidimensional construct (Aleman et al., 2001). In the early 2000s, the LSHS-R was accordingly modified to better incorporate the diverse dimensions of HP. Morrison, Wells, and Nothard presented an adapted version with additional visual hallucination items increasing the total amount to 16 (2000). Subsequently, this adaptation was expanded to dimensions of tactile and olfactory hallucinations as well as the feeling of the presence of someone close who passed away (Larøi et al., 2004). Furthermore, items

referring to God or hearing the devil were removed due to low variability and response rate (Larøi et al., 2004). Finally, a set of follow-up questions for each item was incorporated to assess various characteristics associated with prevalence such as frequency, degree of control, emotional reaction, relationship to stressful events, and personal saliency (Larøi & Van der Linden, 2005a).

Versions of the LSHS have been applied in various non-clinical studies exploring the link between HP and associated characteristics, for example, other psychosis-related traits like delusions, paranoia, and dissociation (e.g., Alderson-Day et al., 2014; Allen et al., 2006; Bradbury et al., 2009; Cella et al., 2008; Larøi, Van der Linden, 2005b; Morrison et al., 2000; Pilton et al., 2015; Stirling et al., 2007; Varese et al., 2011). Moreover, LSHS research has provided insight on cognitive mechanisms associated with psychosis such as thought control, meta-cognitive beliefs, source-monitoring, reality-monitoring (e.g., Collignon et al., 2005; Jones & Fernyhough, 2009; Larøi & Van der Linden, 2005b; Morrison et al., 2000; Perona-Garcelan et al., 2013; Stirling et al. 2007; Varese et al., 2011), as well as speech and language processing (e.g., Alderson-Day et al., 2014; Allen et al., 2006; McCarthy-Jones & Fernyhough, 2011; Vercammen & Aleman, 2010). Other comorbidities with HP such as anxiety, depression, and childhood trauma have been assessed using this tool (Berry et al., 2018; Jones & Fernyhough, 2009; Morrison et al., 2000; Varese et al., 2012).

In the current dissertation, the Larøi & Van der Linden adaptation of the LSHS-R is used (2005a). The questionnaire consists of 16 items relating to the proneness of tactile, sleep-related, visual, and auditory hallucinations, as well as vivid thoughts and daydreaming. The total LSHS-R score provides a powerful indication of overall proneness driven by various factors capturing the multidimensional nature of HP. However, as previous principal component analyses have successfully shown segregation between factors and therefore separate analysis and comparison is of value (e.g., Aleman et al., 2001; Larøi et al., 2004; Larøi & Van der Linden, 2005a; Levitan et al., 1996; Morrison et al., 2000). As the projects involved are closest tied to AVH, analyses of auditory item subtotals in addition to total score are included.

2.2 Functional magnetic resonance imaging

All four studies within the current dissertation include the analysis of magnetic resonance imaging (MRI) data collected by our team or metadata from previous literature. MRI is an established neuroimaging tool commonly in use since the 1970s for diagnostic and research purposes (for a historical review see Viard et al., 2021). Through the application of magnetic fields and radio wave pulses inside the MRI scanner, a matrix of data is acquired to generate detailed 2D and 3D images (detailed description of MRI physics in Currie et al., 2013; Plewes & Kucharczyk, 2012). First, a magnetic gradient longitudinal to the orientation of the body aligns the spin of hydrogen protons. Consecutively, a sequence of radio wave pulses is introduced which temporarily realigns the resonance perpendicular to the longitudinal magnetization, producing a wave signal detectable by the scanner coils. The difference in relaxation time across the voxels allows for the distinction of tissues and the creation of contrast images. Furthermore, by altering the pulse sequence various contrast images can be produced, while an assortment of data at each voxel can be used to analyse different anatomical or physiological processes (Bitar et al., 2006; Young et al., 2020).

In the early 1990s, MRI technology was extended to mapping of activations in the brain (for a historical review see Bandettini, 2012). Although other tomographic imaging methods had previously existed, able to model differential sustained states of brain activation, this form of functional MRI (fMRI) offers several benefits which ultimately led to a burgeoning of empirical findings across neuroscience over the past 30 years. First, it allows for more detailed analysis of the time-course of activations. Second, in addition to contrasting blocks of sustained activation patterns, fMRI can model activations relative to stimulus presentation via event-related paradigms (Amaro Jr. & Barker, 2006). Signal detection in fMRI employs a long-standing discovery that the magnetic susceptibility of haemoglobin changes based on its level of oxygenation (Pauling & Coryell, 1936). When an area of the brain becomes active, energy is expended and oxygen is consumed, leading to an initial rise in deoxyhemoglobin concentration followed by a compensatory increase in blood flow and oxygenated haemoglobin and the eventual return to baseline levels. Due to the paramagnetic properties of deoxyhemoglobin and paramagnetic of oxyhemoglobin, a shift in magnetic

susceptibility can be detected, referred to as the blood-oxygen-level-dependent (BOLD) signal (Gauthier & Fan, 2019; Kim & Ogawa, 2012; Logothesis & Wandell, 2004; Ogawa et al., 1998). Much research has attempted to deconstruct the physiological or neuronal basis of this signal, attributed to temporally discrete components of neuro-vascular coupling including changes in blood volume or flow, oxygen depletion, or metabolic rate (Attwell & Iadecola, 2002; Buxton & Frank, 1997; Jueptner & Weiller, 1995; Raichle & Mintun, 2006). However, in fMRI analysis, net changes in BOLD signals are commonly interpreted as an indirect measure of stimulus- or state-attributed neural activity (Heeger & Ress, 2002; Logothesis, 2008).

Two fMRI tasks were conducted within the dissertation project utilising BOLD contrast estimates, comparing different conditions via event-related paradigms. The first is a widely used functional localizer task which identifies temporal and extra-temporal areas of the cerebral cortex sensitive to voice stimuli (Belin et al., 2000). In **chapter 3**, this localizer for an ROI analysis is used, where contrast estimate values for voice versus silence (voice sensitive), non-voice versus silence (environment sensitive), and voice versus non-voice (voice preference) are extracted and correlated them to subjective levels of hallucination proneness. In **chapter 4**, this localizer task is used to determine a specific voice sensitive region of interest to build a mask for parameter extraction in a voice perception task assessing motor-induced suppression and error-monitoring activity in self- and other-attributed voice signals (vowel excerpts /a/ and /o/). The task was adaptation from an EEG paradigm capable of eliciting prediction error responses to self-initiation vowels independent of vocal production, via button-press (Knolle et al., 2019; Pinheiro et al., 2020b). A 2x3 factorial design was used, with factors of source (self-generated and passively-heard) and voice (self, uncertain, and other). Uncertain self-other voice identity stimuli were produced using a morphing software (STRAIGHT: Kawahara 2003, 2006). An event-related design of randomly-ordered stimuli conditions was selected to avoid fMRI adaptation of the auditory HRF response (Barron et al., 2016; Grill-Spector et al., 2006; Larsson et al., 2016). A delayed-acquisition technique was employed to allow for stimulus to be acquired during periods of relative silence (Belin et al., 1999; Hall et al., 1999).

2.3 Activation-likelihood meta-analysis

Although many functional neuroimaging studies have experimentally altered self-generated sensory feedback, no clear consensus on the brain regions involved had been previously presented. For this reason, a meta-analysis was conducted on pre-existing experiments altering either visual or auditory stimuli to model brain regions responsive to modality-specific or multi-modal feedback error (**chapter 2**). A coordinate-based technique was employed known as activation-likelihood estimation (ALE) (www.brainmap.org/ale). This technique differs from other quantitative models of neuroimaging meta-analysis, as it incorporates each focus in standard space weighted against the n from the contributing study. Each of the activation foci is then modelled as a probability distribution of spatial uncertainty, and the resulting output is a set of coordinates representing those likely to share spatial association (Eickhoff et al., 2009, 2012; Laird et al., 2005).

2.4 Diffusion tensor imaging

To investigate how white matter structure varies with hallucination proneness, diffusion-weighted imaging (DWI) was acquired. DWI is an MRI method that detects the movement of water within organic tissue (Le Bihan, 1986). The science of MR spectroscopy and pulse gradient techniques underlying DWI were discovered several decades ago (Carr & Purcell, 1954; Stejskal & Tanner, 1965), yet development of the method was impeded by technical constraints of the time. Only in the 1990s was it was proposed that tensors quantifying the directionality of water diffusion could be modeled using DWI data (diffusion tensor imaging: DTI), allowing for interpretations regarding the structure, architecture, and orientation of the underlying tissue (Basser et al., 1994). A tensor at each voxel is calculated from the magnitude (eigenvalues) of diffusion in three perpendicular directions (eigenvectors). Using the eigenvalues and eigenvectors, multiple properties of diffusion can be inferred at each voxel (detailed description in Kubicki et al., 2002; O'Donnell & Westin, 2012; Soares et al., 2013). These can be quantified along the main axis of diffusion (axial diffusivity: AD), transverse to the main axis (radial diffusivity: RD), the mean rate across all directions (mean diffusivity: MD), and perhaps most importantly, the preferential direction of diffusion (fractional anisotropy: FA). Additionally, the FA value can indicate within each voxel how evenly

(isotropic) or specific (anisotropic) water diffusion appears. DTI interpretation often attributes change in diffusion patterns to variability in myelin or axonal differences which putatively restrict the flow of diffusion. Measures of FA were applied as the primary measure of diffusion directionality as well as secondary measures of MD, AD, and RD for interpretation of findings. Furthermore, probabilistic tractography was used as a tool for modelling specific pathways of the brain and to quantify streamlines as a measure of connectivity. Finally, diffusion and connectivity data to LSHS scores were compared to assess the relationship between white matter structure and HP.

3 Outline of the dissertation

Following the historical and theoretical account of research describing the emergent phenomenon of hallucinations as false perceptions, **chapter 2** opens the empirical part of this dissertation. In this section, a meta-analysis is provided focusing on all regions across both auditory and visual bodies of functional neuroimaging studies responsive to errors in predicted feedback of self-generated stimuli. The aim of this study was two-fold: to determine if feedback-error regions are specific or non-specific to a sensory modality and if the role of the cerebellum in processing unpredicted sensory feedback is detectable in fMRI as indicated by this body of research. Additionally, as theories of hallucination research posit a central role of misattributed self-generated feedback to another agent, the neural substrates of perceived internalization versus externalization of sensation during action are outlined. The following fMRI investigation in **chapter 3** is focused on how voice sensitive areas may be systematically related to the proneness of a hallucinatory experience. Moreover, this chapter investigates if hallucination proneness relates to changes in brain activity without the involvement of the motor system and is detectable in hypersensitivity to passive sensory stimuli. The findings from these chapters cumulatively informed the fMRI investigation reported in **chapter 4** which explored if feedback-error and voice-processing regions are affected by unexpected changes to the quality of one's own voice during action. Finally, **chapter 5** presents a DTI study that outlines if and how structural changes to white matter pathways involved in prediction, auditory and salience processing underlie sensitivity to subjective levels of hallucination proneness. This investigated a possible correlation between HP and white matter structural changes in pathways selected as reported in

literature for patients with AVH and putative roles in the mechanisms hypothesized to be associated with these experiences. The discussion provided in **chapter 6** elaborates on the involvement of theories underlying prediction-perception and action-perception models in the findings across chapters 2-5. Additionally, the interpretations are re-integrated into established models of psychotic illness in search of coherence with continuum theories or a vulnerability to transition to clinical expression.

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Chapter 2

The role of the cerebellum in adaptation: ALE meta-analyses on sensory feedback error

Johnson, J.F., Belyk, M., Schwartze, M., Pinheiro, A.P., & Kotz, S.A.

Abstract. It is widely accepted that unexpected sensory consequences of self-action engage the cerebellum. However, we currently lack consensus on where in the cerebellum, we find fine-grained differentiation to unexpected sensory feedback. This may result from methodological diversity in task-based human neuroimaging studies that experimentally alter the quality of self-generated sensory feedback. We gathered existing studies that manipulated sensory feedback using a variety of methodological approaches and performed activation likelihood estimation (ALE) meta-analyses. Only half of these studies reported cerebellar activation with considerable variation in spatial location. Consequently, ALE analyses did not reveal significantly increased likelihood of activation in the cerebellum despite the broad scientific consensus of the cerebellum's involvement. In light of the high degree of methodological variability in published studies, we tested for statistical dependence between methodological factors that varied across the published studies. Experiments that elicited an adaptive response to continuously altered sensory feedback more frequently reported activation in the cerebellum than those experiments that did not induce adaptation. These findings may explain the surprisingly low rate of significant cerebellar activation across brain imaging studies investigating unexpected sensory feedback. Furthermore, limitations of functional magnetic resonance imaging to probe the cerebellum could play a role as climbing fiber activity associated with feedback error processing may not be captured by it. We provide methodological recommendations that may guide future studies.

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1 Introduction

To successfully act within a dynamic environment, we continuously monitor sensory feedback associated with our own movements to ensure our actions have the desired outcomes. Even the simplest movements require complex coordination between multiple effectors. The continuous monitoring of sensory feedback helps to refine motor plans and adjust them to contextual and environmental changes. The forward model is a computational process that compares expected to actual sensory consequences of an action [Figure 1] (Jordan & Rumelhart, 1992; Miall & Wolpert, 1996; Wolpert, 1997). This comparison is essential to motor control and relies partly on the cerebellum (Blakemore et al., 2001; Ishikawa et al., 2016; Ito, 1984a; Kawato et al., 1987; Miall et al., 1993; Wolpert et al., 1998). However, there is still no strong consensus on where in the cerebellum unexpected sensory feedback is processed. To this end, the current meta-analysis systematically explores patterns of cerebellar activation in neuroimaging studies of sensory feedback manipulations. Such manipulations create an artificial mismatch between intended and perceived sensory consequences of an action, constituting methods commonly used to probe forward models in brain imaging studies.

A fundamental component of the forward model is the efference copy: when the motor cortex sends a command to the peripheral nervous system for the execution of motor behavior, a copy of the command is fed forward to provide an estimate of the sensorimotor feedback predicted from the movement (von Holst & Mittelstaedt, 1950). The predicted feedback is compared to the actual feedback from the proprioceptive (Miall & Wolpert, 1996; Wolpert & Kawato 1998), visual (Leube et al., 2003), auditory (Hashimoto & Sakai, 2003), and tactile (Blakemore et al., 1998) sensory periphery. Any discrepancy between predicted and actual feedback constitutes an output known as a corollary discharge (Feinberg, 1978). The resultant error signal is processed by the cerebellum, ultimately resulting in an output to cortical areas to adaptively fine-tune behavior. As both the state of the organism and the environment are dynamic, forward models are employed continuously to reduce any discrepancy between predicted and actual feedback through constant monitoring and adjustment of behavior (Desmurget & Grafton, 2000; Jordan & Rumelhart, 1992).

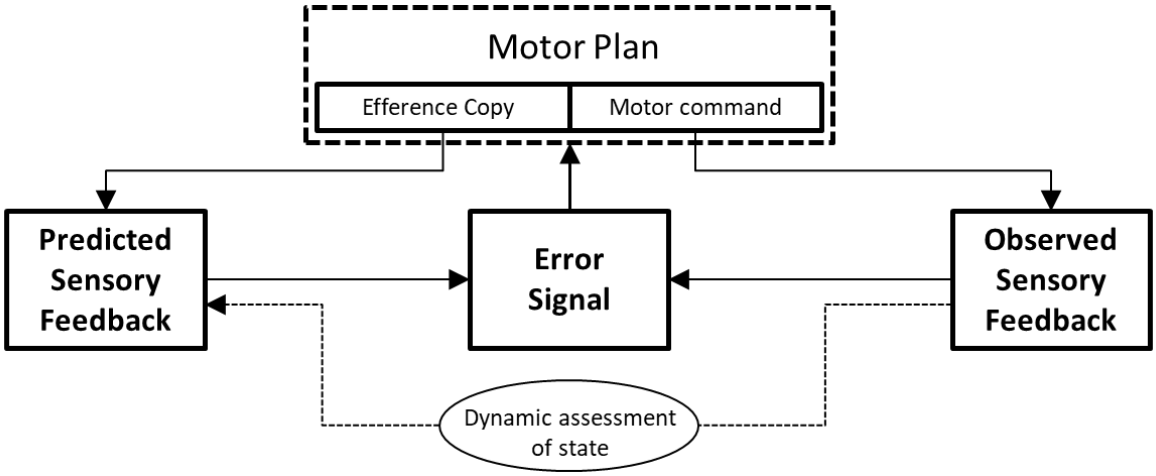


FIGURE 1. *Components of a forward model. A diagram which outlines five major components of a Forward Model. The "Motor Plan" incorporates the components (1) "Efference Copy" and (2) "Motor Command." The implantation of the Motor Command leads to (3) "Observed Sensory Feedback." The efference copy is an expectation of sensory consequences of the enactment of the motor plan, providing (4) "Predicted Sensory Feedback." The Observed Sensory Feedback and the Predicted Sensory Feedback are compared. If they do not match, an (5) "Error Signal" indicating violation of the expected consequences is returned for updating of the motor plan. The "Observed Sensory Feedback" can also notify the "Predicted Sensory Feedback" with contextual information of body or environment in order to make temporary changes to the prediction rather than updating the motor plan. This is denoted as "Dynamic assessment of state"*

The cerebellar cortex comprises overlapping functional zones that process input from specific sensory modalities (Witter & De Zeeuw, 2015). Specific areas respond to auditory or visual stimuli (O'Reilly et al., 2009; Petacchi et al., 2005; Sang et al., 2012). Likewise, portions of the cerebellum segment into somatomotor topographies associated with the control of different body parts (Buckner et al., 2011; Mottolese et al., 2013). These divisions are coupled to associated subdivisions of motor cortex responsible for controlling the same body parts. The motor areas contribute cortical input to communication loops between the cortex and cerebellum employed in ongoing motor control. Therefore, the cerebellar cortex receives two principal types of afferents: mossy fibres primarily via the pons which relay information such as the efference copy from corresponding cortical regions (Raymond et al., 1996), while medullary nuclei relay bottom-up sensory feedback-related signals and induce changes in the influence of cortical top-down signals to the cerebellar cortex (Ito, et al., 1982). The inferior olive monitors the discrepancy between predicted and actual sensory input and relays an error signal to the cerebellum via climbing fibers (Ito, 1984b; Kawato & Gomi, 1992).

The cerebellum also receives signals of unpredicted auditory feedback from the dorsal cochlear nuclei of the medulla (Schwartze & Kotz, 2013). In turn, the cerebellum continuously signals the discrepancy back to the cerebral cortex to induce adaptation of motor behaviour until the expected feedback matches the actual sensory feedback.

In order to guide the complex task of adapting behaviour to resolve discrepancy between expected and actual sensory feedback, the cerebellum must communicate and coordinate with multiple areas of the cerebral cortex. All output from the cerebellum, such as from the processing of the corollary discharge, are issued via deep cerebellar nuclei (Middleton & Strick, 1997), connecting to regions such as the prefrontal cortex (Allen et al., 2005a; Balsters et al., 2010; Kelly & Strick, 2003; Middleton & Strick, 2001; Ramnani, 2012; Watson et al., 2014c), motor areas (Akkal et al., 2007; Balsters et al., 2010; Dum & Strick, 2003; Kelly & Strick, 2003; Lu et al., 2012; Wise & Strick, 1984), and parietal area (Allen et al., 2005a; Clower et al., 2001; Prevosto et al., 2009; Ramnani, 2012). The cerebellum and these frontal and parietal areas also create loops where reciprocal exchange of information can be continuously fed through a cortico-cerebellar system to allow for continuous adaptation of motor activity (Watson et al., 2014c).

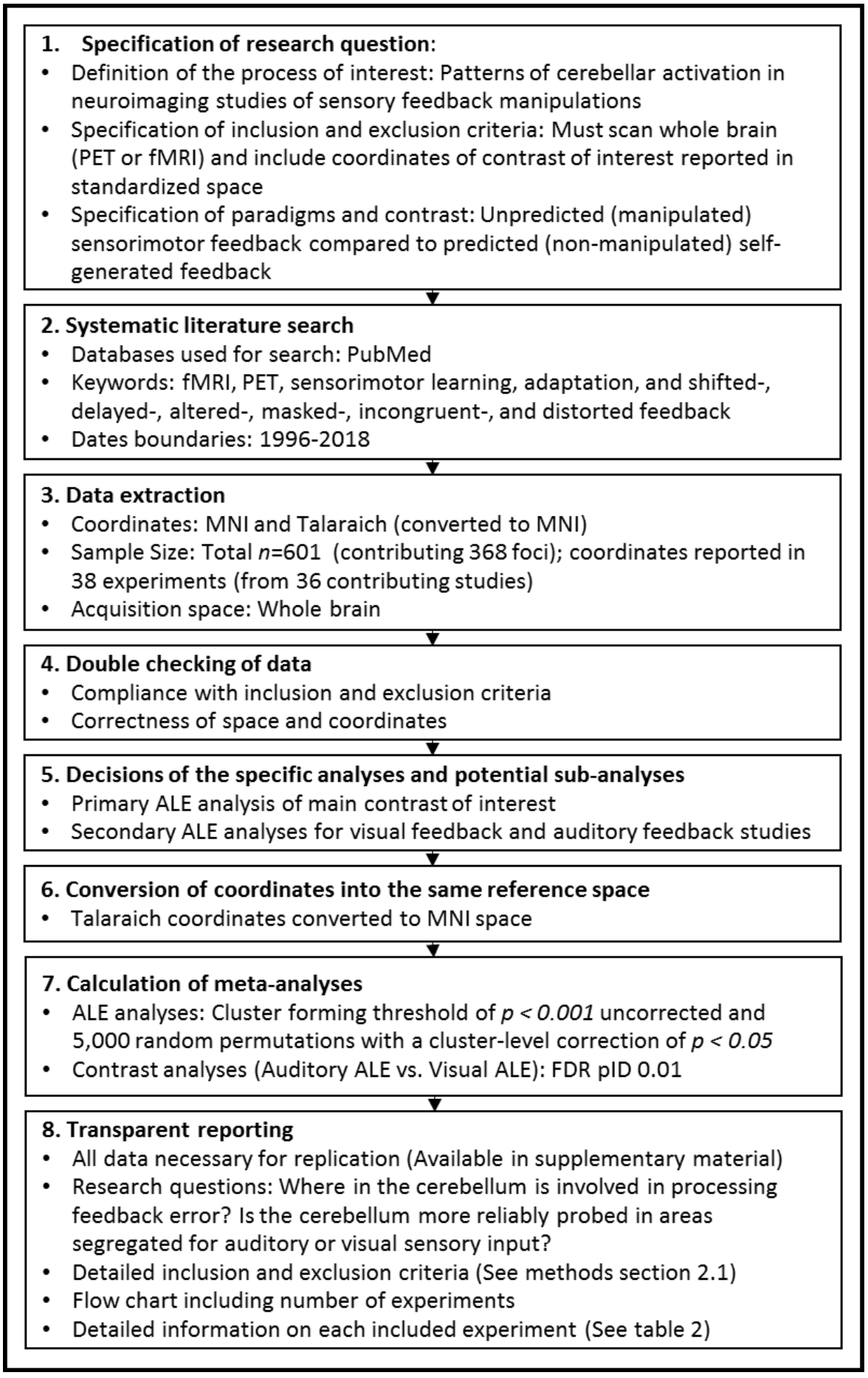
A common experimental approach to study neural substrates that implement processes related to the forward model is to manipulate the sensorimotor feedback of self-generated movements. In these experiments, participants typically produce articulatory or manual movements while feedback is altered. For example, studies have investigated how the brain responds to unpredicted feedback by manipulating the acoustic properties of one's own voice (Tourville et al., 2008; Zheng et al., 2013), by introducing illusory visual displacement of the hand or a mechanically controlled avatar (David et al., 2007; Diedrichsen et al., 2005; Schnell et al., 2007), or by applying an unpredicted external physical force (Diedrichsen et al., 2005; Golfinopoulos et al., 2011). Therefore, there can be much variability in how this mechanism is studied in neuroimaging research in terms of form of motor production, feedback manipulation, and sensory modality of feedback. It was our intention to evaluate over the body of literature eliciting activity in response to various manipulations of self-generated sensory feedback any common areas reported in the brain, with specific interest in

finding consensus on the regions of the cerebellum involved. We conducted three activation likelihood estimation (ALE) meta-analyses of functional neuroimaging studies to identify patterns of neural activation that are reliably affected by these manipulations. A primary analysis was expected to yield a modality independent but anatomically precise global impression of cerebellar contributions to processes related to the forward model. Two secondary ALE analyses were conducted to differentiate this impression in terms of potential modality-specific components, as well as a contrast analysis between auditory and visual feedback results. This distinction is made as although higher processing cortical areas may be responsive irrespective of the sensory modality, there is cerebellar and cortical distinction between areas responsive to auditory and visuomotor feedback manipulations. In doing so, we may shed light on a consensus of where the cerebellum is involved in processing feedback error, and specifically if the cerebellum is more reliably probed in areas segregated for auditory or visual sensory input. Additionally, due to the high diversity in methods across this body of literature, we aimed to further investigate the dependency of cerebellar activity reported on the factors which most commonly varied across the experiments selected for our meta-analyses.

2 Materials and Methods

We generally followed recent best practice guidelines for the conducting of neuroimaging meta-analyses (Müller et al., 2017). These guidelines have been put forward to improve the transparency and replicability of meta-analyses. We accordingly report information advised such as research question, inclusion and exclusion criteria, detailed information for all experiments, and a step-by-step flowchart [see Figure 2].

FIGURE 2. *Meta-analysis flowchart. A flowchart diagram recommended in the best practice guidelines for the conducting of neuroimaging meta-analyses (Müller et al., 2017). The flowchart lists eight sections. (1) Specification of research question. (2) Systematic literature search. (3) Data extraction. (4) Double checking of data. (5) Decisions of the specific analyses and potential sub-analyses. (6) Conversion of coordinates into the same reference space. (7) Calculation of meta-analyses. (8) Transparent reporting. Each section is outlined to provide the reader with all necessary information for replication.*


1. Specification of research question:
• Definition of the process of interest: Patterns of cerebellar activation in neuroimaging studies of sensory feedback manipulations
• Specification of inclusion and exclusion criteria: Must scan whole brain (PET or fMRI) and include coordinates of contrast of interest reported in standardized space
• Specification of paradigms and contrast: Unpredicted (manipulated) sensorimotor feedback compared to predicted (non-manipulated) self-generated feedback
2. Systematic literature search
• Databases used for search: PubMed
• Keywords: fMRI, PET, sensorimotor learning, adaptation, and shifted-, delayed-, altered-, masked-, incongruent-, and distorted feedback
• Dates boundaries: 1996-2018
3. Data extraction
• Coordinates: MNI and Talaraich (converted to MNI)
• Sample Size: Total n=601 (contributing 368 foci); coordinates reported in 38 experiments (from 36 contributing studies)
• Acquisition space: Whole brain
4. Double checking of data
• Compliance with inclusion and exclusion criteria
• Correctness of space and coordinates
5. Decisions of the specific analyses and potential sub-analyses
• Primary ALE analysis of main contrast of interest
• Secondary ALE analyses for visual feedback and auditory feedback studies
6. Conversion of coordinates into the same reference space
• Talaraich coordinates converted to MNI space
7. Calculation of meta-analyses
• ALE analyses: Cluster forming threshold of p < 0.001 uncorrected and 5,000 random permutations with a cluster-level correction of p < 0.05
• Contrast analyses (Auditory ALE vs. Visual ALE): FDR pID 0.01
8. Transparent reporting
• All data necessary for replication (Available in supplementary material)
• Research questions: Where in the cerebellum is involved in processing feedback error? Is the cerebellum more reliably probed in areas segregated for auditory or visual sensory input?
• Detailed inclusion and exclusion criteria (See methods section 2.1)
• Flow chart including number of experiments
• Detailed information on each included experiment (See table 2)

2.1 Study selection

The PubMed (www.ncbi.nlm.nih.gov/pubmed) database was searched for human neuroimaging studies using combinations of relevant keywords (e.g., functional magnetic resonance imaging [fMRI], positron emission tomography [PET], sensorimotor learning, adaptation, and shifted-, delayed-, altered-, masked-, incongruent-, and distorted feedback). We further cross-referenced the articles produced from the search term to corroborate that no relevant articles were overlooked. Studies were selected if they reported activation contrasts of unpredicted (manipulated) compared to predicted (non-manipulated) self-generated feedback, data acquisition covered the whole brain encompassing the cerebellum, and included tables listing peak activations in standard stereotaxic space. Only data from healthy adult participants were selected. Results reported in Talairach space (Talairarch & Tournoux, 1988) were converted to MNI (Holmes et al., 1998) using the GingerALE (2.3.6) icbm2tal conversion algorithm (Lancaster et al., 2007). This search yielded experiments from 36 studies, reporting a total of 368 foci of activation from 601 participants, including 16 studies of manipulated auditory feedback from vocalizations or button-presses, and 20 studies of manipulated visual feedback of hand movements [Table 1].

TABLE 1A | VISUAL FEEDBACK STUDIES IN META-ANALYSIS

Study	*N*	*Manipulation*	*Imaging*	*# of Foci*
Anguera et al. (2010)	21	Spatial shift	3 T	18
Backasch et al. (2014)	16	Temporal	3 T	6
Balsev et al. (2017)	15	Temporal	3 T	4
Brand et al. (2017)	14	Spatial shift	3 T	13
David et al. (2007)	14	Mismatch	1.5 T	11
Diedrichsen et al. (2005)	39	Spatial shift	3 T	8
Farrer et al. (2008)	15	Temporal	1.5 T	10
Fink et al. (1999)	10	Mismatch	PET	1
Grafton et al. (2008)	10	Spatial shift	1.5 T	14
Graydon et al. (2005)	24	Spatial shift	4 T	12
Inoue et al. (2000)	6	Spatial shift	PET	19
Krakauer et al. (2004)	12	Spatial shift	PET	7
Leube et al. (2003)	18	Temporal	1.5 T	12
Limanowski et al. (2017)	16	Temporal	3 T	5
Ogawa et al. (2007)	17	Temporal	1.5 T	3
Seidler et al. (2006)	26	Spatial shift	3 T	12
Schell et al. (2007)	15	Mismatch	1.5 T	11
Spaniel et al. (2015)	35	Spatial shift	3 T	2
Tunik et al. (2013)	12	Mismatch	3 T	12
Yomogida et al. (2010)	28	Mismatch	1.5 T	6

TABLE 1B | AUDITORY FEEDBACK STUDIES IN META-ANALYSIS				
Study	***N***	***Manipulation***	***Imaging***	***# of Foci***
Behroozmand et al. (2015)	8	Acoustic shift	3 T	16
Christoffels et al. (2007)	14	Noise mask	3 T	3
Fu et al. (2005)	13	Acoustic shift	1.5 T	6
Golfinopoulos et al. (2011)	13	Physical	3 T	52
Hashimoto & Sakai (2003)	15	Temporal shift	1.5 T	6
Kleber et al. (2013)	22	Noise mask	3 T	22
McGuire et al. (1996)	6	Acoustic shift	PET	4
Parkinson et al. (2012)	12	Acoustic shift	3 T	6
Pfordresher et al. (2014)	20	Temporal/mismatch	3 T	34
Sakai et al. (2009)	10	Temporal	1.5 T	9
Takaso et al. (2010)	8	Temporal	PET	4
Tourville et al. (2008)	11	Acoustic shift	3 T	18
Toyomura et al. (2007)	12	Acoustic shift	1.5 T	6
Zarate & Zadorra (2008)	12	Acoustic shift	1.5 T	6
Zheng et al. (2010)	21	Acoustic shift	3 T	5
Zheng et al. (2013)	20	Acoustic shift	3 T	4

TABLE 1*. N: participants contributing to pooled dataset. Imaging method: MRI or PET imaging equipment. Number of foci: contributing to pooled dataset.* ***(A) Visual feedback studies****. Feedback manipulation: temporal and spatial shifts, and random mismatch between action and feedback.* ***(B) Auditory feedback studies****. Feedback manipulation: temporal and acoustic shifts, noise masking, and random mismatch between action and feedback.*

2.2 ALE analyses

The software package GingerALE (2.3.6) was used to perform three analyses on coordinates for peak activations derived from the studies identified by the literature search (www.brainmap.org/ale; Eickhoff et al., 2009; Eickhoff et al., 2012; Laird et al., 2005; Turkeltaub et al., 2012). Three single set ALE and one contrast analysis were conducted. ALE computes the likelihood of a voxel for being a source of activation within the set of studies across the whole brain. A primary ALE analysis was conducted on the full dataset of 38 experiments from the 36 studies included, using a cluster forming threshold of $p < 0.001$ and 1,000 random permutations with a cluster-level correction of $p < 0.05$. These parameters were chosen as they were the recommended threshold settings for the cluster-level multiple test correction listed in the GingerALE software user guidelines. This analysis was performed to identify concordant activations regardless of the feedback modality. A secondary set of ALE analyses was

conducted separately on a dataset of 17 experiments from the 16 studies with manipulated auditory feedback, and on 21 experiments from the 20 studies with manipulated visual feedback. Identical cluster forming threshold and correction parameters for the first ALE were applied to the auditory and visual dataset analyses. Contrast analyses were performed comparing auditory and visual ALE results applying a False Discovery Rate assuming independence or positive dependence (FDR *p*ID) of 0.01, with threshold parameters as recommended in the GingerALE manual.

2.3 Tests of independence

We investigated the relative success of the most commonly applied experimental designs to engage the cerebellum. To this end, we categorized the studies in our dataset by sensory modality (visual vs. auditory), acquisition methods (blocked vs. event-related design), response to manipulated feedback (adaption vs. no-adaptation), and type of feedback manipulation (temporal, spatially or acoustically shifted, masked, mismatched, or physical perturbation). Adaptation studies are categorized as eliciting adjustment in response to sustained manipulation of feedback, where the fine-tuning of motor commands may be relevant to the success of future action. This differs from automatic compensatory responses to brief changes in feedback that do not inform successive behaviour. We tested whether the presence of significant activation within the cerebellum was associated with these methodological factors with a chi-squared test of independence applying the Yates correction of continuity using SPSS version 24 (IBM Corp., Armonk, NY). Significance thresholds were set at $p < 0.05$.

3 Results

3.1 Manipulated feedback: ALE analyses

The primary ALE identified five clusters [Table 2a, Figure 3a]. The largest cluster was centred medially in the superior frontal gyrus extending into the right and left supplementary motor area (SMA) (BA 6). There were two right lateralized clusters in the precentral gyrus (preCG) (BA 9) and inferior frontal gyrus (IFG) pars opercularis (BA 44), as well as two left hemisphere clusters in the supramarginal gyrus (SMG) (BA 40) and SMA (BA 6). The secondary ALE on auditory feedback manipulation identified

six clusters, centred at the left superior temporal gyrus (STG) (BA 22) and right STG (BA 41), with four additional right lateralized clusters at the SMA (BA 6), IFG pars opercularis (BA 44), SMG (BA 40), and primary motor cortex (M1) (BA 4) [Table 2b and Figure 3b]. The secondary ALE on visual feedback manipulation also identified six clusters, centred at the right preCG (BA 6), left hemisphere frontal eye fields (BA 8) and SMA (BA 6), as well as right lateralized clusters at the preCG (BA 6), extrastriate body area (EBA) (BA 37), and SMA (BA 6) [Table 2c, Figure 3c]. Remarkably, both the primary and secondary ALE analyses failed to identify clusters of activation in the cerebellum. The subsequent contrast analysis between auditory and visual feedback error ALE results utilized only clusters obtained in at least one of the three individual ALE analyses for comparison. As no cerebellar clusters were produced in either of the ALE analyses, the contrast analysis did not produce any valuable comparisons of modality specific cerebellar clusters. At a cortical level, the auditory compared to visual ALE cluster contrast produced two clusters in the left and right STG specifically for auditory and not visual feedback error. No significant clusters were found for visual and auditory feedback errors nor any common regions between modality in a conjunction of both ALE analyses clusters [see Supplementary Materials Table S1].

3.2 Relationship between experimental design and cerebellar activation

To determine whether certain methodological differences were linked with being more likely to activate the cerebellum we conducted chi-squared tests of independence for four common design features in which experiments differed from one another. With an a priori significance threshold set to $p < 0.05$, finding cerebellar activation was not contingent on sensory modality ($p = 0.243$), manipulation type ($p = 0.306$), or acquisition method ($p = 0.071$). However, cerebellar activation was contingent on adaptation ($p = 0.005$). Then, 14 of 18 studies (78%) that elicited adaption responses reported significant activation in the cerebellum. In summary, of the four factors tested, cerebellar activation proved to depend significantly on the need for adaptation.

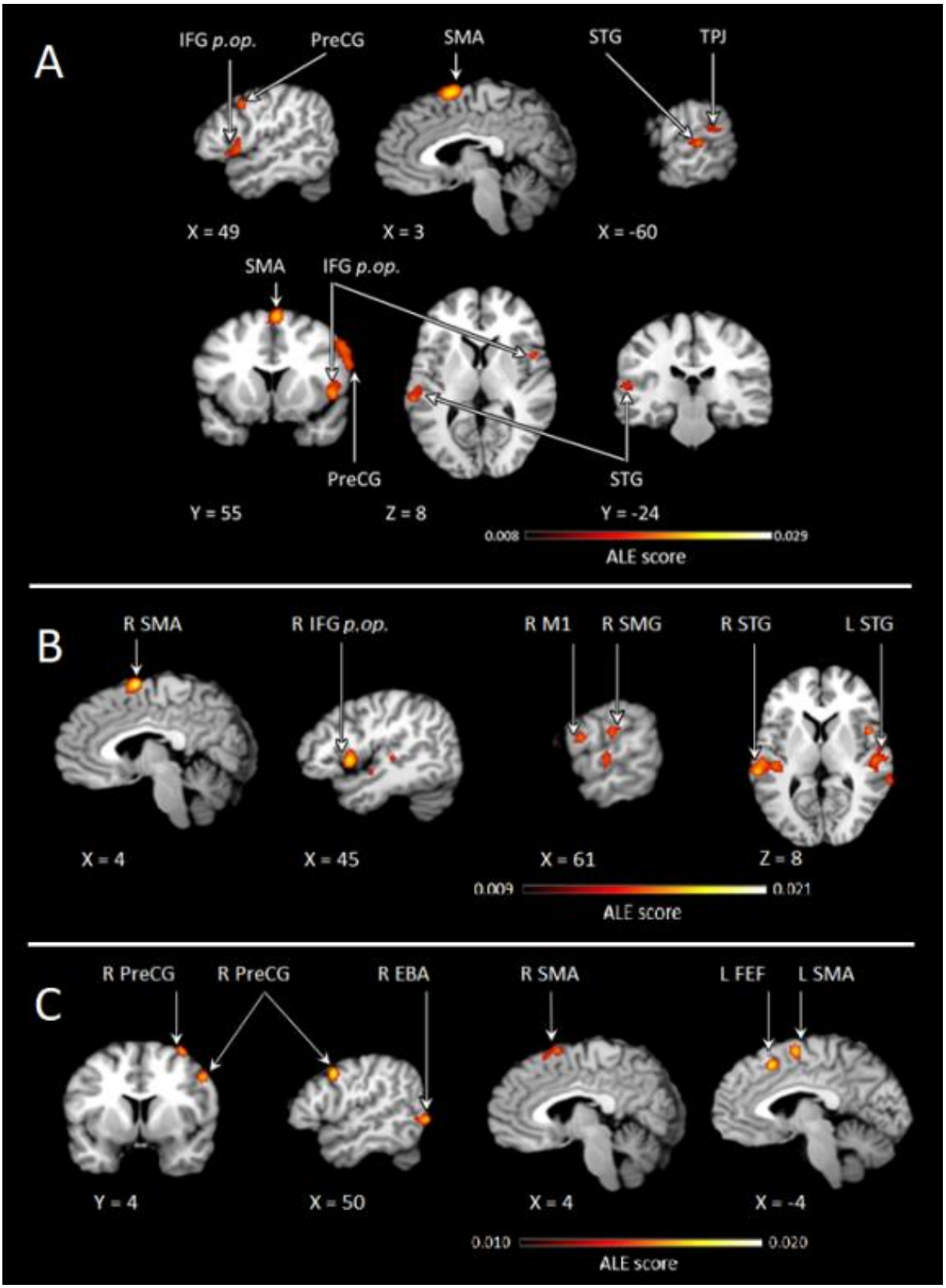

A
IFG p.op.
PreCG
SMA
STG
TPJ
X = 49
X = 3
X = -60
SMA
IFG p.op.
PreCG
STG
Y = 55
Z = 8
Y = -24
0.008
0.029
ALE score
B
R SMA
R IFG p.op.
R M1
R SMG
R STG
L STG
X = 4
X = 45
X = 61
Z = 8
0.009
0.021
ALE score
C
R PreCG
R PreCG
R EBA
R SMA
L FEF
L SMA
Y = 4
X = 50
X = 4
X = -4
0.010
0.020
ALE score

FIGURE 3. *Meta-analyses results. ALE: activation likelihood estimation, MNI: Montreal Neurological Institute, STG: superior temporal gyrus, TPJ: temporoparietal junction, IFG (p.op): Inferior frontal gyrus (pars opercularis), PreCG: precentral gyrus, SMA: supplementary motor area, M1: primary motor cortex, SMG: supramarginal gyrus, FEF: frontal eye fields, EBA: extrastriate body.* ***(A) Sensory feedback error ALE.*** *Images: six slices at MNI space x axis 49, 3, −60, y axis 55, −24, and z axis 8. Units of measurement: ALE scores with a minimum value of 0.008 and maximum of 0.029. A threshold of likelihood calculated from a cluster-forming threshold of p < .001, with a cluster-level correction of 0.05, 1,000 random permutations.* ***(B) Auditory feedback error ALE*** *Images: four slices at MNI space x axis 4, 45, 61, and z axis 8. Units of measurement: ALE scores with a minimum value of 0.000 and maximum of 0.021. A threshold of likelihood calculated from a cluster-forming threshold of p < .001, with a cluster-level correction of 0.05, 1,000 random permutations.* ***(C) Visual feedback error ALE****. Images: four slices at MNI space x axis 50, 4, −4, and y axis 4. Units of measurement: ALE scores with a minimum value of 0.010 and maximum of 0.020. A threshold of likelihood calculated from a cluster-forming threshold of p < .001, with a cluster-level correction of 0.05, 1,000 random permutations.*

TABLE 2 | **META-ANALYSES RESULTS**					
Brain regions	***BA***	***MNI coordinates*** *(x, y, z)*			***Cluster size*** *(mm^3)*
A. Sensory feedback error: Manipulated feedback > non-manipulated feedback					
R/L SMA	6	0.5	3.3	60.3	3,576
R PreCG	9	55.9	9.7	31.7	2,528
R IFG (p. op.)	44	45.6	13.9	1.2	1,824
L STG	42	-57.6	-26	9.2	1,072
L TPJ	22	-61.4	-40.2	19.9	848
B. Auditory feedback error: Manipulated feedback > non-manipulated feedback					
L STG	22	-56.7	-29.6	14.2	6,400
R STG	41	55.5	-19.3	4.8	4,064
R SMA	6	2.5	3.9	61.3	1,792
R IFG (p. op.)	44	45.8	9.7	3.8	1,400
R SMG	40	63.8	-21.1	20.3	832
R M1	4	62.3	-2	18	792
C. Visual feedback error: Manipulated feedback > non-manipulated feedback					
R PreCG	6	51.5	7.3	38.7	1,000
L FEF	8	-4.5	17.2	46.4	856
L SMA	6	-3.9	-2.4	58	768
R PreCG	6	36.4	1.2	58.4	768
R EBA	37	48.9	-68	2.3	712
R SMA	6	4.6	14	60.1	696

TABLE 2. *Abbreviations: ALE, activation likelihood estimation; L FEF, left frontal eye fields; L SMA, left supplementary motor area; MNI, Montreal Neurological Institute; R EBA, right extrastriate body area; R PreCG, right precentral gyrus; R SMA, right supplementary motor area. Abbreviations: BA, Brodmann area; EBA, extrastriate body area; FEF, frontal eye fields; IFG (p. op.), inferior frontal gyrus pars opercularis; M1, primary motor cortex; L, left; PreCG, precentral gyrus; R, right; SMA, supplementary motor area; SMG, supramarginal gyrus; STG, superior temporal gyrus; TPJ, temporoparietal junction.*

4 Discussion

We conducted three meta-analyses of neuroimaging studies that altered sensory feedback during ongoing movements with the aim of localizing the cerebellar contributions to processes related to the forward model. In doing so, we attempted to reconcile an apparent lack of consensus of the role of the cerebellum in experiments of unexpected changes to sensory consequences of our own action. Contrary to our expectations and to broad scientific consensus suggesting that the cerebellum is involved in this process, we did not observe a convergence of activation foci in the cerebellum, although the analyses successfully identified the expected network of cortical areas. We therefore systematically assessed methodological factors that could potentially increase the likelihood of specific experimental designs to activate the cerebellum. Our findings confirm that studies that require adaptation of behaviour in response to sensory feedback manipulations most reliably evoke cerebellar activation.

4.1 Responses to unpredicted feedback

4.1.1 Cerebral cortex. The primary ALE analysis of the manipulated sensory feedback dataset indicated clusters in the SMA, preCG, IFG, STG, and TPJ. Two of the clusters, the SMA and preCG, were centered in the right secondary motor cortex, regions associated with the production of an efference copy for assessing sensory consequences of movement (Christensen et al., 2006; Ellaway et al., 2004; Haggard & Whitford, 2004). The cluster centered at the IFG and extending into the prefrontal frontal regions incorporates areas which lend themselves to a broader self-awareness network and are thought to reflect agency over action (David et al., 2008; Fink et al., 1999; Jardri et al., 2007; Leube et al., 2003; Nahab et al., 2010). The right IFG plays a role in detecting cues which are relevant to inhibiting motor activity (Corbetta & Shulman, 2002; Hampshire et al., 2010) and in subsequent reorienting and updating of action plans (Levy & Wagner, 2011). The cluster centered in the TPJ extends into much of the IPL, which is associated with monitoring motor outflow (Desmurget & Sirigu, 2009; Sirigu et al., 1996), as well as with awareness of consistency of intended and actual motor consequences (Farrer et al., 2008), where activity is higher when another agent is active (Decety et al., 2002; Farrer & Frith, 2002; Ruby & Decety, 2001). The TPJ

has been shown to be active in response to changes in visual and auditory stimuli that are task-relevant (Downar et al., 2001). Almost all clusters identified by the full dataset primary ALE comprised foci from both auditory and visual studies, suggesting a multisensory network. This asserts that some cortical areas process error in both auditory and visual self-generated feedback. The cluster centred in the left STG however was an exception, with only foci contributed from auditory studies.

Analyses of the auditory subset ALE revealed bilateral STG activation. These auditory cortical processing areas have shown a stronger response to auditory stimuli initiated by others than by oneself (Christoffels et al., 2007; Curio et al., 2000; Heinks-Maldonado et al., 2005; Houde et al., 2002; Knolle et al., 2012; Numminen & Curio, 1999). Increased activation of auditory cortex has also been correlated with the degree of delay in auditory feedback in speech production (Hashimoto & Sakai, 2003) and in response to sound masking compared to expected auditory feedback (Christoffels et al., 2007). Likewise, there was a large medial cluster in the auditory subset in the right SMA. The SMA has been suggested to play a role in auditory sensorimotor associations, for example, in using auditory information to elicit automatic motor responses (Lima et al., 2016) and auditory conditioning in a motor task (Kurata et al., 2000).

Studies that manipulated visual feedback were more likely to activate the premotor cortex of the right preCG, and a cluster in the EBA extending into the IPL. The EBA is involved in the visual processing of perceiving the human body (Downing et al., 2001; Peelen & Downing, 2007), of goal-directed action of body parts (Astafiev et al., 2004), and in processing incoherent human biological motion sequences (Downing et al., 2006). David et al. (2007) suggest that the EBA may be part of a larger network including posterior parietal cortex, premotor cortex, and the cerebellum, that is involved in correcting sensorimotor discrepancy. The IPL engages in the monitoring and comparison of one's own action and the visual feedback that it generates (Schnell et al., 2007), and in visuomotor incongruences (Balslev et al., 2006), reported as well more broadly in the parietal cortex (Fink et al., 1999; Shimada et al., 2005).

4.1.2 Cerebellum. Despite a strong consensus suggesting that forward models generally rely on the cerebellum (Bastian, 2006; Ishikawa et al., 2016 Ito, 2005; Kawato & Gomi, 1992; Wolpert et al., 1998), cerebellar activations were reported in only half of

the 38 experiments that formed the dataset for the current analyses. The majority of these activations were localized in lobules VI and VIII. Cerebellar lobule VI contains overlapping functional zones that are sensitive to auditory and visual stimulation while a functional zone of lobule VIII is associated with sensorimotor processing (O'Reilly et al., 2009; Sang et al., 2012). The cerebellum receives climbing fibre inputs from the sensory periphery and corticopontocerebellar mossy fibre inputs from cortical motor areas. The cerebellar cortex is arranged into functional modules which act as points of convergence of these inputs (Odeh et al., 2005). These modules in turn send cerebellothalamocortical projections back up to the corresponding cortical motor areas to inform further movements (Palesi et al., 2017). Through functional connectivity magnetic resonance imaging in humans, there have been efforts to accurately map the functional segmentation of the cerebellum to their corresponding cortical regions (Allen et al., 2005a; Habas et al., 2009; Krienen & Buckner, 2009; O'Reilly et al., 2009). For example, Buckner et al. (2011) performed functional connectivity analyses on data from 1,000 participants to provide a comprehensive view of all cerebellar connections with cortex. Their results provide strong evidence for two somatomotor homunculi in the anterior and posterior lobes. The anterior map is centred on lobule VI, while the posterior is centred on lobule VIII. The majority of cerebellar activations in studies included in our analyses were reported in lobules VI (Brand et al., 2017; Diedrichsen et al., 2005; Grafton et al., 2008; Graydon et al., 2005; Inoue et al., 2000; Pfordresher et al., 2014; Tunik et al., 2013; Yomogida et al., 2010; Zheng et al., 2013), and VIII (Anguera et al., 2010; Krakauer et al., 2004; Pfordresher et al., 2014; Tourville et al., 2008). This demonstrates that where cerebellar activations were observed they were not distributed randomly, making these findings unlikely to be false positives.

The majority of activations in the cerebellum reflecting fine-tuning motor control will be predominately in either of these two somatotopically organized lobules. However, in the ALE analyses, we do not see clustering at each of those regions. The reason for this may be due to the histological organization of the cerebellum. Its structure is completely uniform in its cortex, made up of repeating modules intermixed and overlapping with modules of separate function, having no integral borders (Ito, 1984b). This can lead to a lack of clear separation of focal activity for one specific function. For instance, electrical stimulation at different sites within the cerebellum can induce

contraction of the same muscles (Mottolese et al., 2013), while stimulating areas directly adjacent to responsive areas for movement of a body part would cause no movement at all. This creates a difficult picture to deconstruct, where representations in the cerebellum may be very spatially specific, where there may be multiple representations for movement of one body part, and if so where these representations may be sparsely distributed. For these reasons although many studies will report cerebellar activation in response to the same contrast, attempting to find meaningful clusters of functional localization common across studies may be limited.

4.2 Considerations for reliable probing of the forward model

Although all studies included in this meta-analysis contrasted experimental conditions of manipulated feedback with non-manipulated feedback, there was a considerable degree of diversity in methodology. Experiments varied in terms of feedback modality, the quality and quantity of the feedback manipulation, whether participants were able to adapt behaviour, and whether similar experimental trials were blocked together or intermixed.

4.2.1 Choice of feedback manipulation. There was considerable variability in the way that different studies implemented manipulations of sensory feedback from self-produced action. The most common feedback manipulations employed delays, a mismatch leading to an abrupt loss of control over feedback, noise masking, spectral shifting of auditory feedback or spatial shifting of visual feedback, and the physical application of an external force. The relative amount of cerebellar foci contributing to the study pool from different forms of manipulations differed accordingly. For example, shifted and feedback mismatch studies were twice as likely to elicit cerebellar activity than not (12/18 and 4/6), while temporal manipulations only elicited cerebellar activity in a quarter of the respective experiments (2/8). Among studies that implemented a continuous shift of feedback, six activations were in lobule VI (Brand et al., 2017; Diedrichsen et al., 2005; Grafton et al., 2008; Graydon et al., 2005; Inoue et al., 2000; Zheng et al., 2013), three in lobule VIII (Anguera et al., 2010; Krakauer et al., 2004; Tourville et al., 2008), with two foci just anterior to Lobule VI in the IV/V region (Anguera et al., 2010; Seidler et al., 2006), and two in Crus I/II (Krakauer et al., 2004). The same regions were reported for studies of mismatched feedback, with three foci incorporated

in Lobule VI (Pfordresher et al., 2014; Tunik et al., 2013; Yomogida et al., 2010), two in Crus II (Pfordresher et al., 2014; Schnell et al., 2007), and one in Lobule VIII (Pfordresher et al., 2014). However, there was no significant statistical contingency shown between type of manipulation and the report of cerebellar activity in our test of independence analyses.

4.2.2 Motor response or motor disturbance. Feedback manipulations can either elicit an adjustment to feedback or disrupt movement altogether. For instance, there is a tendency to speak louder when auditory feedback is masked (Lane & Tranel, 1971; Lombard, 1911) or to speak more quietly when auditory feedback is amplified (Chang-Yit et al., 1975). In both cases, feedback from the auditory periphery is at an unpredicted level and motor behavior is adapted accordingly. Likewise, by applying an external physical force to speech effectors (Abbs & Gracco, 1984; Gomi et al., 2002; Honda et al., 2002; Saltzman et al., 1998; Shaiman & Gracco, 2002), or shifting the frequency or pitch of feedback (Donath et al., 2002; Elman, 1981; Houde & Jordan, 1998, 2002; Jones & Munhall, 2000; Larson et al., 2000; Natke et al., 2003; Purcell & Munhall, 2006; Xu et al., 2004; Zarate & Zatorre, 2008; Zarate et al., 2010), automatic compensation responses are elicited as the speaker attempts to reach their intended auditory targets of their natural sounding speech. These responses of automatic compensation differ from adaptation as they are instantaneous shifts to counteract perturbation, while adaptation can be a conscious process of adjusting motor commands for future behaviour in response to continuous change of sensory feedback.

High magnitude manipulations may cause feedback to be perceived as entirely outside a range of control of the actor, and thus no longer triggering an automatic compensation in motor production. This stems from the theory that our sense of agency over sensory feedback from the environment is dependent on the magnitude of discrepancy between the predictable consequences of our own action and the unpredictable external influences of sensory input (David et al., 2008). For example, singers can successfully suppress the automatic compensation response when the pitch of their voice is shifted by a large amount, but not when a smaller shift is applied (Zarate & Zatorre, 2008). The duration of manipulated feedback also influences compensation responses. Pitch shifts with short durations prompted automatic

adjustments while pitch shifts with longer durations were more easily ignored (Burnett et al., 1998; Hain et al., 2000; Zarate & Zatorre, 2008; Zarate et al., 2010). Some forms of feedback can disrupt movement altogether. By applying delayed auditory feedback (DAF), speech and musical performance are interrupted (Black, 1951; Fukawa et al., 1988; Havlicek, 1968; Howell & Powell, 1987; Lee, 1950; Mackay, 1968; Siegel et al., 1982). The magnitude of delay is also an important consideration, with DAF of approximately 200ms being the most disruptive (Fairbanks & Guttman, 1958; Hashimoto & Sakai, 2003; Stuart et al., 2002). Further increase of delay may lead to similar disregard of feedback as irrelevant to the agency of the actor.

4.2.3 Adaptation to changes in feedback. Unpredicted feedback informs not only adjustments to ongoing movements, but also updates predictions for future movements by means of adaptation. This response to changes in environmental feedback is a form of motor learning and differs qualitatively from motor sequence learning (Doyon et al., 2003). This type of motor learning therefor must not be seen as planning new coordinated motor plans, and instead viewed specifically as reoptimization that seeks to minimize future costs to the motor system by forming more accurate predictions of existing motor plans (Izawa et al., 2008). Across the studies in our analyses, cerebellar foci were most common in studies driving adaptation in response to physical perturbation of the mouth (Golfinopoulos et al., 2011) and arm (Diedrichsen et al., 2005), learning new associations between the spatial consequences of movement when visual feedback is shifted (Anguera et al., 2010; Brand et al., 2017; Grafton et al., 2008; Graydon et al., 2005; Inoue et al., 2000; Krakauer et al., 2004; Seidler et al., 2006; Zheng et al., 2013), and when vocal pitch was shifted during continuous speech (Tourville et al., 2008; Zheng et al., 2013). All of these manipulations evoke the fine-tuning of accurately predicting movement outcomes in response to changes in the environment (Ishikawa et al., 2016). Indeed, adaptation was found to be the only factor in our analyses that showed a significant dependency with the elicitation of cerebellar activation.

The cerebellum plays an important role in adapting future predictions in light of error. Cerebellar patients are able to react to changes to feedback (Morton & Bastian, 2006; Smith et al., 2000), but are unable adapt by calibrating their predictions for subsequent

behaviour (Maschke et al., 2004; Morton & Bastian, 2006; Smith & Shadmehr, 2005). This suggests that adjustments to feedback, which inform subsequent fine-tuning, require cerebellar engagement. Monkeys with experimental lesions to areas of the cerebellum which receive mossy fibres from cortex such as the posterior lobe parafloculus and uvula are unable to adapt to changes in feedback (Baizer et al., 1999). Inactivation of deep cerebellar nuclei impairs adaptation to physical and visuomotor perturbation (Kerr, Miall & Stein, 1993). Cerebellar activity may change over time as the system moves from a state of adapting predictions that have failed, to executing predictions that have been adapted (Gilbert & Thach, 1977).

The effect of activation of the cerebellar cortex on adaptation has been reported as well in humans. Cerebellar excitation in transcranial direct current stimulation can lead to faster adaptation to visuomotor feedback transformation (Galea et al., 2010; Jayaram et al., 2012). Moreover, activity in the cerebellum is greatest immediately after conditions in the environment change (e.g., when feedback is first manipulated) but decreases over time (Friston et al., 1992; Nezafat et al., 2001). This has strong implications for the choice of design in experiments seeking to probe the cerebellum's involvement in the forward model.

4.3 The implications of fMRI

4.3.1 Neurovascular coupling in the cerebellum. The blood-oxygen-level-dependent (BOLD) response that is measured by fMRI may not be sensitive to some of the neural processes of sensory feedback error in the cerebellum. The BOLD response is correlated with local field potentials (LFP) rather than the spiking rate of neurons (Ekstrom, 2010), which has implications for fMRI studies of the cerebellum. Mossy fibre inputs to the cerebellum synapse thousands of Purkinje cells via parallel fibres and strongly influence LFP, leading to strong increases in the BOLD signal. Climbing fibres communicate via one-to-one inputs to Purkinje cells and are thus poorly coupled to the BOLD signal. Experiments that drive cerebellar activity via bottom-up climbing fibre error signals may be unsuited to measurement by fMRI, whereas experiments that drive cerebellar activity via mossy fibre inputs may lead to detectable BOLD responses (Diedrichsen et al., 2010). This is consistent with the view that the mossy fibre input system is more strongly associated with processes of motor learning

in adapting to sensorimotor prediction errors (Giovannucci et al., 2017; Ito, 2000; Thach, 1998).

4.3.2 Choice of experimental design. Our findings suggest that some experimental fMRI designs are more appropriately suited to elicit BOLD responses in the cerebellum. Designs that prevent participants from habituating to altered feedback, and continually cause them to adapt their motor responses, may be most effective in eliciting a detectable BOLD response. McGuire et al. (1996) illustrate habituation in block designs as they observed increased activation of the cerebellum during the first half of their study, but not in the latter half. This is consistent with the broader finding that the cerebellum may be more strongly engaged in adjusting to altered feedback than applying adjustments that have already been computed (Andersson & Armstrong, 1987; Flament et al., 1996; Horn et al., 2004; Imamizu et al., 2000; Moberget, et al., 2014). However, the arrangement instead of fast cycling between trials of different conditions in event-related designs may hinder adaptation responses if feedback is not consistent from trial to trial. Long events of consistent perturbation (e.g., Christoffels et al., 2007; Grafton et al., 2008; Limanowski et al., 2017) or similarly with short blocks (e.g., Inoue et al., 2000; McGuire et al., 1996; Seidler et al., 2006) may be best suited for probing processes related to the forward model associated BOLD response in the cerebellum.

5 Conclusions

We performed three ALE meta-analyses and one contrast analysis of functional neuroimaging studies that manipulated predicted self-initiated auditory, visual, and sensory feedback with the primary aim to identify cerebellar areas responsive to prediction error. No cerebellar clusters were produced as a result of these analyses. Contrary to common presumptions, we found that not all studies that used such approach show significant activation of the cerebellum, as well as variability in where in the cerebellum activations were reported. Our study suggests that this discrepancy stems from differential sensitivity and specific limitations of the experimental paradigms employed across MR neuroimaging altered sensory feedback experiments. These method- specific characteristics can restrict compatibility with other frameworks, which overwhelmingly support the involvement of the cerebellum in responding to errors in

predicted feedback as part of the forward model. We therefore assessed methodological variations that may determine the success of brain imaging experiments in evoking cerebellar activation. The results indicate that experimental designs which most reliably evoked cerebellar activation employed continuous feedback manipulations relevant for adapting motor plans for future action. Due to constraints of neurovascular coupling in cerebellar activity, it is possible that only mossy fibre inputs in response to adaptation elicit demonstrable BOLD signals, while error signals conveyed via climbing fibre spike firing increase may not suitable for fMRI testing. The results further suggest that short-blocked designs may offer the most effective approach, engaging a period of adaptation to changes in feedback without reaching a state of habituation, leading to reliable activation of the cerebellum.

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***Conflict of interests*.** The authors state no conflict of interest.

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

Supplementary information

SUP TABLE 1 | CONTRAST AND CONJUNCTION RESULTS					
Brain regions	***BA***	***MNI coordinates*** *(x, y, z)*			***Cluster size*** *(mm^3)*
A. Auditory Feedback Error > Visual Feedback Error					
L STG (A1)	41	-59.9	-28.2	10.7	4,456
R STG (A1)	41	53.9	-16.3	4.0	2,528
B. Auditory Feedback Error > Visual Feedback Error					
-	-	-	-	-	-
C. Auditory Feedback Error ∩ Visual Feedback Error					
-	-	-	-	-	-

SUPPLEMENTARY TABLE 1. *Contrast and conjunction analyses using ALE clusters reported (see Table 2) (cluster-level inference threshold correction (P<0.05) with a cluster-forming threshold of uncorrected p<0.001 using 1,000 permutations). Contrast thresholding with False Discovery Rate (pID) 0.01. Abbreviations: BA, Brodmann area; L, left; R, right; STG, superior temporal gyrus; A1, primary auditory cortex*

Chapter 3

Hypersensitivity to passive voice hearing in hallucination proneness

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Abstract. Voices are a complex and rich acoustic signal processed in an extensive cortical brain network. Specialized regions within this network support voice perception and production and may be differentially affected in pathological voice processing. For example, the experience of hallucinating voices has been linked to hyperactivity in temporal and extra-temporal voice areas, possibly extending into regions associated with vocalization. Predominant self-monitoring hypotheses ascribe a primary role of voice production regions to auditory verbal hallucinations (AVH). Alternative postulations view a generalized perceptual salience bias as causal to AVH. These theories are not mutually exclusive as both ascribe the emergence and phenomenology of AVH to unbalanced top-down and bottom-up signal processing. The focus of the current study was to investigate the neurocognitive mechanisms underlying predisposition brain states for emergent hallucinations, detached from the effects of inner speech. Using the temporal voice area (TVA) localizer task, we explored putative hypersalient responses to passively presented sounds in relation to hallucination proneness (HP). Furthermore, to avoid confounds commonly found in in clinical samples, we employed the Launay-Slade Hallucination Scale (LSHS) for the quantification of HP levels in healthy people across an experiential continuum spanning the general population. We report increased activation in the right posterior superior temporal gyrus (pSTG) during the perception of voice features that positively correlates with increased HP scores. In line with prior results, we propose that this right-lateralized pSTG activation might indicate early hypersensitivity to acoustic features coding speaker identity that extends beyond own voice production to perception in healthy participants prone to experience AVH.

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1 Introduction

The human voice is a complex signal that carries rich information. This allows the listener not only to identify linguistic messages but also who speaks and how something is said (Belin et al., 2004; Lavan et al., 2019). Some individuals experience auditory verbal hallucinations (AVH), in which they perceive voices in the absence of a corresponding incoming voice signal (Anthony, 2004; Bentall, 1990; Brookwell et al., 2013). Experience of AVH is a key symptom of schizophrenia (Bauer et al., 2011; Hugdahl & Sommer, 2018; Larøi et al. 2012). Yet, it is also reported in multiple other psychiatric, developmental, and neurological disorders (Reininghaus et al., 2016; Rollins et al., 2019; van Os et al., 2000; Waters & Fernyhough, 2017; Zhuo et al., 2019) and in a minority of otherwise healthy people (Beavan et al., 2011; Linscott & Van Os, 2013; McGrath et al., 2015). Variability in AVH phenomenology exists within and across brain disorders (Stephane et al., 2003; Jones, 2010) and between clinical and non-clinical voice hearers (Baumeister et al., 2017; Daalman et al., 2011; Larøi, 2012; Johns et al., 2014). However, hallucinated voices commonly carry information regarding the identity or emotion of a perceived speaker (Badcock & Chhabra, 2013; Larøi & Woodward, 2007; McCarthy-Jones et al., 2014; Stephane et al., 2003), therefore involving a wide range of cortical areas in a voice perception network.

Multiple cognitive theories have been proposed delineating the emergence and phenomenology of AVH (Ćurčić-Blake et al., 2017a; Jones, 2010; Rollins et al., 2019). One long standing model considers hallucinations as the misattribution of self-generated input to an outside source (Feinberg, 1978). In terms of AVH, signals from voice production cortical regions during inner speech are misperceived as hearing someone else speak (Allen et al., 2007a; Gregory, 2016; Jones & Fernyhough, 2007a,b; Swiney & Sousa, 2014). Recently, competing theories have gained traction, claiming that the initiation of hallucinations does not require motor activity while they are, at their core, misperceived sensations from the environment (e.g., Ford & Mathalon, 2019; Thakkar et al., 2021).

The selection and processing of sensory inputs from the environment relevant to learning, adaptation, or behavioural responses involves multiple regions and distributed networks across the brain. The role of salience attribution within this

integrated system provides the necessary trigger to shift processing from a state of rest to active sensation and perception (Menon & Uddin, 2010; Menon, 2011; Palaniyappan & Liddle, 2012; Uddin, 2015). According to this framework, increased auditory cortex activation associated with AVH can be ascribed to a bottom-up hypersensitivity, or salience bias, towards irrelevant sounds. The modulation and over-weighting of top-down predictions may influence this salience bias as well as guide the system to perceive what it expects in meaningless unimodal and multimodal stimuli (Deneve & Jardri, 2016; Fletcher & Frith, 2009; Friston, 2005a, 2012b; Jardri et al., 2016; Leptourgos et al., 2017). Since voice signals in humans are inherently salient to human listeners, they may be particularly implicated in hypersensitive responses leading to false perceptions. Furthermore, for those who experience AVH, the engagement of brain regions controlling inner speech signals, memory retrieval, and emotion may then guide the phenomenology of the perceived speech in terms of content and speaker-related features (Waters et al., 2012a). Abnormal salience processing has been strongly linked to positive symptoms of schizophrenia (Miyata, 2019).

Researching the contribution of these mechanisms to AVH in non-clinical samples may be particularly useful as it avoids potential confounds seen in clinical populations such as medication, age of onset, and duration of symptoms that may affect brain structure and function (Kelleher et al., 2010; Kelleher & Cannon, 2011; Verdoux & Van Os, 2002). This perspective is in line with the experiential continuum of psychosis (Beavan et al., 2011; de Leede-Smith & Barkus, 2013; Johns, 2005; Johns et al., 2014; Larøi et al., 2012; Zhuo et al., 2019), whereby functional variability in the mechanisms serving perception across the population account for the spectrum of normal experience, vivid perceptions and imagery, sub-clinical forms of hallucinations, and those seen in full-blown psychosis. The revised Launay Slade Hallucination Scale (LSHS) is as a measure of perceptual experience and beliefs associated with vivid daydreams, thoughts, imagery, and those related to false perceptions such as visual and auditory hallucinations (Larøi & Van der Linden, 2005a). The LSHS provides a measure of hallucination proneness (HP), where higher scores signify increasing abnormality in perceptual experience and beliefs, including true hallucinations. Although individual items from the LSHS can be used to identify the prevalence of AVH (e.g., Kompus et al., 2015), HP itself is not a measure of risk for psychosis.

Two critical factors have been incorporated into the formulation of our hypotheses. First, differential brain activity may indicate abnormal voice processing as a predisposition for false perceptions, i.e., activation patterns similar to those during hallucinations. Second, the localization of reported changes in brain responses may indicate a specific stage within hierarchical voice processing at which this predisposition manifests. To date, no consensus has been empirically established regarding a trait-based association between hallucinations and brain responses to the voice. For example, when presented with voices, patients who commonly experience hallucinations display decreases (Copolov et al., 2003), increases (Escarti et al., 2010; Martí-Bonmatí et al., 2007; Parellada et al., 2008), or no activation differences in voice selective temporal regions (Simons et al., 2010; Woodruff et al., 1997). Such inconsistency is likely due to methodological heterogeneity (Bohlken et al., 2017). For example, these studies differed in terms of stimulus type, stimulus content, and the inclusion of a non-hallucinating patient control group. Moreover, patients with chronic hallucinations can experience spontaneous AVH during scanning (Jardri et al., 2011; Kühn & Gallinat, 2012; van Lutterveld et al., 2013; Zmigrod et al., 2016), which may even be unintentionally elicited by tasks (e.g., Copolov et al., 2003; Parellada et al., 2008). Although this hallucinatory state elicits brain activity in voice perception regions, simultaneous external voice input during AVH results in a paradoxical net activity decrease (Hugdahl & Sommer, 2018; Kompus et al., 2011).

The localization of changes in functional brain activity within the voice processing network can be particularly informative in determining how hallucination proneness may arise. Within the upper bank and lateral regions of the temporal lobe, voice signals are processed hierarchically along a pathway composed of multiple functional subsystems or components (Belin et al., 2004; Pernet et al., 2015; Zhang et al., 2021). The engagement of these temporal voice areas (TVA) starts with the evaluation of low-level acoustic features in the posterior superior temporal gyrus (STG), an area specialized in processing spectro-temporal properties of complex sounds (Griffiths & Warren, 2004; Warren et al., 2005a,b). Further processing occurs along hemispherically specialized pathways, with linguistic features predominantly in the left and paralinguistic (i.e., speaker-related information) in the right side of the brain (Belin et al., 2000; Formisano et al., 2008). However, some stimuli such as emotional

vocalizations contain both speaker-and speech-relevant information and involve bilateral processing of separate features in the signal (Schirmer & Kotz, 2006). Importantly, AVH often contain marked paralinguistic information about speaker identity or emotion (Larøi & Woodward, 2007; Larøi et al., 2012; McCarthy-Jones et al., 2014). In non-clinical voice hearers, however, the degree of perceived emotional valence is less prominent (Daalman et al., 2012; de Boer et al., 2016). Speaker-related feature processing operates along a multi-stage hierarchy in the right temporal cortex along a posterior to anterior gradient (Belin & Zatorre, 2003, Nakamura et al., 2001; von Kriegstein et al., 2003, von Kriegstein and Giraud, 2004). The TVA localizer is a widely used fMRI task which reliably identifies activation peaks localized in the bilateral anterior, middle, and posterior superior temporal cortex (Pernet et al., 2015). By comparing voice to non-voice activation in response to passively heard sounds, regions of interest (ROI) can be defined for further investigation. Using ROIs produced by this task, we predicted HP-related early sensitivity to low-level voice features to be isolated to the posterior STG ROI. Alternatively, changes to voice processing in the anterior direction of the right STG might indicate an abnormal salience bias for identity or emotion associated with an increasing propensity to hallucinate.

2 Methods

2.1 Participants

Twenty-six participants took part in this study, recruited through the SONA system and social media channels at Maastricht University, the Netherlands. Participants were provided with informed consent and offered university study credit for compensation. Exclusion criteria included any history of psychotic disorder, neurological impairment, history of drug dependence or abuse, and traumatic brain injury. Participants were screened for MRI safety and reported no metal implants, claustrophobia, or pregnancy. Furthermore, all participants reported no known hearing deficits. Robust statistics using the interquartile range rule for participant age revealed one outlier (Rousseeuw & Hubert, 2011), leading to the exclusion of the dataset from further analysis. Of the resulting 25 individuals (17 female), the average age was 20.92 years (*SD* = 3.95; range 18 to 32). The Ethical Review Committee of the Faculty of Psychology and Neuroscience at Maastricht University (ERCPN-176_08_02_2017) approved this study.

2.2 Hallucination proneness

The revised LSHS was employed as a self-report measure of HP (Larøi & Van der Linden, 2005a). The questionnaire consists of 16 items targeting tactile, sleep-related, visual, and auditory modalities of psychosis-like experience as well as vivid thoughts and daydreaming. Responses were given using a five-point Likert scale, measuring the extent to which each statement applied to them. The sum of all responses equated to an overall HP measure. Furthermore, to investigate the exclusivity of auditory-only items, subscores of three items were summed to produce a composite score (Larøi et al., 2004; Larøi & Van der Linden, 2005a).

2.3 Voice area fMRI-localizer task

Voice selective cortical brain regions were identified using a standard fMRI-localizer task (Belin et al., 2000). This widely used tool reliably probes activity across three bilateral peaks in the superior temporal gyrus (e.g., Pernet et al., 2015), often designated as anterior, middle, and posterior temporal voice areas (TVA). Furthermore, many studies applying this task have reported extra-temporal voice regions, such as the inferior frontal cortex (IFC). The voice area localizer consists of 20 vocal (V) and 20 non-vocal (NV) trials. Additionally, 20 silence (S) trials are included allowing relaxation of the hemodynamic response to auditory stimuli. The voice condition is composed of human speech (words, syllables, or sentence excerpts) and non-speech voices produced by male and female speakers of different ages (7 babies, 12 adults, 23 children, and 5 elderly). This broad selection of voice stimuli allows for the probing and inclusion of functionally diverse regions of TVA. Conversely, the non-voice condition includes environmental (natural and animal) and man-made (e.g., cars, alarm clocks, instrumental music) sounds. Sound clips are presented at a standard 70db volume (for a detailed report of the included sounds and recording duration, amplitude, and frequency see Pernet et al., 2015). Trials were presented in a pseudorandom order, each with a duration of eight seconds. With a two second inter-trial interval, the total run time of the task was 10 minutes.

2.4 FMRI data acquisition

Scanning was conducted using a Siemens 3T Magnetom Prisma Fit equipped with a 32-channel head coil (Siemens Healthcare, Erlangen, Germany), at the Scannexus

facilities (Maastricht, the Netherlands). Structural whole-brain T1-weighted images were acquired with a single-shot echoplanar imaging (EPI) sequence (field of view (FOV) 256 mm; 192 axial slices; 1mm slice thickness; 1 × 1 × 1mm voxel size; repetition time (TR) of 2250 ms; echo-time (TE) 2.21 ms). For the functional localizer task, T2-weighted EPI scans were collected (FOV 208mm; 60 axial slices; 2mm slice thickness; 2 × 2 × 2mm voxel size; TE 30 ms; flip angle = 77°). To reduce scanner noise interference, auditory stimuli were presented via S14 MR-compatible earphones, fitted with foam earplugs (Sensimetrics Corporation). Furthermore, to provide relative silence during playback of auditory stimuli, a long inter-acquisition-interval was adopted where time between consecutive acquisition was delayed, resulting in a TR of 10 seconds. The delayed TR was timed to allow a 2000ms acquisition period during peak activation in the auditory cortex (Belin et al., 1999; Hall et al., 1999).

2.5 Data pre-processing and analysis

Pre-processing of the TVA localizer blood-oxygen-level-dependent (BOLD) signal was conducted in SPM12 (Wellcome Department of Cognitive Neurology, London, UK). A standard pipeline was applied using slice timing correction, realignment and unwarping, segmentation, normalization to standard (MNI) space (Fonov et al., 2009), and 8mm isotropic Gaussian kernel full width at half maximum (FWHM) smoothing. Analysis followed a two-level procedure in which contrast estimates were first determined as fixed effects at the level of individual participants then modelled as random effects at the level of the sample. Contrast estimates were computed on BOLD data to assess voice sensitivity (V > NV) and sensitivity to environmental sounds (NV > S) for each participant. A first-level fixed-effects GLM analysis for the conjunction analysis ((V > NV) ∩ (V > S)) was computed to localize the temporal voice areas. A second-level random-effects analysis tested for group-level significance and determined the ROIs for parameter extraction. Contrast estimates of V >S and NV > S were then used to contrast voice with non-voice activity, corrected for baseline, in the subsequent hypothesis-driven ROI analysis to investigate the correlation of voice-preferential TVA activity compared to HP. Contrast estimates were extracted from a 5mm radius of the centre coordinates from each region of peak activity produced in the TVA-localizer using the SPM MARSbar toolbox (Brett et al., 2002). Pearson's

correlation analysis using bootstrapping (5000 samples) and bias-corrected confidence intervals was then employed to test for significant relationships between the sensitivity of the voice ROIs and HP measures.

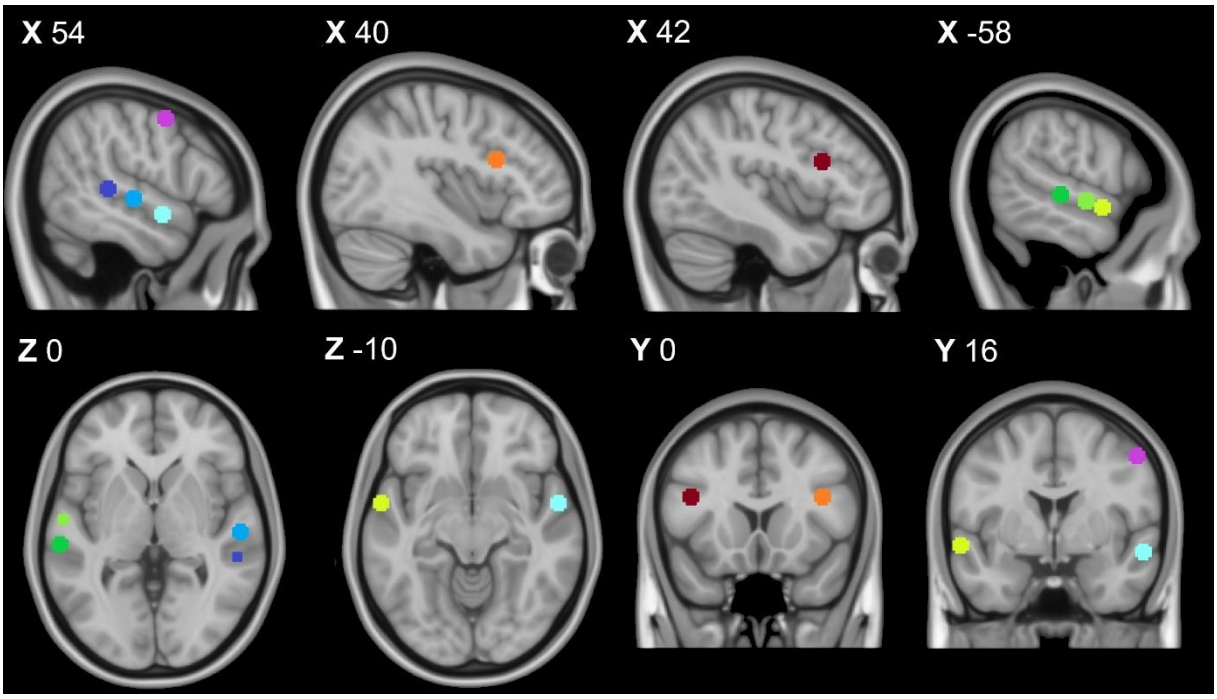

FIGURE 1. *Temporal voice area fMRI localizer task results: Purple = right premotor cortex, dark blue = right posterior temporal gyrus, middle blue = right middle temporal gyrus, light blue = right anterior temporal gyrus, orange = right inferior frontal cortex, dark green = left posterior superior temporal gyrus, middle green = left middle superior temporal gyrus, light green = left anterior superior temporal gyrus, red = left inferior temporal cortex. All coordinates listed in MNI space (x,y,z).*

3 Results

3.1 Hallucination proneness

For the HP composite score (possible maximum score of 80), the mean self-reported rating was 25.20 (*SD* = 10.47; range 0 to 42). The HP auditory subscale mean score (possible maximum score 15) was 3.92 (S*D* = 2.74; range 0 to 11). To test for normality of the distribution of demographics and hallucination proneness across the sample, Shapiro-Wilk tests were conducted. Both total LSHS (0.948, df = 25, *p* = 0.229) and auditory subscale (0.928, df = 25, *p* = 0.078) were not different from normal. A moderately strong correlation was also found between LSHS auditory subscale and non-auditory item totals (*r* = 0.457, df = 25, *p* = 0.019).

3.2 Voice area localizer

The fMRI localizer task produced 5 clusters covering bilateral lateral temporal cortices, bilateral inferior frontal gyri, and the right precentral gyrus (preCG) [Table 1, Figure 1]. Within each bilateral temporal cortex 'voice patch', peak activity localizations were distinguished in three distinct regions: posterior (pSTG), middle (mSTG), and anterior STG (aSTG). These regions correspond to the expected divisions of the TVA localizer (Pernet et al., 2015).

TABLE 1 | TVA LOCALIZER RESULTS

#	Hem.	Label	BA	MNI coordinates (x, y, z)			Cluster-level p-FDR	Peak-level p-FDR	Size (voxels)
		mSTG	22	-58	-10	-4		1.46e-09	
1	L	pSTG	22	-60	-26	0	1.68e17	1.46e-09	4145
		aSTG	22	-58	0	-8		1.36e-08	
		mSTG	22	56	-18	-2		1.46e-09	
2	R	aSTG	22	56	0	-12	2.07e-17	1.60e-08	4010
		pSTG	22	54	-34	4		1.60e-08	
3	R	pMC	5	52	2	48	0.005	4.15e-05	285
4	L	IFC	44	-42	16	22	0.038	0.002	142
5	R	IFC	44	40	16	22	0.023	0.030	180

TABLE 1. *Results from Temporal Voice Area fMRI localizer task. Hem = hemisphere, (a/m/p) STG = (anterior/middle/posterior) superior temporal gyrus, pMC = premotor cortex, IFC = inferior frontal cortex, BA = Brodmann's Area, p-FDR = false discovery rate corrected p-value (threshold = 0.05). All coordinates listed in MNI space (x,y,z).*

3.3 FMRI correlation

Correlational tests were performed between contrast estimates representing voice preference ([V > S] > [NV > S]) observed in each TVA-ROI with both the composite HP score and the auditory subscore of the LSHS. All thresholds for significance were Bonferroni-adjusted for multiple comparisons using ($p < 0.025$). Only the right pSTG reached statistical significance ($r = 0.470$, df = 25, $p = 0.020$) [Table 2, Figure 2]. Post-hoc correlation analyses were run to assess the relative contributions of both voice (V > S) and non-voice (NV > S) contrasts to correlational analyses (see detailed results in supplementary information). We conducted these analyses in order to rule out a general hypersensitivity of temporal cortex activity nonspecific to the conditions of interest probed by the conjunction analysis. No significant correlations with HP were found in any ROI for voice (V > S), however, a significant negative correlation was reported in the right IFC for non-voice (V > S) sensitive activity ($r = -0.614$, df = 25, $p = 0.001$).

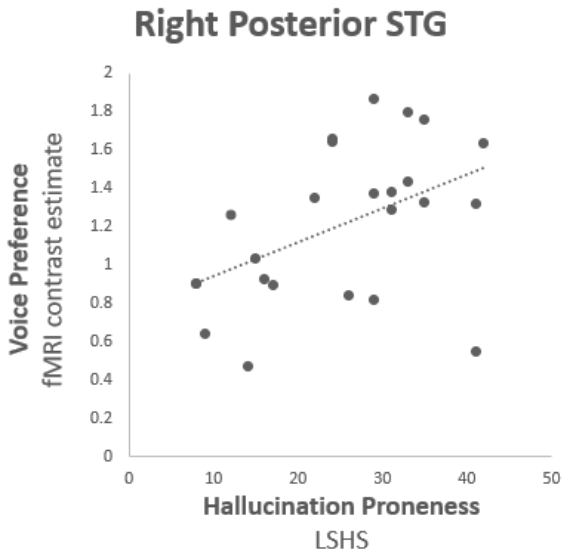

FIGURE 2*. Hallucination proneness fMRI correlation analysis results: Right posterior superior temporal gyrus (BA 22; MNI 54, -34, 4), Voice preference = contrast estimate ([Voice>Silence]>[Non-voice>Silence]), LSHS = Launay Slade Hallucination Proneness scale. Correlation coefficient r 0.470, df = 25, p = 0.020.*

TABLE 2 | VOICE PREFERENCE RESPONSE CORRELATION								
		ROI			***LSHS***		***LSHS-Auditory***	
Hem.	***Label***	***μ***	***SD***	***CI (95%)***	***r***	***p***	***r***	***P***
	mSTG	1.189	0.479	0.203-0.434	0.120	0.576	0.178	0.406
L	pSTG	1.505	0.586	0.997-1.380	-0.237	0.267	-0.024	0.915
	aSTG	1.511	0.560	1.271-1.740	-0.058	0.791	0.055	0.797
	mSTG	1.019	0.452	0.838-1.200	0.266	0.208	0.165	0.440
R	aSTG	1.295	0.515	1.089-1.501	-0.177	0.408	-0.033	0.882
	pSTG	1.213	0.406	1.051-1.375	0.470	*0.020	0.276	0.192
R	pMC	0.625	0.447	0.446-0.804	0.087	0.685	-0.103	0.635
L	IFC	0.319	0.288	0.204-0.434	-0.048	0.827	-0.025	0.911
R	IFC	0.293	0.323	0.164-0.422	0.231	0.277	0.134	0.534

TABLE 2. *Voice preference response ([Voice>Silence]>[Non-voice>Silence]) correlation with hallucination proneness: ROI = region of interest, (a/m/p) STG = (anterior /middle /posterior) superior temporal gyrus, pMC = premotor cortex, IFC = inferior frontal cortex, μ = mean activation from contrast, SD = standard deviation, LSHS = Launay-Slade Hallucination Proneness scale, LSHS-Auditory = subset of 3 auditory items, r = correlation coefficient, Bonferroni-corrected significance level (*p < 0.025)*

4 Discussion

The current study investigated whether a measure of abnormal perceptual experience (HP) in a non-clinical sample is associated with variability in the functional brain responses of the temporal cortex regions serving detecting and processing of voice signals. Considering the well-established roles of specific voice sensitive regions of the

cerebral cortex, we aimed to determine if this putative relationship would be limited to specific subprocesses in hierarchical voice perception. As hypothesized, activity for voice versus non-voice processing correlated positively with HP only in the pSTG, a region associated with the early processing of low-level acoustic features in complex auditory signals (i.e., Griffiths & Warren, 2004; Warren et al., 2005a,b). Furthermore, this finding was restricted to the right hemisphere and therefore is likely linked to the processing of paralinguistic voice information (Belin et al., 2000; Formisano et al., 2008). Additionally, post-hoc analysis revealed a negative correlation with HP in the right IFC for non-voice versus silence. Together, these findings may confirm that as the propensity to hallucinate increases, right posterior temporal lobe voice hypersensitivity increases and is accompanied by a decreased prefrontal response to non-vocal environmental sounds.

4.1 Hallucination proneness and hypersensitvity

Multiple neurocognitive mechanisms underlying hallucinations have been proposed. Most commonly, these theories have focused on describing the emergence and phenomenology of pathological voice hearing in patients with psychotic disorders such as schizophrenia (Allen et al., 2008; Ćurčić-Blake et al., 2017a; Hugdahl, 2015). The most influential models describe atypical increases in brain activity in cortical voice regions. The current investigation was approached from the perspective of perceptual salience models claiming a central role of hypersensitivity to irrelevant sensory stimuli in auditory regions (Menon & Uddin, 2010; Menon, 2011; Palaniyappan & Liddle, 2012; Uddin, 2014). Conversely, prominent self-monitoring models of hallucinatory experience describe increased activity as the result of insufficient suppression of sensory cortices during inner speech (Allen et al., 2007a, 2008; Frith & Done, 1988; Jones & Fernyhough, 2007b; Tracy & Shergill, 2006; Weiss & Heckers, 1999). According to this theory, the activation of speech production regions is required for the emergence of AVH. However, the current results demonstrate that variability in voice processing cortical regions in relation to HP exists without motor activity.

It is possible that theories proposing divergent involvement of speech production and perception mechanisms in AVH may be not mutually exclusive. Experiences of people who hallucinate are diverse. As theories of hallucination proneness become more

specific and concrete, they may become less well aligned with the phenomenology of the hallucinator. Therefore, hallucinatory experience might be best characterized by multiple subtypes, to which specific theories might apply better than others (Jones, 2010). For example, models describing the phenomenology of voice hearing ascribe the top-down contribution of intrusive memories and thoughts to the quality of false perception experiences (Bohlken et al., 2017; Ćurčić-Blake et al., 2017a; Hugdahl, 2015; Upthegrove et al., 2016). A core abnormality in brain function central to the emergence of false perceptions likely rests in the interactive process of top-down predictions and bottom-up sensory input (Allen et al., 2008; Hugdahl, 2009; 2015; Kowalski et al., 2021). Regarding perceptual salience, bottom-up hypersensitivity to sensory input is congruent with established computation neuroscience accounts of predictive coding in false perceptions (Sterzer et al., 2018). Here, weighted top-down predictions and bottom-up explanations of sensation interact along a hierarchical network, constantly updating via Bayesian inference to form the most reliable percept (Feldman & Friston, 2010; Fletcher & Frith, 2009; Friston, 2005a, 2012b; Hohwy, 2017). When internal prediction signals are weighted too strongly, one 'senses what they expect'. Moreover, when the top-down input is too strong, the threshold for active perception may be reached under minimal sensory input. However, the self-monitoring theory posits a delayed or absent prediction signal resulting in increased activation of sensory cortical regions and is therefore in apparent conflict with the former account (Corlett et al., 2019; Leptourgos & Corlett, 2020). These expectations could operate on separate time scales, at different levels of the information processing hierarchy, or simply serve two different functions in hallucinations (Thakkar et al., 2021).

The role of perceptual salience in a multistage process leading to false perceptions has gathered substantial support in functional neuroimaging. Namely, research into large-scale functional brain networks has provided a resting-state hypothesis, outlining brain states serving as a predisposition for hallucinations, including voice hearing (Northoff & Qin, 2011; Northoff, 2014). While at rest, activation of the salience network, under conditions of irrelevant stimuli, may interrupt the Default Mode Network (DMN) and engage active sensory processing (Alderson-Day et al., 2015, 2016; Schmidt et al., 2015). The salience network therefore operates as a switch between the DMN and central executive network and how attention is directed towards incoming sensations,

constituting a triple network model (TMN) subserving the advent of hallucinatory experience (Menon, 2011). Although we did not acquire behavioural data from the participants with ratings of perceived salience while listening to stimuli during scanning, we suggest that the change in brain activity that we observed in the right pSTG is indicative of the TMN in response to voice stimuli.

4.2 Hierarchical voice network processing

Voices are processed along a series of bilateral voice patches in the posterior, middle, and anterior STG. These temporal voice areas are reliably identified by a standardized TVA localizer task (Pernet et al., 2015). Participants with greater HP displayed increased right pSTG activation in response to vocal stimuli. Activity in this region may reflect sensitivity to low-level acoustic features during early stages of voice processing (Griffiths & Warren, 2004; Warren et al., 2005a,b). Furthermore, the pSTG is not specialized for voice processing per se, and likely plays a broader role in extracting spectro-temporal acoustic features from complex sounds, of which voices are an example. However, activation in these regions preferentially responds to salient stimuli, such as voices, over and above other similarly complex environmental sounds (Pernet et al., 2007).

In terms of the salience hypothesis for hallucinatory experience, the assignment of salience to irrelevant, neutral, events must be considered in terms of the paralinguistic factors which may be involved. Indeed, the phenomenology of AVH is often marked by prominent paralinguistic features in the identity and emotional valence of the hallucinated speaker (Larøi & Woodward, 2007; Badcock & Chhabra, 2013; McCarthy-Jones et al., 2014; Stephane et al., 2003). Individuals who experience hallucinations often express difficulty in discerning the identity of veridical voices. For example, in schizophrenia patients who experience hallucinations, there is a bias to externalize voices to another person (Allen et al., 2007b; Johns et al., 2001; Mechelli et al., 2007; Pinheiro et al., 2016, 2017). Likewise, severity of AVH in patients is increasingly altered by emotional processing (Alba-Ferrara et al., 2013; Rossell & Boundy, 2005; Shea et al., 2007; Tseng et al., 2013). The role of salience may be influential in perceptions of speaker identity, as misattributions are more prevalent for emotional stimuli (Costafreda et al., 2008; Ditman & Kuperberg, 2005; Pinheiro et al., 2016, 2017). However, the

effects of emotional valence in perceiving voice identity for people prone to false perceptions of voices has not shown clear consensus (i.e., Brookwell et al., 2013). Comparisons of AVH severity in patients with schizophrenia with judgements of speaker identity have indicated an increasing proneness to externalize voices with negative content (Allen et al., 2004; Pinheiro et al., 2016). In non-clinical groups, the involvement of salient emotional features in voices is less clear. For example, higher levels of hallucination proneness in the general population are not associated with atypical evaluation of emotional valence in words or vocalizations (Pinheiro et al., 2019). However, it has been indicated that non-clinical individuals prone to voice hearing require stronger emotional information to consider a stimulus as emotional (Amorim at al., 2021) or may allocate similar attention to voices irrespective of their emotional salience (Castiajo & Pinheiro, 2021). Future research is required into how variability in perceived salience of speaker-related features may affect processing in the hierarchical voice network and, in particular, how posterior STG activity related to HP may be influenced.

In addition to the TVA findings, the localizer task often provides a subset of extra-temporal regions indicating an extended voice processing network (Pernet et al., 2015). In our sample, extra-temporal peak activations were ascribed to bilateral inferior frontal and right hemisphere premotor cortex. Prefrontal involvement of the left IFC is commonly found in voice perception, with different subregions serving various functions. For example, the pars orbitalis is involved in processing semantic and emotional information (Belyk et al., 2017). Here, the left IFC peak was found in Broca's area, which has been theorized to represent mirror neuron activity which may be useful in guiding conversational turn-taking (Grafton & Hamilton, 2007; Kilner et al., 2007; Rizzolatti & Craighero, 2004). Likewise, precentral motor regions are involved in the perception and production of speech (Cheung et al., 2016; Pulvermüller et al., 2006; Wilson et al., 2004). This could explain speech production region activity sometimes reported during AVH (Jardri et al., 2011; Kühn & Gallinat, 2012; Zmigrod et al., 2016). However, self-monitoring theories take this as evidence for top-down inner speech signals guiding the perceived hallucinatory voice. Notably, transcranial direct-current stimulation targeting a fronto-parietal sensorimotor network is an effective treatment for the alleviation of AVH in patients with schizophrenia (Yang et al., 2019). In our post-

hoc analysis, the right IFC ROI shows an intriguing negative correlation to HP, however, only for non-voice sounds. The right IFC may serve a role in salience processing, for example in recognizing salient cues in voice signals (Bestelmeyer et al., 2012; Johnstone et al., 2006; Johnson et al., 2021; Jones et al., 2015). Additionally, this area shares a high functional integration with temporal regions serving voice perception and may assist successful voice recognition (Aglieri et al., 2018). Although this finding is difficult to interpret on its own, it may indicate a decrease in salience attribution for environmental sounds during a voice perception task. This may indicate not only an HP-related salience bias affecting the sensitivity of cortical responses to voice sounds, but also a general bias away from non-voice sounds between hypersalient responses to intermittent voice stimuli.

4.3 Limitations and recommendations

We identify a number of limitations within the current study and provide suggestions for future research. First, although the use of the established TVA localizer task facilitated the testing of our hypotheses regarding an early hypersensitivity to voice sounds, it did not preclude further investigation into how more complex stages of the voice processing hierarchy may relate to HP. Specifically, BOLD responses from this task are averaged across the trials containing different types of voice stimuli. This implies that signals extracted from ROIs serving different functional roles in voice processing, e.g., emotion or identity, do not represent the processing of specific features, but rather constitute a generalized voice detection signal. Second, in this study, behavioral measures of perceived stimulus salience were not collected. Therefore, interpretations of a salience bias attributed to increased functional brain responses cannot be directly linked to the subjective perception of the participants. Third, participants in the current study were sampled from a relatively homogenous sample of university students, similar in age, ethnicity, and cultural backgrounds. Due to the uneven distribution of environmental risk factors for psychotic symptoms throughout the population (Baumeister et al., 2017; DeRosse & Karlsgodt, 2015; Johns & van Os, 2001), our sample may unintentionally capture a set of protective factors. To address these limitations in future studies, we suggest a two-step procedure using a novel task that systematically varies paralinguistic voice features. This may allow

investigations into how hierarchical processing downstream of initial HP-related hypersensitivity may influence responses to the perceived emotion or identity of the speaker. Furthermore, behavioural appraisals of perceived salience may be included to compare fMRI response patterns and HP scores. Finally, subsequent research may benefit from an increased sample size and diversity, including a structured collection of additional demographic data and associated environmental risk factors as possible covariates for HP-related brain changes.

5 Conclusion

We observed that hallucination proneness is positively correlated with increased activation in the right pSTG in response to passively heard voices. This suggests a hypersensitivity associated with a propensity to hallucinate in a region of the brain which extracts low-level acoustic features from complex auditory signals. The right pSTG comprises the early processing of voice signals along the paralinguistic information pathway of the cortical voice processing network. We propose that this increases activity in response to voices represents a perceptual salience bias as a precursor for the emergence of hallucinations. This interpretation is in line with functional network models that posit abnormal engagement of a salience network during irrelevant stimulus exposure as the underlying neurocognitive mechanism of false perceptions. Furthermore, the current findings conflict with self-monitoring accounts of inner speech models that propose a critical role of voice production regions in the inception of auditory verbal hallucinations. We have demonstrated that HP is associated with right pSTG activation driven by external auditory signals. Although we do not reject self-monitoring accounts, we suggest that a state of cortical hypersensitivity to irrelevant sensory input may be the first step in the emergence of a hallucinatory experience, possibly followed by the influence of top-down signals such as inner speech, memory, and thought that together contribute to the phenomenology of AVH.

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

Supplementary information

SUP TABLE 1 | VOICE SENSITIVE RESPONSE CORRELATION							
		ROI				LSHS	
Hem.	Label	μ	Min	Max	SD	r	p
	aSTG	1.189	0.398	2.288	0.479	-0.255	0.229
L	mSTG	1.505	0.470	2.650	0.586	-0.210	0.327
	pSTG	1.511	0.250	2.600	0.560	-0.096	0.655
	aSTG	1.019	0.180	1.720	0.452	0.026	0.903
R	mSTG	1.295	0.440	2.500	0.515	-0.128	0.551
	pSTG	1.213	0.471	1.867	0.406	0.095	0.660
R	pMC	0.625	-0.290	1.510	0.447	-0.174	0.419
L	IFC	0.319	-0.273	1.009	0.288	-0.190	0.376
R	IFC	0.293	-0.440	0.920	0.323	-0.385	0.064

***SUPPLEMENTARY TABLE 1**. Voice selective response (Voice>Silence) correlation with hallucination proneness results: ROI = region of interest, (a/m/p) STG = (anterior /middle /posterior) superior temporal gyrus, pMC = premotor cortex, IFC = inferior frontal cortex, μ = mean activation from contrast, Min = minimum score; Max = maximum score, SD = standard deviation, LSHS = Launay-Slade Hallucination Proneness scale*

SUP TABLE 2 | NON-VOICE SENSITIVE RESPONSE CORRELATION							
		ROI				LSHS	
Hem.	Label	μ	Min	Max	SD	r	p
	aSTG	1.189	0.398	2.288	0.479	-0.326	0.120
L	mSTG	1.505	0.470	2.650	0.586	-0.086	0.689
	pSTG	1.511	0.250	2.600	0.560	-0.088	0.683
	aSTG	1.019	0.180	1.720	0.452	-0.145	0.499
R	mSTG	1.295	0.440	2.500	0.515	-0.050	0.817
	pSTG	1.213	0.471	1.867	0.406	-0.106	0.622
R	pMC	0.625	-0.290	1.510	0.447	-0.329	0.118
L	IFC	0.319	-0.273	1.009	0.288	-0.208	0.329
R	IFC	0.293	-0.440	0.920	0.323	-0.614	0.001

***SUPPLEMENTARY TABLE 2**. Non-voice selective response (Non-voice>Silence) correlation with hallucination proneness results: ROI = region of interest, (a/m/p) STG = (anterior /middle /posterior) superior temporal gyrus, pMC = premotor cortex, IFC = inferior frontal cortex, μ = mean activation from contrast, Min = minimum score; Max = maximum score, SD = standard deviation, LSHS = Launay-Slade Hallucination Proneness scale*

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Chapter 4

Expectancy changes the self-monitoring of voice identity

Johnson, J.F., Belyk, M., Schwartze, M., Pinheiro, A.P., & Kotz, S.A.

Abstract. Self-voice attribution can become difficult when voice characteristics are ambiguous, but functional magnetic resonance imaging (fMRI) investigations of such ambiguity are sparse. We utilized voice-morphing (self-other) to manipulate (un-)certainty in self-voice attribution in a button-press paradigm. This allowed investigating how levels of self-voice certainty alter brain activation in brain regions monitoring voice identity and unexpected changes in voice playback quality. FMRI results confirmed a self-voice suppression effect in the right anterior superior temporal gyrus (aSTG) when self-voice attribution was unambiguous. Although the right inferior frontal gyrus (IFG) was more active during a self-generated compared to a passively heard voice, the putative role of this region in detecting unexpected self-voice changes during the action was demonstrated only when hearing the voice of another speaker and not when attribution was uncertain. Further research on the link between right aSTG and IFG is required and may establish a threshold monitoring voice identity in action. The current results have implications for a better understanding of the altered experience of self-voice feedback in auditory verbal hallucinations.

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1 Introduction

The self-monitoring of the voice relies on comparing what we expect to hear and what we actually hear (Frith, 1992; Wolpert & Kawato, 1998). However, in a dynamic environment sensory feedback is often ambiguous, e.g., when listening to multiple speakers. Any judgment of the voice source further depends on how much sensory feedback deviates from expectations (Feinberg, 1978). Minor deviations regarding one's own voice are typically self-attributed and used to compensate motor control. Major deviations may lead to source-attributing the voice to another person. The study of misattributed self-voice is often associated with auditory verbal hallucinations (AVH) in patients with psychotic disorders (Kumari et al., 2010; Sapara et al., 2015). However, under conditions of ambiguous feedback, healthy individuals also display uncertainty in attributing the source of their own voice (Asai & Tanno, 2013; Pinheiro et al., 2019). Functional neuroimaging studies of self-voice monitoring have examined the neural substrates of self-other voice attribution but have so far not examined responses to uncertainty in ambiguous conditions (e.g., Allen et al., 2006; Fu et al., 2006). However, we need to better understand how the brain establishes correct self and other voice attribution and where and how the voice is processed in uncertain conditions to better understand the mechanisms underlying dysfunctional self-monitoring.

Previous research has reported that unaltered self-voice production leads to reduced functional brain activity in the auditory cortex (Christoffels et al., 2007). This motor-induced suppression (MIS) is compatible with findings of numerous studies employing diverse methodology. It is similar to the N1 suppression effect, a modulation of the event-related potential of the electroencephalogram (EEG) (e.g., Behroozmand & Larson, 2011; Heinks-Maldonado et al., 2005; Pinheiro et al., 2018; Sitek et al., 2013; Wang et al., 2014), or M1 suppression in magnetoencephalography (Houde et al., 2002; Numminen et al., 1999; Ventura et al., 2009), weakened activity in electrocorticography and at intracranial electrodes (Chang et al., 2013; Greenlee et al., 2011), or direct- and inter-cell recordings in non-human primates (Eliades & Wang, 2008; Müller-Preuss & Ploog, 1981).

In addition to suppressed activity in the auditory cortex, self-voice monitoring activates a widespread system of functionally connected brain regions, including cortical motor

and speech planning areas as well as subcortical regions such as the thalamus and cerebellum (Behroozmand et al., 2015; Christoffels et al., 2007). Moreover, within the auditory cortex different regions contribute specialized roles in the processing of voice. Notably, the left lateral temporal cortex demonstrates a larger role in speech-related processing, while the right lateral temporal cortex plays an essential role in speaker-related features of voice (Belin et al., 2002; Ethofer et al., 2006, 2007; Formisano et al., 2008; Grandjean et al., 2005; Kotz et al., 2003; Moerel et al., 2012; Schirmer & Kotz, 2006; Wiethoff et al., 2008). In the current study, we focused on the perception of voice identity particularly ascribed to the right anterior superior temporal gyrus (aSTG) and the adjacent upper bank of the superior temporal sulcus (STS) (Belin and Zatorre, 2003; Belin et al., 2004; von Kriegstein et al., 2003; von Kriegstein & Giraud, 2004). Patient studies support this assumption as lesions or damage to the aSTG can lead to deficits in voice identity recognition (Gainotti et al., 2010; Gainotti & Marra, 2011; Hailstone et al., 2011; Van Lancker & Canter, 1982; Van Lancker & Kreiman, 1987).

MIS in voice monitoring is not only effective in voice production but also in response to voice recordings activated via a button press (Ford et al., 2007; Knolle et al., 2019; Pinheiro et al., 2018; Pinheiro et al., 2020b; Whitford et al., 2011) as well as for non-verbal sounds including tones (e.g., Aliu et al., 2009; Baess et al., 2009; Knolle et al., 2013). Furthermore, MIS seems to operate across modalities of sensory feedback and arises from various motor effectors (e.g., Blakemore et al., 1998; Leube et al., 2003; Miall & Wolpert, 1996; Wolpert & Kawato, 1998). One explanation for MIS is that the internal model of an expected action outcome is fed-forward to the relevant cortical regions to cancel out impending activity to the anticipated stimulus (Jordan & Rumelhart, 1992; Miall & Wolpert, 1996; Wolpert, 1997). Studies that experimentally manipulated sensory feedback created a mismatch between expected and actual outcome and indicated concomitant modulation or absence of MIS under such circumstances. EEG studies typically show decreased N1 suppression (e.g., Behroozmand & Larson, 2011; Heinks-Maldonado et al., 2005), while functional magnetic resonance imaging (fMRI) studies report a relative increase of STG activity when expected feedback is altered (Christoffels et al., 2007, 2011; Fu et al., 2006; McGuire et al., 1996; Zheng et al., 2010). With this approach, it is not only possible to make listeners uncertain about self- or other-voice attribution (Allen et al., 2004, 2005,

2006; Fu et al., 2006; Versmissen et al., 2007), but to also lead listeners to incorrectly attribute self-voice to another speaker (Allen et al., 2004, 2005, 2006; Fu et al., 2006; Johns et al., 2001, 2003, 2006; Kumari et al., 2010; Sapara et al., 2015). STG suppression only persists when the voice is correctly judged as self-voice in distorted feedback conditions (Fu et al., 2006). Critically, data reflecting uncertain voice attribution are often removed from fMRI analyses (Allen et al., 2005b; Fu et al., 2006). However, to gain a better understanding of voice attribution to internal or external sources, it is mandatory to specify such data and to define how the known voice attribution region of the STG reacts to uncertainty.

Next to the auditory cortex, activation in the right inferior frontal gyrus increases in response to distorted auditory feedback (Johnson et al., 2019). However, while attenuation of the right aSTG activation reflects expected voice quality, the right IFG is selectively responsive to unexpected sensory events (Aron et al., 2004). Increased right IFG activity has been reported when voice feedback is acoustically altered (Behroozmand et al., 2015, Fu et al., 2006; Guo et al., 2016; Tourville et al., 2008; Toyomura et al., 2007), delayed (Sakai et al., 2009; Watkins et al., 2005), replaced with the voice of another speaker (Fu et al., 2006), or physically perturbed during vocal production (Golfinopoulos et al., 2010). In response to unexpected sensory feedback in voice production, the right IFG produces a "salient signal", indicating the potential need to stop and respond to stimuli that may be affected by external influence.

In the current fMRI experiment, we investigated how cortical voice identity and auditory feedback monitoring brain regions respond to (un)certain self-other voice attribution. Participants elicited their own voice that varied along a morphing continuum from self to other voice, including intermediate ambiguous voices. Region of interest (ROI) analyses motivated by a priori hypotheses focussed on the right aSTG and the right IFG. The right aSTG ROI stems from a well-replicated temporal voice area (TVA) localizer task (Belin et al., 2000). The right IFG ROI conforms to a region responsive to experimental manipulation of auditory feedback previously identified in an activation-likelihood estimation (ALE) analysis (Johnson et al., 2019). Due to possible individual variability in thresholds for self-other voice attribution (Asai & Tanno, 2013), each participant underwent psychometric testing to determine individualized points of

maximum uncertainty on a continuum from self to other voice. The primary goal was to test if (a) MIS of self-voice in the right aSTG is present, and the degree of suppression is greater when self-voice attribution is certain compared to uncertain, and (b) right IFG activation would increase in response to voice uncertainty or externalization. Confirming these results would further substantiate EEG findings regarding MIS for self-voice elicited via button-press (Ford et al., 2007; Knolle et al., 2019; Pinheiro et al., 2018, 2020b; Whitford et al., 2011), indicating that suppressed activity in auditory cortex aligns with predicted self-voice quality and not only as a function of expected quality of voice feedback.

2 Materials and methods

2.1 Participant recruitment

Twenty-seven participants took part in the study. The data of two participants were discarded due to scanning artefacts. Of the remaining 25 (17 female), the average age was 21.88 years (*SD* = 4.37; range 18 to 33). Inclusion criteria assured that participants had no diagnosis of psychological disorder, normal or corrected-to-normal vision, reported no hearing loss, and no evidence of phonagnosia. The latter was tested with an adapted version of a voice-name recognition test (Roswandowitz et al., 2014). All participants gave written informed consent and received university study participant credit. This study was approved by the Ethical Review Committee of the Faculty of Psychology and Neuroscience at Maastricht University (ERCPN-176_08_02_2017).

2.2 Phonagnosia screening

Phonagnosia is a disorder restricting individuals from perceiving speaker identity in the voice (Van Lancker et al., 1988). We screened for phonagnosia using an adapted version of a phonagnosia screening task (see Roswandowitz et al., 2014). The task was composed of four rounds of successive learning and testing phases, in which participants initially listened to the voices of three speakers of the same gender. Identification of each speaker was subsequently tested 10 times with response accuracy feedback provided during the first half of the test trials. Finally, the task was repeated with stimuli of the gender not used in the first run. The presentation order of these runs was counterbalanced across participants.

2.3 Psychometric task

In a voice attribution task (VAT), participants heard neutral samples of the vowels /a/ and /o/. These samples varied in voice identity, which was morphed along a continuum from "self-voice" to "other-voice" using the STRAIGHT voice morphing software package (Kawahara 2003, 2006) running in MATLAB (R2019A, v9.6.0.1072779, MathWorks, Inc., Natick, MA). Samples of the self-voice (SV) and other-voice (OV), producing the two vowels were obtained from each participant and normalized for duration (500ms) and amplitude (70db), using the Praat software package (v6.0.28, http://www.praat.org/). The OV sample matched the gender of the participant. On this basis, 11 stimuli for each vowel were created along a morphing spectrum in steps of 10% morphing from SV to OV. In a two-alternative forced-choice (2AFC) task, participants listened to each stimulus 10 times presented in random order and responded to the question: Is the voice "more me" or "more other"? This procedure was repeated twice. In one run, stimuli were presented passively while in the other run participants were visually cued to press a button that elicited the next stimulus [Figure 1]. A total of 440 trials were presented across both runs. The duration across both runs was 26.7 minutes. This task was used to identify an individualized point of maximum ambiguity (PMA) along the morphing spectrum for each participant. The PMA was defined as the stimulus that was closest to chance level (50%) self-other judgement and used as the uncertain voice (UV) to inform subsequent fMRI analyses.

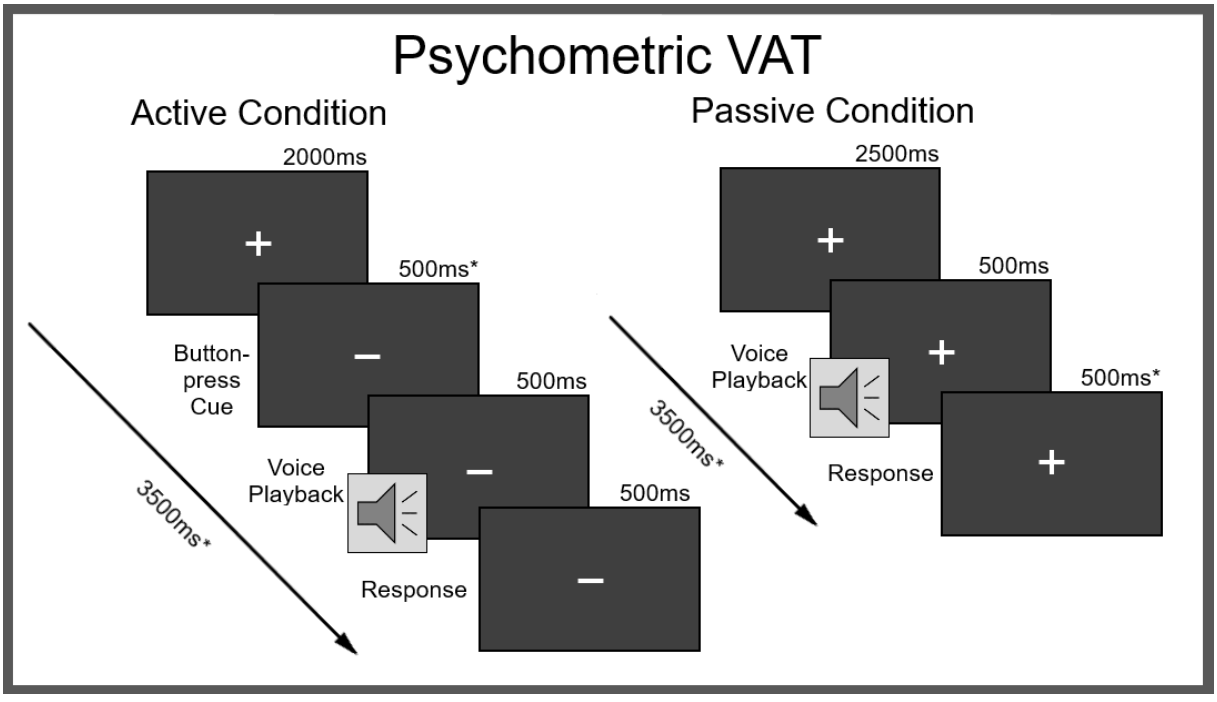

FIGURE 1. *Psychometric Voice Attribution Task (VAT): Active = button-press condition; Passive = hearing conditions, * = affected by individual motor response-time variability; Response = two-alternate forced-choice ("The voice sounded more like me." or "The voice sounded more like someone else.").*

2.4 FMRI tasks

2.4.1 Temporal Voice Area (TVA) Localizer. To identify voice sensitive brain areas, participants were scanned during a voice localizer task (Belin et al., 2000). This task is widely used to reliably probe activity along the bilateral temporal cortices (e.g., Pernet et al., 2015) designated as anterior, middle, and posterior TVA regions. Stimuli consisted of 8-second auditory clips with 20 vocal and 20 non-vocal sounds. In a single run, participants passively listened to these sounds and 20 silent trials of the same duration in pseudorandom order. A 2-second inter-stimulus-interval separated each trial, resulting in a total task duration of 10 minutes. Contrasting responses of vocal and non-vocal sounds identified brain regions selectively sensitive to voice processing. The peak activation in the anterior TVA (aSTG) of the right hemisphere was then chosen as the voice-attribution ROIs in the subsequent empirical fMRI investigation.

2.4.2 Voice Perception Task (VPT). Participants listened to passively presented or self-generated voice stimuli. When shown a cue signifying the active button-press condition, participants pressed a button to elicit voice stimuli, and conversely when shown a cue signifying the passive condition were instructed to do nothing [Figure 2]. In the active condition, half of the trials elicited a voice following the button press, while in the other half no voice was presented. In the passive condition, all trials involved the presentation of a voice. A subset of stimuli used in the VAT was selected for the VPT, specifically the 100, 60, 50, 40, and 0% self-voice morphs. Intermediate steps of 60, 50, and 40% were selected as pilot data had revealed that individual PMA fell within a range of 35-65% morphing, while morphs outside of this range produced high degrees of confidence in self versus other judgement. This ensured that every participant received the voice stimuli nearest to their subjective PMA. Trial onsets were 9 seconds (+/- 500ms) apart to allow the BOLD response to return to baseline before the presentation of the next stimulus started. To avoid the effects of adaptation suppression (Andics et al., 2010, 2013; Belin et al., 2003; Latinus & Belin., 2011; Wong et al., 2004), voice conditions were presented in a random order. Stimuli were presented via Sensimetrics S14 MR-compatible earphones, fitted with foam earplugs to reduce

interference from scanner noise (Sensimetrics Corporation). While in the scanner participants were required to confirm successful perception of the control stimulus at the standard 70db volume. Over two runs, a total of 100 trials were presented in each condition of Source (active and passive). Within each condition of Source, each voice stimulus (100, 60, 50, 40, and 0% morphs from self-to-other) was heard 20 times. 20 null trials were included to provide a baseline comparison of activity in response to experimental trials. The total duration of this task over both runs was 33 minutes.

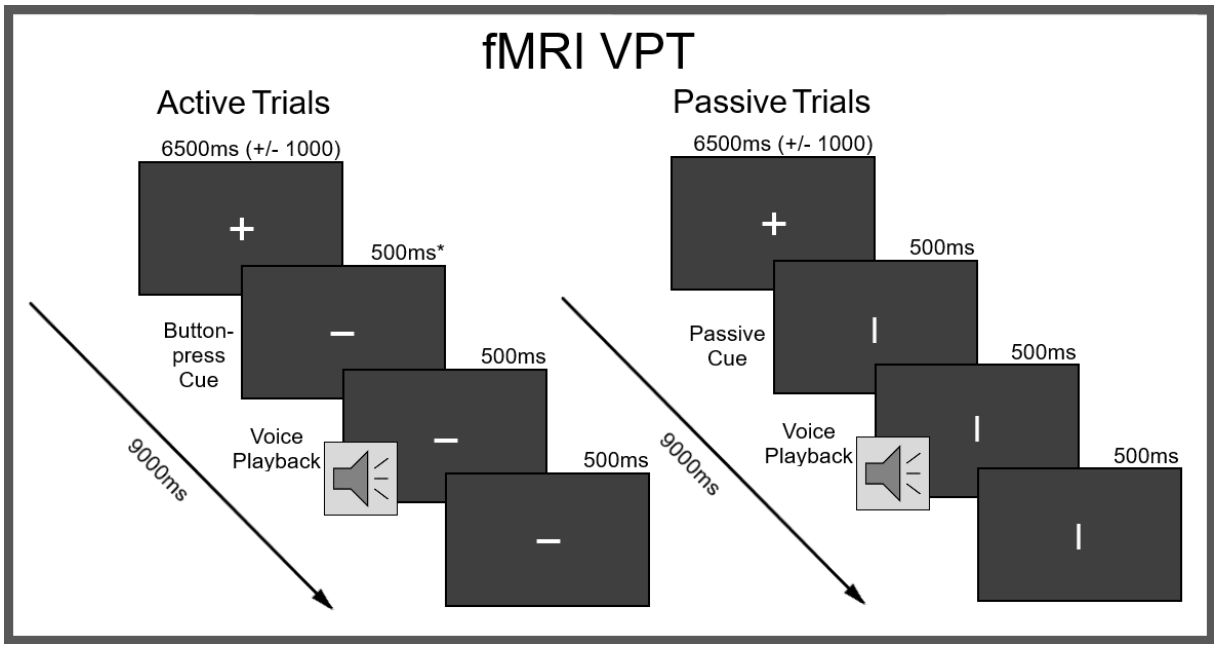

FIGURE 2. *fMRI Voice Perception Task (VPT): Active = button-press condition; Passive = hearing conditions, * = affected by individual motor response-time variability.*

2.5 FMRI data acquisition and analysis

Data acquisition was performed at a Siemens 3T Magnetom Prisma Fit MRI scanner at Scannexus facilities (Maastricht, the Netherlands), equipped with a 32-channel head coil (Siemens Healthcare, Erlangen, Germany). A structural whole brain T1-weighted single-shot echoplanar imaging (EPI) sequence was collected for each participant (field of view (FOV) 256mm; 192 axial slices; 1mm slice thickness; 1 x 1 x 1mm voxel size; repetition time (TR) of 2250ms seconds; echo-time (TE) 2.21ms).

Two functional tasks were conducted with T2-weighted EPI scans (FOV 208mm; 60 axial slices; 2mm slice thickness; 2 x 2 x 2mm voxel size; TE 30ms; flip angle = 77°). Both tasks applied a long inter-acquisition-interval where the time between consecutive image acquisition (2000ms) was delayed, resulting in a TR of 10 and 9 seconds for the

TVA localizer and VPT, respectively. This allowed auditory stimuli to be presented during a period of relative silence to reduce noise artefacts and for volume acquisition to proceed during a period of peak activation in the auditory cortex (Belin et al., 1999; Hall et al., 1999).

2.5.1 Preprocessing. DICOM image data were converted to 4D NIFTI format using the Dcm2Nii converter provided in the MRIcron software package (https://www.nitrc.org/projects/mricron/). The topup tool (Smith, et al., 2004) implemented in FSL (www.fmrib.ox.ac.uk/fsl) was used to estimate and correct for susceptibility induced image distortions. Pre-processing was performed using SPM12 (Wellcome Department of Cognitive Neurology, London, UK). A pre-processing pipeline applied slice timing correction, realignment and unwarping, segmentation, normalization to standard Montreal Neurological Institute (MNI) space (Fonov et al., 2009) as well as smoothing with a full width at half maximum (FWHM) 8mm isotropic Gaussian kernel.

2.5.2 General Linear Model (GLM) Analysis. The TVA localizer and experimental VPT fMRI data were analysed with a standard two-level procedure in SPM12. For the TVA localizer, contrast images for Vocal > Non-Vocal and Vocal > Silent were estimated for each participant. To test for the main effect of interest, a conjunction analysis ((V > NV) ∩ (V > S)) was performed. A second level random-effects analysis tested for group-level significance. A first-level fixed-effects GLM of the VPT data calculated contrast estimates for each participant. Contrast estimates were then used in the subsequent hypothesis-driven ROI analysis to investigate TVA activity.

2.5.3 Linear Mixed Model (LMM) ROI Analyses. Two spherical (5mm) ROIs were selected for analysis: the right aSTG/S in Brodmann Area (BA) 22 (MNI coordinates x 58, y 2, z -10) defined by the TVA fMRI localizer task, and the right IFG opercular region in BA 44 (MNI coordinates x 46, y 10, z 4) defined in a previous fMRI meta-analysis (Johnson et al., 2019) [see Figure 3]. For both ROIs, the mean contrast estimates were produced for each SV, UV, and OV condition against the null trials. Using these values as input, a 2x3 factorial design was formulated using the factors of Source and Voice. The two-levelled factor Source included self-generated (A) and passively-heard (P)

playback of voice recordings. The three-levelled factor Voice included self-attributed (SV), other-attributed (OV), and ambiguous voice (UV).

Data were analysed in R v3.6.1 (R Core Team, 2019) running on OS v10.11.6. Data handling and visualization were supplemented with the tidyverse (Wickham, 2017). Linear Mixed Models (LMMs) were fit with lme4 (Bates et al., 2015). Separate LMMs were fitted for contrast estimates of the IFG and the aSTG ROIs with Source (A and P), Voice (SV, OV and UV), and their interaction as fixed effects. Participant was modelled as a random intercept. Model residuals were examined for potential outliers. Five data points were removed from the IFG analysis and one was removed from the aSTG analysis.

The main effects of Voice, Source and their interaction were tested with the afex package using Kenward-Rogers degrees of freedom (Singmann et al., 2015). Estimated marginal means and confidence intervals were computed with the emmeans package (Lenth, 2020) for visualization. All p-values are corrected for multiple comparisons controlling at a false-discovery rate (FDR) of 0.05. Furthermore, to investigate the effect that (un)certainty has on the suppression of the right aSTG, we compared contrasts of A > P in each voice condition to see if it differs for SV stimuli as compared to OV or UV stimuli.

Finally, to provide clear effects of each treatment condition (UV and OV) compared to the control variable of one's own voice (SV), contrast estimates were reported via two-tailed paired sample t-tests. These comparisons were done within both active (A) and passive (P) conditions. Furthermore, we provide BOLD whole-brain activation maps for each Source condition (P and A) against null trials [see Supplementary Information].

3 Results

3.1 VAT results

Psychometric analysis of the VAT indicated little variability in the degree of morphing between SV and OV required to elicit responses at chance level (50%), which we identified as the point of maximum ambiguity. For the A condition, nine participants had PMAs at 40%, eight at 50% and ten at 60% morphing. In the passive condition, eleven participants required 40%, seven 50%, and nine 60% morphing. There was no

significant difference between the average morphing required to elicit PMA in A (μ = 50%, *SD* = 0.085) and P (μ = 50%, *SD* = 0.087) conditions. Although no participant matched criteria for phonagnosia as specified by the screening task, VAT data from one participant was excluded due to an inability to reliably differentiate between their own and other voices.

3.2 TVA localizer results

The TVA fMRI localizer produced four significant cluster-level activations [see Table 1 for details]. Within two large bilateral STG (BA 22) clusters, each included three peak-level significant activations. These peaks correspond to the posterior, middle, and anterior STG. Two smaller clusters were found in the right precentral gyrus (BA 6), the left IFG (BA 44), and the left inferior parietal lobule (BA 40). All significant cluster- and peak-level coordinates survived FDR correction of 0.05. These results replicate the pattern of TVA regions of peak activity (e.g., Belin et al., 2000; Fecteau et al., 2004; Latinus et al., 2013; Pernet et al., 2015). The right aSTG peak was chosen for the ROI analysis of self-voice-attribution.

TABLE 1 | TVA LOCALIZER RESULTS

#	Hem.	Label	BA	MNI coordinates (x, y, z)			Cluster-level p-FDR	Peak-level p-FDR	Size (voxels)
		pSTG	22	-60	-24	0		9.29e-12	
1	L	aSTG	22	-58	-10	-2	2.76e-14	9.04e-11	4551
		mSTG	22	-66	-16	-2		3.51e-08	
		pSTG	22	58	-24	-2		1.07e-09	
2	R	aSTG	22	58	2	-10	2.05e-14	2.00e-09	4565
		mSTG	22	58	-8	-6		2.74e-09	
3	R	preCG	6	52	52	0	0.007	3.44e-04	408
4	L	IFG	44	-42	14	22	0.019	0.002	294

TABLE 1. *L: left, R: right, (p/a/m)STG: posterior/anterior/middle superior temporal gyrus, preCG: precentral gyrus, IFG: inferior frontal gyrus; All listed significant regions survived FDR-corrected threshold 0.05.*

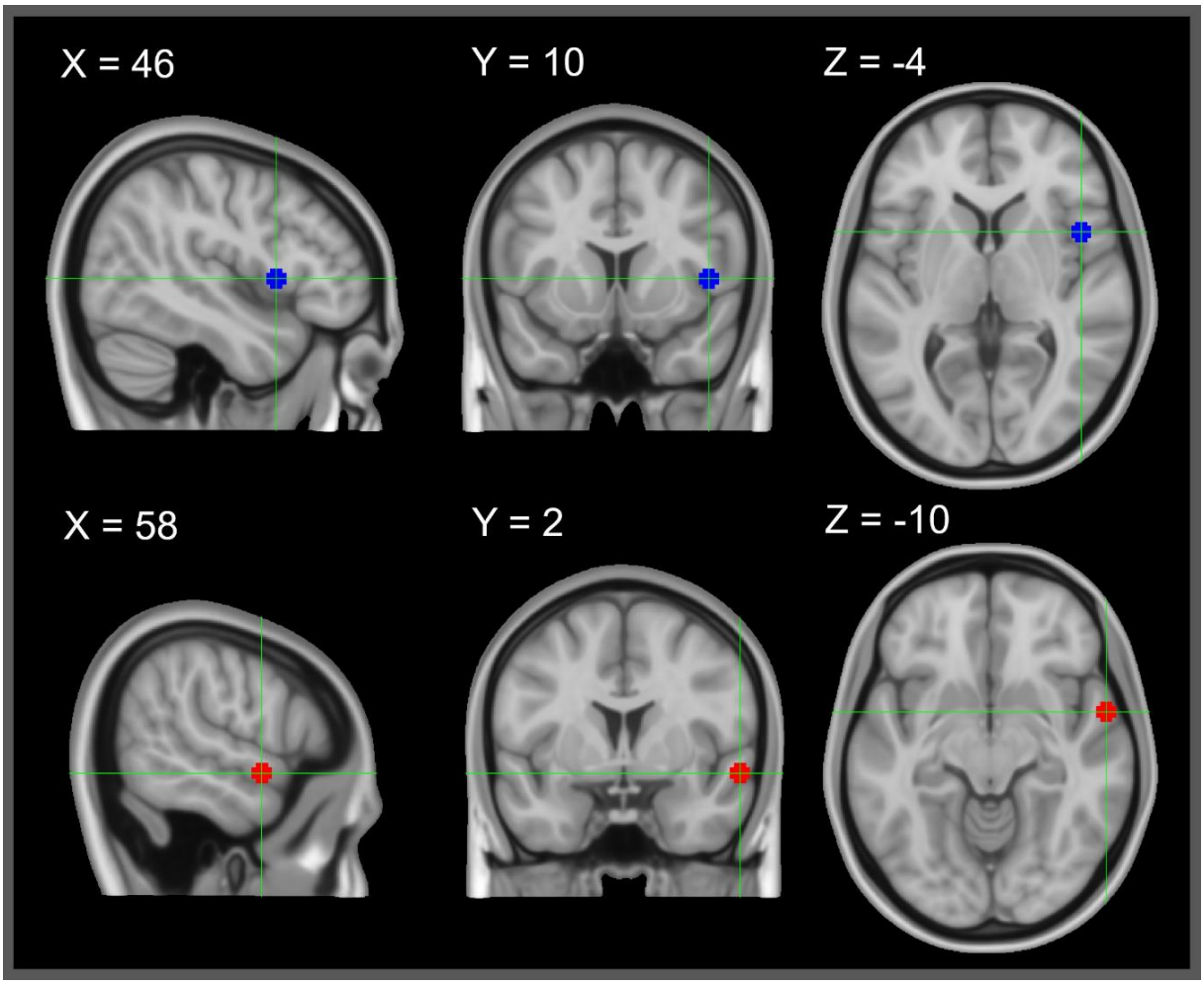

FIGURE 3. *Regions of Interest: Blue: right inferior frontal gyrus; MNI coordinates x 58, y 2, z −10; determined from ALE neuroimaging meta-analysis (Johnson et al., 2019). Red: right anterior superior temporal gyrus; MNI coordinates x 46, y 10, z 4; determined in our sample from fMRI temporal voice area localizer task.*

3.3 LMM ROI results

Contrast estimates calculated for each Voice condition (SV, UV, and OV) within each Source condition (A and P) were used as input for the 2x3 factorial design [see Supplementary Information]. Linear mixed model analysis of the right aSTG [Table 2A, Figure 4A] produced an FDR-corrected significant main effect for the factor of Voice ($F_{2,118.94}$ = 4.90, p = 0.021). No significant effect was observed for Source ($F_{1,118.92}$ = 0.53, p = 0.47). A trend for the expected interaction effect between Voice and Source was observed, although it did not survive FDR correction for multiple comparisons ($F_{2,118.94}$ = 3.40, p = 0.065). Our a priori hypotheses regarding a difference in

suppression effects (A > P) between voice conditions however was supported by the finding that MIS is observed preferentially for SV stimuli (t_{119} = -2.7, p = 0.021).

The LMM analysis was repeated for the right IFG ROI [Table 2B, Figure 4B]. A significant FDR-corrected main effect of Source was observed ($F_{1,116.04}$ = 9.93, p = 0.002). No main effect was found for the factor of Voice ($F_{2,115.95}$ = 1.52, p = 0.26), and no interaction between Voice and Source were observed ($F_{2,115.81}$ = 1.60, p = 0.26).

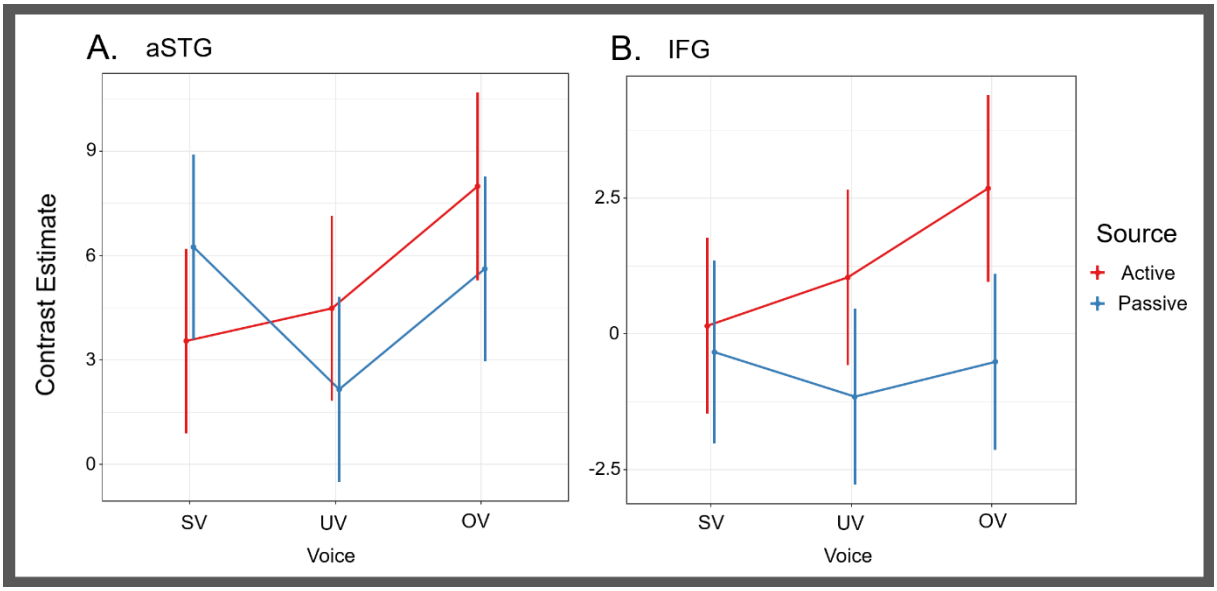

FIGURE 4. *fMRI Voice Perception Task (VPT) LMM Results: Linear mixed model analysis on ROIs in A) right anterior superior temporal gyrus (aSTG) and B) right inferior frontal gyrus (IFG). Active: button-press condition, passive: hearing condition, SV: self-voice, UV: uncertain-voice, OV: other-voice. Hypothesis-driven analysis in right aSTG confirmed motor induced suppression (for contrast active > passive) for only SV as compared to UV or OV (t(119) = −2.7, p = .021)*

Finally, for both ROIs we report direct comparisons of treatment (UV and OV) and control (SV) conditions within each source conditions (A and P). Activation of the right aSTG was reduced when participants actively produced SV compared to OV (t_{121} = -2.773, p = 0.0064), but not compared to UV (t_{121} = -0.591, p = 0.5553). Conversely, activation of the right aSTG was no different when participants were passively exposed to SV compared to the OV (t_{118} = 0.401, p = 0.6891), but increased compared to UV (t_{118} = 2.612, p = 0.0102). Therefore, the right aSTG was less active when self-generating a voice certain to be one's compared to an external source, and more active when passively hearing a voice certain to be one's own compared to when uncertain.

In the right IFG, activation was reduced when participants actively produced SV compared to OV (t_{116} = -2.316, p = 0.0223), but not compared to UV (t_{115} = -0.846, p = 0.3993). When passively exposed, activation of the right IFG was no different when presented with SV compared to OV (t_{117}= 1.66, p= 0.8681), or to UV (t_{117} = 0.758, p = 0.4499). In summary, in the right IFG a greater activation relative to the self-voice control was reported in only the externalized self-generated voice

4 Discussion

The current study investigated how unexpected sensory feedback affects certainty in self-voice attribution. We report first fMRI evidence that aligns with EEG reports, namely self-voice MIS is observed in the anterior region of the STG even when one's own voice is elicited by a button press. Expected self-voice quality, learned through long-term experience with self-voice feedback sufficiently modulates MIS. Importantly, this effect was specific to vocal properties matching the producer's own voice and was not observed when hearing another voice or being uncertain about the voice of a speaker. The right IFG showed increased activation in response to the self-generated voice compared to listening to the same voice. It is possible that this response is driven by voice trials not attributed to oneself. This region is known to be more active when perceived stimuli conflict with expected sensory feedback. Together, these findings suggest a differentiation between and a potential interplay of right IFG and anterior STG in voice processing, and more specifically feedback monitoring of self-generated voice and voice attribution.

4.1 Voice identity and motor-induced suppression in the anterior STG

Our results confirm right anterior STG/S involvement for voice identity and indicate that this region plays a specific role in segregating the speaker's voice from other voices in the monitoring of auditory feedback. We replicate previous TVA findings that the STG and upper bank of the STS contain three bilateral voice patches [Table 1] (Belin et al., 2000; Pernet et al., 2015). The processing of speech-related linguistic ("what") features have been attributed predominantly to the left hemisphere, while speaker-related paralinguistic ("who") features have been attributed predominantly to the right hemisphere (Belin et al., 2002; Ethofer et al., 2006, 2007; Formisano et al., 2008;

Grandjean et al., 2005; Kotz et al., 2003; Moerel et al., 2012; Schirmer & Kotz, 2006; Wiethoff et al., 2008). Furthermore, regions of the right lateral temporal cortex are specialized for different speaker-related information. Identity attribution is localised to the anterior region of the STG/S (Belin et al., 2003; Fecteau et al., 2004; Latinus et al., 2013; Schelinski et al., 2016; von Kriegstein et al., 2003; von Kriegstein & Giraud, 2004). Considering voice-identity processing as a multi-stage process, low-level acoustics features are evaluated in the posterior STG for cues relevant to speaker identification; the extracted cues are then further processed and compared to prototypes for deviance detection in the middle STG, and finally voice identity recognition occurs in the anterior STG (Maguiness et al., 2018).

We conducted ROI analyses to test voice identity in the right anterior STG due to its responsiveness to variation in voice identity but did not include other TVA regions in our analysis. This allowed us to detect fine-grain differences in activation patterns influenced only by voice identity in a region that is related to the perception of one's own voice. To provide sufficient information for the extraction of paralinguistic speaker-related features, steady 500ms vowel excerpts were chosen as voice samples (Pinheiro et al., 2018; Schweinberger et al., 1997, 2011; van Berkum et al., 2008). Although vowels provide fundamental cues that allow differentiating between speakers (Belin et al., 2004; Kreiman & Sidtis, 2011; Latinus & Belin, 2011; Schweinberger et al., 2014), to the best of our knowledge no study has yet confirmed whether such basic stimuli carry enough identity cues to allow for explicit self-recognition (Conde et al., 2018). Our results confirm that the use of short vowels is sufficient to accurately recognize self verses other voices.

Suppression of self-generated relative to passively-heard voice in the right anterior STG occurred only within SV (see Figure 4A). One possible interpretation for this selective finding is that participants are most familiar with their own voice and they can therefore predict the features of their own voice more efficiently. In the right anterior STG, voice identity is defined by the extent that speaker-related cues deviate from prototypes of expected voice qualities (Andics et al., 2010, 2013; Bruckert et al., 2010; Latinus & Belin, 2011; Latinus et al., 2013; Mullennix et al., 2011; Petkov & Vuong, 2013; Schweinberger et al., 2014). These referential prototypes are learned through mean-

based coding (Hoffman & Logothesis, 2009), and differ for male and female voices (Latinus et al., 2013; Charest et al., 2013). While it is clear that low-level acoustic processing is involved in recognizing the identity of a speaker (Baumann & Belin, 2010; Gaudrain et al., 2009; Kreitewolf et al., 2014; Nolan et al., 2011; Smith & Patterson, 2005; Smith et al., 2007; Zheng et al., 2011), the specific features that drive voice identification vary from voice to voice (Kreiman et al., 1992; Latinus & Belin, 2012; Lavner et al., 2000, 2001; Xu et al., 2013). Furthermore, variable acoustic features of the voice do not only exist between speakers, but also within individual speakers (Lavan et al., 2019). Therefore, increased experience with the voice of a specific speaker facilitates more efficient recognition of voice identity. As speakers are most experienced with their own voice, little divergence from mean-based coding is expected.

Alternatively, MIS of the self-voice in a dynamic multi-speaker environment is important for the segregation of internally- and externally-controlled voice stimuli. During vocalization, an efference copy of the motor command is sent from motor planning areas to auditory and sensorimotor cortical regions to notify of impending feedback (Hickok et al., 2011; Hickok, 2012; Kearney & Guenther, 2019; Rauschecker & Scott, 2009; Rauschecker, 2011; Tourville & Guenther, 2011). Error-cells in the posterior STG (planum temporale) receive these signals from Broca's area to remain inactive in response to the expected self-voice, and to engage when perceiving voice feedback outside the control of the speaker (Guenther et al., 2006). To date, fMRI research using vocal feedback paradigms, has provided evidence for this form of MIS dependent on vocal production. For example, MIS has been reported for unaltered vocal production relative to hearing a recording of self-voice or in a noisy environmental (Christoffels et al., 2007), when acoustically distorted (Christoffels et al., 2011; Fu et al., 2006; McGuire et al., 1996; Zheng et al., 2010), or replaced with the voice of another speaker (Fu et al., 2006; McGuire et al., 1996). However, as these paradigms all rely on vocal production, they could not isolate how voice identity engages the anterior STG in voice production. EEG research has provided evidence for MIS in the auditory cortex that does not depend on vocal speech production as it is observed even when sounds are elicited by a button press. For example, MIS of the N1 response was reported for both, vocal (Behroozmand & Larson, 2011; Heinks-Maldonado et al., 2005; Sitek et al., 2013; Wang et al., 2014) and button-press elicited self-voice (Ford et al., 2007; Knolle et al.,

2019; Pinheiro et al., 2018, 2020b; Whitford et al., 2011). In line with previous EEG evidence, the current findings confirm self-voice suppression as a marker of voice identity in the right anterior STG. The reported MIS is specific to self-voice processing, providing further evidence of voice identity suppression independent of previously reported cortical suppression during unperturbed speech. Importantly, this pattern was observed only for own voice attribution, and was not present when the voice was distorted to an extent that self-attribution was uncertain.

4.2 Expected feedback and the IFG

The right IFG was more strongly activated when participants generated their own voice with a button press as compared to passive listening to their own voice. This finding confirms that this region is more responsive to sounds triggered by oneself, potentially as part of an auditory feedback loop. Increased activity in this region has been observed in response to acoustically altered (Behroozmand et al., 2015, Fu et al., 2006; Guo et al., 2016; Tourville et al., 2008; Toyomura et al., 2007), physically perturbed (Golfinopoulos et al., 2010), and externalized voice feedback (Fu et al., 2006).

In response to unexpected sensory information, the right IFG plays a significant role in relaying salient signals to attention networks. Moreover, the right IFG is part of a prediction network, which forms expectations and detects unexpected sensory outcomes (Siman-Tov et al., 2019). When prediction errors are detected, an inferior frontal network produces a salience response (Cai et al., et al., 2014; Chang et al., 2013; Power et al., 2011; Seeley, 2010). Salience signals engage ventral and dorsal attention networks, overlapping the right inferior frontal cortex. The ventral attention network responds with bottom-up inhibition of ongoing action (Aron et al., 2004, 2014), such as halting manual or speech movement (Aron & Poldrack, 2006; Aron, 2007; Chevrier et al., 2007; Xue et al., 2008). Correspondingly, damage to prefrontal regions affects the ability to stop one's own actions (Aron et al., 2003), and is similarly diminished when the IFG is deactivated with TMS (Chambers et al., 2006). The salience response may also engage the dorsal attention network to facilitate a top-down response (Corbetta & Shulman, 2002; Dosenbach et al., 2007; Eckert et al., 2009; Fox et al., 2006), for example, in goal-directed vocal compensation to pitch-shift (Riecker et al., 2000; Toyomura et al., 2007; Zarate and Zatorre, 2005) or somatosensory perturbation

(Golfinopoulos et al. 2011). The right IFG in the current study maps with a region determined by an ALE meta-analysis of neuroimaging studies that experimentally manipulated auditory feedback in vocal and manual production (Johnson et al., 2019). As the current experiment required no explicit response to a change in stimulus quality, we hypothesized that increased activity in the right IFG may represent the initial salience response to unexpected voice quality. However, the effect of voice identity in the right IFG did not reach significance, and there was no significant interaction between stimulus source and voice identity in this region. We note that the main effect of source appears most strongly driven by unfamiliar or ambiguous voices, with an intermediate level increase in the uncertain condition [see Figure 4B]. It is possible that substantial variability in the data limiting these results was due to the passive nature of the task with no overt attention to stimulus quality. As activity in this region is associated with attention and subsequent inhibition/adaptation responses, the degree to which each participant attended to the change in stimulus quality is unclear. However, simple contrast analyses relative to self-voice demonstrated a significant increase in activation for only self-generated other-voice. It is possible that this region is solely activated when voice feedback is manipulated to the extent that it is externalized. Conversely, although psychometric testing confirmed the subjective ability of participants to correctly recognize their own and other voices at a behaviour level, it is possible that the brief vowel stimuli did not provide sufficient information to signal a strong response to unexpected changes in self-voice leading to uncertainty. Further research is therefore needed to clarify whether the right IFG is responsive to voice identity, and to which extent this may be driven by the degree of salience elicited in divergence from expected qualities of self-voice.

5.3 Variability in self-monitoring thresholds

Although recordings of self-voice can produce a feeling of eeriness for listeners as compared to when spoken (Kimura et al., 2018), people nevertheless recognize recorded voice samples as their own (Candini et al., 2014; Hughes & Nicholson, 2010; Kaplan et al., 2008; Nakamura et al., 2001; Pinheiro et al., 2016a, 2016b, 2019; Rosa et al., 2008; Xu et al., 2013). However, in ambiguous conditions (i.e., acoustic distortion), the ability to accurately attribute a voice to oneself is diminished (Allen et al., 2004,

2005, 2006, 2007; Fu et al., 2006; Kumari et al., 2010). As ambiguity increases, an attribution threshold is passed, initiating a transition from uncertainty to externalization (Johns et al., 2001, 2003, 2006; Versmissen et al., 2007). This threshold, however, varies from person to person (Asai and Tanno, 2013). It was therefore necessary to determine the degree of morphing required to elicit uncertainty in the attribution of voice identity via separate 2AFC psychometric analysis for each participant. In doing so, we could confirm that fMRI responses in the PMA condition were specific to the experience of maximum uncertainty, regardless of any variability in the individual thresholds. These results confirmed that participants were able to discriminate their self-voice from an unfamiliar voice, with relatively little variation regarding the point of maximum ambiguity.

An externalization bias is particularly prominent in schizophrenia patients who experience AVH (Allen et al., 2004, 2007; Costafreda et al., 2008; Heinks-Maldonado et al., 2007; Johns et al., 2001, 2006; Pinheiro et al., 2016a). It has been hypothesized that the processing of salient stimuli with minimal divergence from expectations leads to an externalization bias that may manifest in the experience of AVH (Sommer et al., 2008). Correspondingly, as the severity of AVH symptoms increase, accuracy in self-attribution voice diminishes (Allen et al., 2004, 2006; Pinheiro et al., 2016a). Notably, this symptomology does not only exist within patient groups. Individuals who present sub-clinical symptoms but are at a high risk to develop psychosis, display levels of self-monitoring performance similar to patients who meet a clinical diagnosis of schizophrenia (Johns et al., 2010; Versmissen et al., 2007). Indeed, proneness to hallucinate is a continuum and AVH are experienced in the general populations as well, although at lower rates (Baumeister et al., 2017). Even in non-clinical populations, AVH are associated with a bias towards external voice attributions (Asai & Tanno, 2013; Pinheiro et al., 2019). The current findings may be of value in the understanding of the neural substrates underlying dysfunctional self-other voice attribution. In light of our observation that the anterior STG displays a qualitatively different activation for self-voice relative to an unfamiliar voice and the hypothesized influence of right IFG overactivity in salience detection in AVH, we suggest future research in high-risk groups to assess a possibly altered interaction between these two regions. Structural and functional connectivity MRI analysis may help explain whether aberrant

communication between these two regions, or individual changes in either or both regions lead to this symptomatology.

5 Conclusion

The goal of the current experiment was to investigate how levels of self-voice certainty alter brain activity in voice identity and feedback quality monitoring regions of the brain. By replicating earlier findings using a voice area localizer task, we isolated a putative voice identity region in the right anterior STG. Our results indicate activity in this TVA is suppressed only for the self-generated voice. Although the involvement of the right IFG was not confirmed in processing unexpected features of uncertain voice identity, increased IFG activity in response to the self-generated other-voice was observed indicating a possible role of feedback-monitoring for externalized voice. Using a novel self-monitoring paradigm, we provide the first fMRI evidence for the effectiveness of button-press voice-elicitation in modulating identity-related MIS in the auditory cortex. Further, we present novel findings on the effectiveness of brief vowel excerpts to provide sufficient paralinguistic information to explicitly identify one's own voice. Finally, we suggest a potential dynamic interaction of the right anterior STG and IFG in self-voice monitoring. The feedback monitoring frontal region may inform the temporal voice identity region whenever a salience threshold has been passed and voice feedback is influenced by or under control of an external actor. The implications of the current results may be particularly relevant to the externalization of self-generated voice in AVH.

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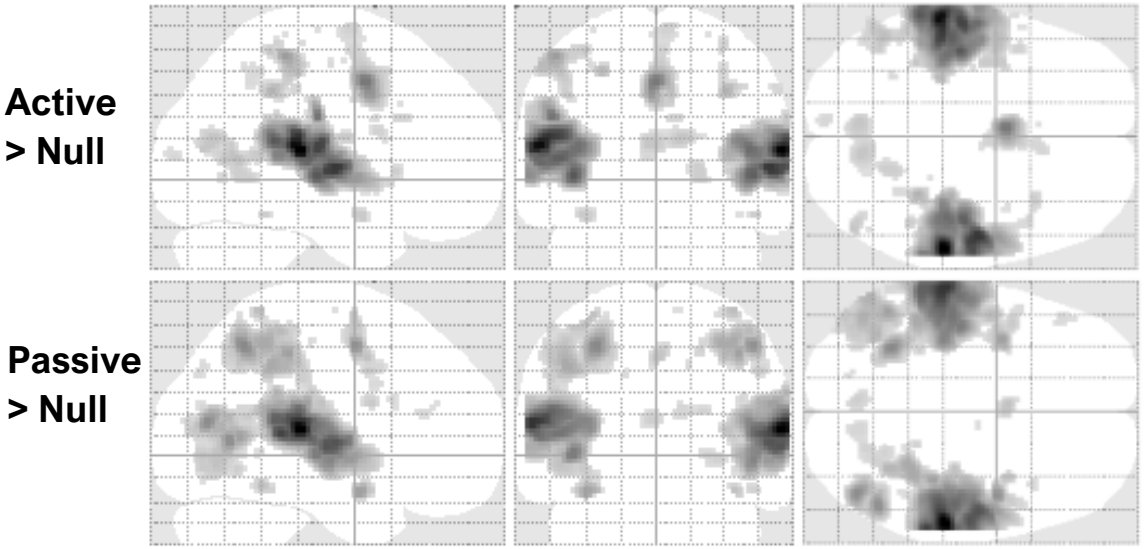
Disclosures. All authors disclose no potential sources of conflict of interest.

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

Supplementary information

SUP TABLE 1 | CONTRAST ESTIMATES				
Source	***Voice***	***N***	***μ***	***SD***
A. Right aSTG				
P	SV	24	6.29	6.74
	UV	24	2.55	7.75
	OV	24	5.49	6.30
	All	24	4.78	7.05
A	SV	24	3.71	6.69
	UV	24	4.46	7.98
	OV	24	6.29	9.19
	All	24	4.82	7.98
B. Right IFG				
P	SV	21	-0.35	5.12
	UV	21	-1.72	4.82
	OV	21	-0.19	3.18
	All	21	-0.75	4.44
A	SV	21	0.37	4.05
	UV	21	1.02	3.31
	OV	21	2.06	4.74
	All	21	1.15	4.07

SUPLEMENTARY TABLE 1. *Contrast estimates of each condition vs. null trials (input data for factorial design used in LMM). Source condition: P = passively-heard, A = active button-press. Voice condition: SV = self-voice, UV = uncertain-voice, OV = other-voice. , N = number of datapoints, μ = mean activation from contrast, SD = standard deviation,*



SUPPLEMENTARY FIGURE 1*. Contrast estimates of Source condition vs. null trials: FWE-corrected 0.05, extent threshold k = 0, degrees of freedom 1.0, 24.0.*

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Chapter 5

Variability in white matter structure relates to hallucination proneness

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Abstract. Hallucinations are a prominent transdiagnostic psychiatric symptom but are also prevalent in individuals who do not require clinical care. Moreover, persistent psychosis-like experience in otherwise healthy individuals may be related to an increased risk to transition to a psychotic disorder. This suggests a common aetiology across clinical and non-clinical individuals along a multidimensional psychosis continuum that may be detectable in structural variations of the brain. The current diffusion tensor imaging study assessed 50 healthy individuals (37 female) to identify possible differences in white matter associated with hallucination proneness (HP). This approach circumvents potential confounds related to medication, hospitalization, and disease progression common in clinical individuals. We determined how HP relates to white matter structure in selected association, commissural, and projection fibre pathways putatively linked to psychosis. Increased HP was associated with enhanced fractional anisotropy (FA) in the right uncinate fasciculus, the right anterior and posterior arcuate fasciculus, and the corpus callosum. Although FA in cortico-cerebellar pathways revealed no relationship, streamline quantity between the left cerebellum and the right motor cortex positively correlated with HP. These findings support the notion of a psychosis continuum, providing first evidence of structural white matter variability associated with HP in healthy individuals. Furthermore, alterations in the targeted pathways likely indicate an association between HP-related structural variations and the putative salience and attention mechanisms that these pathways subserve.

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Chapter 6

Integrated Discussion

Across the four empirical studies in this thesis, a number of findings were reported which help shed light on the mechanisms and brain regions associated with false perceptions. Outcomes are identified and discussed which independently inform about the structure and function of brain regions involved in the processing of predicted sensory feedback (and prediction errors), sensitivity to voice signals, and changes in perceived voice identity related to the experience of hallucinations. Based on these findings, the involvement of regions and pathways previously associated with prediction, inhibition and salience may indicate a specific etiological cognitive mechanism underlying the emergence of false perceptions.

1 Cortical and cerebellar processing of (un)expected sensory input

The primary goal of the first study (**chapter 2. The role of the cerebellum in adaptation: ALE meta-analyses on sensory feedback error**) was to use the existing literature to determine which brain regions consistently activate when sensory feedback from one's own action is experimentally manipulated. Critically, the resultant activation pattern, representing the feedback-error response, conforms to regions putatively involved in self-monitoring models of hallucinations. The secondary and more explorative goal was to define potential modality-specific cerebellar regions responsive to manipulations of either visual or auditory feedback. However, no consensus regarding such regions was indicated by the respective ALE meta-analyses. None-the-less, considering that a cerebellar role in the adaptive processing cycle of sensorimotor control is well-established from other sources of data (Bastian, 2006; Caligiore et al., 2017; Doya, 2000; Doyon et al., 2009; Halsband & Lange, 2006; Hardwick et al., 2013; Krakauer et al., 2019; Schmuelof & Krakaeur, 2011; Schwartze & Kotz, 2013, 2016) (see **chapter 1. Introduction** section 1.1.3), this outcome was interpreted as a possible methodological shortcoming of fMRI paradigms that are commonly used to probe this system (see **chapter 2**). Tests of independence across the included studies - in terms of sensory modality and task design - revealed that those few studies which

did observe activation of the cerebellum were also methodologically similar. Cerebellar activity was more reliably probed by studies eliciting an adaptation or compensatory responses (e.g., for sustained manipulation of sensory feedback).

Cerebellar patients exhibit a behavioural impairment in motor adaptation tasks (Bastian, 2008, 2011; Manto et al., 2011; Therrien & Bastian, 2015). Moreover, schizophrenia patients often exhibit structural or functional cerebellar abnormalities (Ding et al., 2019; Koziol et al., 2014; Moberget et al., 2018). These irregularities are often linked to additional motor symptoms, yet a relationship has also been observed between the cerebellum and psychotic symptoms such as hallucinations (Abboud et al., 2017; Andreasen & Pierson, 2008; Martin & Albers, 1995; Picard et al., 2008; Schmahmann, 2000). Within the cognitive dysmetria model of psychosis, the cerebellum holds a central role, whereby an impairment in mental coordination of memory, attention, emotion, and motor activity may account for the broad range of symptom profiles within schizophrenia (Andreasen et al., 1998; Andreasen & Pierson, 2008; Cao & Cannon, 2019). Although this presents an apparent interrelation between cerebellar pathology, the sensorimotor system, and psychotic symptoms, direct evidence is currently sparse for the specific link between cerebellar changes and the cognitive mechanisms underlying AVH such as inner voice or acoustic imagery (Moberget & Ivry, 2019; Pinheiro et al., 2021). The particular point of dysfunction in the self-monitoring loops incorporating the cerebellar forward model must be established (Pinheiro et al., 2020a): are hallucinations due to deficient descending efference copy signaling from the cortex, comparison of reafferent signals for forward model predictions, or signaling back to the cortex for updating motor commands? In the final study of the dissertation (**chapter 5. Variability in white matter structure related to hallucination proneness**), two components of a cortico-cerebellar-cortical processing loop between the motor cortex and the cerebellar cortex were investigated. Applying a measure of structural connectivity (number of streamlines), a positive correlation with one's proneness for hallucinations from the left cerebellar cortex to the right motor cortex was found. This might indicate variability associated with the pathway that propagates error signals for adaptation in sensorimotor control.

In terms of cortical responses, the meta-analysis (**chapter 2**) indicated activation of the inferior frontal and premotor regions putatively involved in a network serving salience detection and action planning (Cai et al., et al., 2014; Chang et al., 2013; Power et al., 2011; Seeley, 2010). This may signify the detection of cues to inhibit and reorient action plans (Corbetta & Shulman, 2002; Hampshire et al., 2010; Levy & Wagner, 2011), via the engagement of an overlapping ventral attention system (Aron et al., 2003, 2004, 2014; Aron, 2007). Additionally, an observed increase in sensory cortex activation likely indicates the interruption of motor-induced suppression (MIS) due to the discrepancy between forward model (predicted) and reafferent (actual) feedback as outlined by the self-monitoring theory (Frith et al., 2000; Miall & Wolpert, 1996; Wolpert et al., 1995). MIS is present across various sensory modalities, resulting in the "cancelling out" of cortical responses to predicted self-generated stimuli. This phenomenon may be best illustrated via the often-cited observation that one cannot tickle oneself (Blakemore et al., 1998), or the attenuated response one has to one's own voice while speaking (Hickok et al., 2012; Houde & Nagarajan, 2011). Importantly, patients prone to hallucinate are more likely to express an impairment in MIS (Ford & Mathalon, 2012, 2019; Thakkar et al., 2021; van Lutterveld et al., 2011; Whitford, 2019). This mechanism is the likely cause for the externalization bias or misattribution of self-generated stimuli (Allen et al., 2007a, 2008; Brookwell et al., 2013; Cho & Wu, 2013; Damiani et al., 2022; Ditman & Kuperberg, 2005; Waters et al., 2012a,b; Zmigrod et al., 2016). In accordance with the self-monitoring theory, the cortical sensory processing regions reported in the meta-analysis are taken as likely candidates to show abnormally increased activity during action that leads to false perceptions. This interpretation is also in line with a set of functional neuroimaging studies which report increased activation in these areas during hallucinations (Allen et al., 2008; Bohlken et al., 2017; Jardri & Sommer, 2013; Upthegrove et al., 2016).

Another critical aspect was raised in the interpretation of the feedback-error meta-analysis: how does error-related activity in these regions relate to an inner speech model of AVH? Inner speech theory is built around the fundamental assumption that thoughts are a complex form of action (Ford & Mathalon, 2019; Jones & Fernyhough, 2007; Subramanian et al., 2019). The evolution of a cortical suppression mechanism not only allowed the brain to discern self-generated sensory input, but also an

exaptation trait whereby covert motor actions can lead to vivid imagery such as the voice inside your head without overt motor production (see recent arguments for the evolution and development of inner speech in Agnati et al., 2012, 2017, 2018; Alderson-Day & Fernyhough, 2015; Geva & Fernyhough, 2019; O'Connor, 2020; Perrone-Bertolotti et al., 2014; Vandervert, 2020). This may also explain the mechanistic and phenomenological link between hallucinations and the emergence of dreams as internally-generated signals perceived as sensory events (Feinberg, 2011; D'Agostino et al., Scarone 2012). Here, false perception and attribution to an external actor is due to misperceived inner speech. This is the result of a delayed, or absent, corollary discharge during inner speech and the ensuing (erroneous) co-activation of speech planning/production and perception regions (Allen et al., 2007a; Jones & Fernyhough, 2007a,b; Swiney & Sousa, 2014; Gregory, 2016). This framework converges with the traditional speech production-perception network, as top-down predictions are sent to both cortical and subcortical regions to facilitate MIS (Golfinopoulos et al., 2010; Guenther, 2006; Tourville & Guenter, 2011). However, inner speech models link the weak predictive signaling (corollary discharge) to the connection between speech planning and processing regions (cortico-cortical) and not to descending projections (cortico-subcortical) incorporating cerebellar error processing. In light of these divergent positions, interpreting the results from this meta-analysis (utilizing only data from overt sensory feedback paradigms) has to be done cautiously. Next to the cerebellar pathway results, The DTI study (**chapter 5**) specifically assessed the directionality of diffusion in the white matter pathways associated with (inner) speech theories of hallucinatory experience. These connections, from prefrontal to auditory cortex, correspond to the arcuate fasciculi (AF). A positive correlation with hallucination proneness (HP) was found in the anterior and posterior segments of the AF in the right hemisphere. This selective finding stands in contrast to inner speech theories of hallucinations which ascribe the putative prediction signaling to the left hemisphere and the sound-to-action mapping stream (Hickok & Poeppel, 2004, 2007; Rauschecker, 2011, 2018).

Informed by the results of the feedback-error meta-analysis (**chapter 2**), an fMRI experiment was conducted (**chapter 4. Expectancy changes the self-monitoring of voice identity**). This study was designed to further investigate the prefrontal (error-

monitoring) and temporal cortex (voice processing) responses to the externalization of self-generated auditory input. Using a novel paradigm, a factorial design assessing the main effects and interaction of source of feedback (self-generated or passively-heard) and voice identity (self, other, or uncertain) was applied. The self-generated voice was elicited via a button-press, to see if MIS during one's own voice would occur in the absence of direct input from Broca's area to the auditory cortex. Results indicated MIS in the right anterior STG voice identity ROI for own-voice but not for uncertain or other. The interpretation of this finding is not straightforward: how can a response to one's own voice be suppressed when there is no corollary discharge from voice production regions? However, first, it is essential to differentiate the MIS in this particular context from auditory cortex activity that is "cancelled out" while speaking. Instead, the initial MIS reported here is comparable to commonly applied manually-generated auditory signals, for example hearing an expected sound when typing on a keyboard or stroking a string on a guitar. Therefore, MIS observed in this study may reflect inadequate error signals from the subcortical cerebellar comparator system (i.e., a reduced sense of ownership over sounds), as opposed to any direct influence of Broca's area predictions sent directly to the STG. Second, the observed MIS modulation requires the interaction of inputs from coded predictability of voice features (Andics et al., 2013; Hoffman & Logothetis, 2009) and inputs signaling ownership over current auditory feedback via subcortical-cortical pathways (sensorimotor self-monitoring). Importantly, this requires mean-based coding to count own-voice as *more expected*, as one has the most experience with the qualities of their own voice. The decreased predictability in voice identity (other-voice perception) may negate the ownership that one infers from the sensorimotor feedback, resulting in the negation of an MIS response. A similar effect was observed in other studies in which the expectancy of manually-generated tones (Aliu et al., 2009; Baess et al., 2009; Knolle et al., 2013) or voices were modulated (Ford et al., 2007; Knolle et al., 2019; Pinheiro et al., 2018; Whitford et al., 2011). The majority of cortical response suppression research relies on EEG. This is the first time fMRI has demonstrated button-press MIS in voice processing, providing a higher level of spatial resolution regarding the specific structural correlates of self-attributed voice.

2 Cortical processing of voice and identity

Two studies included in this dissertation (**chapter 3. Hypersensitivity to passive voice hearing in hallucination proneness** and **chapter 4**) investigated functional brain responses in well-established regions of interest (ROI) forming part of the voice processing network. The reasoning behind this approach was two-fold: 1) to provide further evidence for specialized roles in subsections of the temporal voice perception network, and 2) to determine how particular regions may serve the emergence of false perceptions of AVH. The temporal voice areas (TVA) of the left and right superior temporal cortex compose the network exhibiting preferentially responses to voice (as compared to non-voice signals). Since the TVA localizer fMRI task was first established over 20 years ago (Belin et al., 2000), it has been widely applied - by an informal count in over 100 papers - in research involving healthy and pathological groups. It has proven a reliable tool that indicates bilateral patches consisting of separate anterior, middle, and posterior peak activations, and additional extra-temporal regions recruited in voice processing (Pernet et al., 2015). In both studies, the localizer task was applied to delineate ROIs at separate TVA peak activity regions. First, the correlation between subjective levels of HP in a non-clinical sample and TVA voice-preferential BOLD activations was investigated (**chapter 3**), where contrast estimates (voice and non-voice versus silence) were extracted at each location to calculate a voice preference response ([voice > silence] > [non-voice > silence]). As outlined in the previous section, an experiment using a novel fMRI task (**chapter 4**) was conducted to investigate how the TVA region susceptible to differential responses of voice identity would respond in a healthy sample to self-other attributed voice during action.

The findings pertaining to HP-related voice hypersensitivity (**chapter 3**) help to disentangle both the hierarchical nature of TVA processing as well as theoretical claims for the emergence of AVH. According to the self-monitoring theory of hallucinations, increased activation in voice perception regions during hallucinations is attributed to absent MIS and the subsequent misperception of inner speech (Frith et al., 2000; Jones & Fernyhough, 2007; Weiss & Heckers, 1999). In other words, voice perception cortical areas are active because they are perceiving voice, even though no corresponding sensory voice input is present. Additionally, it has been suggested that a general

hypersensitivity of these regions is what causes the hallucination in the first place (Deneve & Jardri, 2016; Friston, 2005a, 2012b; Fletcher & Frith, 2009). Here, a propensity for increased activation in regions processing bottom-up sensory signals incorrectly attributes salience and engages attention networks towards irrelevant stimuli. As abnormal perceptual experiences related to hallucinations occupy a continuum throughout the general population (Beavan et al., 2011; de Leede-Smith & Barkus, 2013; Johns, 2005; Johns et al., 2014; Larøi et al., 2012; Zhuo et al., 2019), it seems possible that a degree of covariance with TVA localizer data from the general population may be present. Therefore, caution should be taken when assessing voice-related activations within the general population. For instance, the subsequent study (**chapter 4**) utilized the same TVA localizer task to produce a specific ROI in the right anterior superior temporal gyrus (STG) as a mask for analysis of data from a novel voice identity fMRI task. Prior to conducting this experiment, the possible susceptibility of a HP-related covariance in the ROI (right anterior STG) selected for analysis was investigated and rejected.

The comparison between HP and TVA voice-preference activity (**chapter 3**) revealed a positive correlation in only the right posterior STG. The right hemisphere has been linked to speaker-related features in voice processing (Belin & Zatorre, 2003, Nakamura et al., 2001; von Kriegstein et al., 2003, von Kriegstein and Giraud, 2004). This includes the inference of voice identity, a multi-stage process whereby detection of the relevant low-level acoustic features is associated with posterior regions of the STG, with subsequent comparison to mean-based coded prototypes and recognition occurring in the middle and anterior regions, respectively (Maguinness et al., 2018). As elaborated earlier (see **chapter 1. Introduction** section 1.2.2), this is a hierarchical pathway (Belin et al., 1999; Warren et al., 2003, Warren et al., 2005a; Zhang et al., 2021). Notably, this hierarchical processing does not occur in isolation and is not purely serial in nature, as an interaction with visual and affective inputs is frequently reported (Ethofer et al., 2013; Davies-Thompson et al., 2019; Stevenage & Neil, 2014; Watson et al., 2014b). For example, visual information from faces is involved in the identity attribution of voice signals (Blank et al., 2011, 2014; Von Kriegstein et al., 2005; Watson et al., 2014a; Yovel & Belin, 2013). As this correlation was limited to only the right posterior STG involved in lower-level acoustic processing of paralinguistic voice processing, HP was not

applied as a covariate to data in the subsequent fMRI study on activations in the right anterior STG voice identity ROI (**chapter 4**). However, this study did report the differential processing in the voice identity region in response to self-other attribution during action versus when passively heard.

Taken together, these two findings indicate a relation of the TVA network to hallucinations: A) As the propensity for aberrant perceptual experience (i.e., HP) increases, the hierarchical processing of voice signals may be vulnerable at only early stages of voice feature detection in passively-heard sensory-input. B) Downstream regions of voice processing involved in decisions of internalization or externalization of voice are influenced by sensorimotor processing during self-generated feedback. Although this clarifies where TVA responses are directly affected by HP, it opens the door to two major complexities: can a vulnerability to passive sensory processing alone lead to hallucinations and, perhaps more importantly, how might passive and active models of hallucinations interact?

3 The nature of prediction (error) in the brain and alternative accounts for emergent hallucinations

In the introduction (**chapter 1**), background on the predominant sensorimotor theories associated with hallucinatory experience was provided. These perspectives are related to the multidisciplinary science of perceptual inference, where the interaction of top-down (predicted) and bottom-up (sensory) inputs can provide us with the best estimate of the sensory causes of our perception. This research is approached from the perspective of self-monitoring, where abnormal internal model processing of sensorimotor transformation is linked to the emergence of false perceptions. However, alternative theories have been proposed and have gained a substantial degree of traction in the past decades, in parallel to sensorimotor accounts of hallucinations. In this section, the current findings are reflected against competing accounts underlying the emergence of hallucinations. According to the first (primary concept), hallucinations are due to the insufficient top-down sensory predictions during action, resulting in the misattribution of self-generated sensation to an external source (see **chapter 1. Introduction** section 1.1.2). For example, this is outlined in sensorimotor control theories of self-monitoring (Feinberg, 1978; Frith, 1992; Frith & Done, 1998). The

second (alternate concept) describes hallucinations as the excessive top-down sensory predictions during passive perception. For example, this may be due to deficient updating of prior belief weighting (predictive coding: Fletcher & Frith, 2009; Friston, 2005a, 2012b) or deficient inhibition leading to overcounting of priors (circular inference: Deneve & Jardri, 2016; Jardri et al., 2016; Leptourgos et al., 2017). An important distinction between these two concepts is in what hallucinations truly represent. Are they the externalization of one's own action and thought, or misperceptions of meaningless input from the environment, respectively (Ford & Mathalon, 2019).

3.1 Alternative concept: Hallucinations as meaning in meaningless noise

In the early 2000s (overlapping with the development of sensorimotor control theory), a computational theory of perception outlined essential components and biologically plausible cortical substrates of the brain capable of supporting unconscious inference (see historical account in Friston, 2012a). Notably, the scale-free nature of these models has allowed for research at the micro-, meso-, and macro-levels to build a comprehensive hypothesis of this passive perceptual inference in the brain (Smith et al., 2021). Across a hierarchical cortical network, higher-level regions provide top-down predictions of lower-level bottom-up responses, which receive continuous driving inputs until conflict between expected and actual input is achieved (Friston, 2002, 2003). In addition to structural and functional evidence, predictive coding is supported by theoretical assumptions of synaptic physiology and psychophysical accounts (Friston, 2005a). Although this model was initially developed in the field of vision (Rao & Ballard, 1999), the general Bayesian inference mathematical framework on which it is based has since been expanded as a unified model for optimization processes in the brain (Knill & Pouget, 2004). Specifically in predictive coding, the error is precision-weighted, thereby minimizing uncertainty (the inverse of precision) through a process of belief updating (Feldman & Friston, 2010; Hohwy, 2014, 2016, 2017). It is in the failure to encode uncertainty (or inversely the precision of prior and sensory evidence) that resulting excessive weighting of priors is hypothesized by some to lead to false inference such as hallucinations (Adams et al., 2013a; Fletcher & Frith, 2009; Friston, 2005a, 2012b). In the analysis of TVA responses (**chapter 3**), an

increasing propensity for the engagement of voice processing regions during passively-heard stimuli perceived as voice appears congruent with this model. If cortical neurons are coded to expect increasing certainty in the cause of voice signals - one of the most salient environmental stimuli in our human environment - then increasing levels of HP may account for this hypersalient response.

The predictive coding framework has provided a strong explanation for how a system designed to predict the sensory causes of exteroceptive input may be dysfunctional in those who experience false perceptions. However, in the case of hallucinations it does not concretely explain how a percept can occur in the complete absence of external stimuli. Moreover, the formulation of this theory lacks a focus on the contribution of inhibitory connections necessary to balance activity across hierarchical processing operating in top-down, bottom-up, and lateral directions. To address these issues, an alternative Bayesian inference model was presented to describe how an imbalance in excitatory-inhibitory inputs may influence the strength of top-down priors responsible for the manifestation of hallucinations (Jardri & Deneve, 2013). Within this model, inhibitory loops are integrated to modulate excitatory connections and ensure reliable belief propagation. For example, if inhibitory interneurons are not effective, then top-down or bottom-up signaling will reverberate through a process of circular inference and inflate priors or sensory precision weighting, respectively. In this sense, alteration of descending loops will result in prior beliefs to be over-counted, and ultimately to "sense what we expect", i.e., in hallucinations (Deneve & Jardri, 2016). Importantly, as in predictive coding, circular inference can explain multiple symptoms of psychosis via a single Bayesian framework (Fletcher & Frith, 2009; Jardri & Deneve, 2013). Interneurons controlling inhibition via ascending pathways left unchecked may result in the opposite, to "expect what we sense" and increase the weighting of sensory input precision. Leptourgos et al. (2017) applying Marr's three levels of analysis, found support for inhibitory inputs into the belief propagation model at the computational, algorithmic, and neural level. Notably, at the neural level inhibitory loops might present not only at the local level subserved by interneurons, but also at the long-range level connecting sensory-motor, limbic system, and subcortical with cortical regions. Long-range inhibitory connections of the neocortex may be substrates for inhibitory control of feedback signals, i.e., requiring processing across distal regions.

In a report from the International Consortium on Hallucinations Research a wide range of supporting evidence for an inhibition abnormality was presented from molecular, pharmacological, neuroimaging, neurophysiological, neuromodulation, and cognitive research (reviewed in Jardri et al., 2016). Notably, they address the plurality of the term "inhibition" in neuroscience, and operationalize inhibitory differences associated with hallucinations to pertain to post-stimulus processes related to salience as opposed to automatic pre-stimulus or inference control. Finally, to distinguish biological substrates associated with false perceptions the authors suggest combining methodological (multiscale) approaches, and to isolate hallucinatory experience from pathology such as in non-clinical samples. The latter proposal may be of insight as both circular inference and predictive coding accounts suggest that abnormalities may exist over a continuum spanning normal and pathological states (Deneve & Jardri, 2016; Nazimek et al., 2012).

3.2 Comparing internal prediction models of perception

Although the theory of predictive coding has expanded beyond purely passive perception to incorporate active inference of self-generated feedback, this is not synonymous with theories pertaining to sensorimotor self-monitoring theories. This is due to an apparent inconsistency in *how* prediction functions in predictive coding versus self-monitoring, and therefore the mechanisms by which it is hypothesised to malfunction to produce false perceptions. Importantly, this does not mean the two are mutually exclusive.

The conflict between predictive coding and self-monitoring accounts of false perceptions is rooted deeply in the divergent ways that prediction is placed in the overall flow of information for perceptual processing (Adams et al., 2013b). Predictive coding considers a hierarchical flow of information along the direction from sensory input (low-level) to perceptual belief as an output (high-level) (detailed description of predictive coding microcircuits in Bastos et al., 2012; Huang & Rao, 2011; Shipp, 2016). Therefore, information is *fed-forward* from sensation (bottom-up) while higher-level layers signals are *fed-back* (top-down) to shift the accuracy of predictions in lower-level units. By viewing the network as a system driven to optimize accuracy, it can be said that the information moving along the forward/bottom-up direction is the propagation

of prediction error, while signals in the backwards/top-down direction are the adjustment of probabilistic representations (review of predictive coding in the auditory system in Carbajal & Malmierca, 2018; Heilbron & Chait, 2018). The prediction is therefore *coded-in* to the units across the network, creating a filter through which information regarding error passes.

Conversely, self-monitoring theories posit information flow as a loop between action and sensation (sensorimotor transformation) (Jordan & Rumelhart, 1992; Miall & Wolpert, 1996). As elaborated in the introduction (**chapter 1. Introduction** section 1.1.2), internalized models of sensorimotor transformations can be applied in the forward and inverse directions. In forward (internal) models, the motor command is the input selected to produce the desired sensory outcome, while inverse models hold sensation as the input to derive selection of the necessary motor command (Jordan, 1995; Kawato et al., 1987; Miall & Wolpert, 1996; Wolpert et al., 2001). The forward model in particular is essential for hypothesis testing or inference in self-generated sensation. During action, top-down predicted consequences of efferent motor commands (efference copies) are *fed-forward* for comparison with actual (bottom-up) sensory outcomes, resulting in error signals being fed-back to the cortex. Therefore, the main difference between predictive coding and forward modelling can be seen in the input necessary to begin the process of inference. The former requires only the stimulus, as the structure and weights of the connections through which information passes represent the predictions themselves, while the latter requires action for the prediction and reafferent feedback to be simultaneously engaged. As the prediction must be “sent-ahead”, the false perception must be the result of a weak or absent prediction signal where the sensory processing regions are unprepared for the coming stimuli.

If the predictive coding account of *strong predictions* in hallucinations extends to active inference, then these *weak predictions* of the forward model appear to be fully incongruent. Few researchers have attempted to reconcile this ‘strong versus weak priors' debate for the emergence of hallucinations. For example, they may both be present at different levels of hierarchical perceptual inference, affecting the phenomenological aspects of false perceptions and beliefs (Powers et al., 2016).

Recently, Corlett et al. (2019) have suggested that the existence of evidence for both strong and weak prior accounts indicate a process whereby sensorimotor self-monitoring and cortical predictive coding inference operate together as one integrated network. Within this model, abnormal (weak) forward model predictions result in the overreliance (strong priors) on expected causes of external sensory input (detailed schematics for system in Leptourgos & Corlett, 2020). Simply put, a feeling of agency over sensory feedback is gradually lost, and the brain determines the most likely cause of the external input, progressively becoming more and more certain. As proposed by Thakkar and colleagues (2021), the effects of these two interacting mechanisms are best illustrated by the phenomenological differences between clinical and non-treatment-seeking voice hearers. In healthy voice hearers, an ability to explain the false perception remains as a form of volitional control (e.g., shaping the hallucination into something beneficial or viewing their affliction as a meta-physical gift). This way, although self-monitoring impairments (weak or sensorimotor predictions) remain, they hold some form of control over forming the predictions of their cause. In a similar observation, healthy voice hearers often acknowledge the internal nature of the bottom-up false perception, due to the preserved capacity of prefrontal top-down inhibition (Hugdahl, 2009, 2015). Together, these propositions of interacting sensorimotor prediction errors, cortical predictions, and top-down control allow for an integrated account of false perceptions.

4 A unified account for salience and self-monitoring abnormalities in hallucinations

Findings regarding the regions and pathways functionally and structurally associated with the cognitive mechanisms underlying false perceptions are presented, as well as direct comparisons to a proneness for abnormal perceptual experience. Included are consistent findings on the functional role of the prefrontal cortex in error monitoring (**chapter 2** and **chapter 4**), as well as prefrontal-temporal connections serving salience and auditory perception (**chapter 5**). In the superior temporal gyrus, further consistent results are provided in the perceived externalization of the control (**chapter 2**) and identity of auditory stimuli (**chapter 4**). At first glance, it may appear that these findings are congruent with the fronto-temporal networks connecting speech production to

perception regions widely hypothesized to underlie AVH (Alderson-Day et al., 2015; Allen et al., 2007; Ćurčić-Blake et al., 2018; Frith & Done, 1998; Whitford et al., 2012). However, all results were reported in the right hemisphere, and therefore should remain separate from interpretations of (inner) speech models of AVH (i.e., direct influence of Broca's area on auditory cortex suppression). In line with the current findings, indicating cerebellar adaptation responses to feedback-error (**chapter 2**) and increased HP-related connectivity in the cerebello-cortical motor pathway (**chapter 5**), the findings associated with button-press MIS (right STG) for own voice (**chapter 4**) are suggested to involve a general self-monitoring process involving the cerebellar forward model system interacting with cortically coded expectations of stimulus features. Although this frames hallucinations as misattributed self-generated feedback, one particular finding - indicating HP-related functional brain changes without motor production (**chapter 3**) - opened the door to a wider interpretation. The above findings can arguably be integrated under a single framework, tied together by a string called salience.

The role of abnormal salience processing in false perceptions has been at the forefront of psychosis research for the last two decades. This focal point was primarily spotlighted by two parallel streams of research: A) psychopharmacological findings from the field of psychiatry, and B) functional brain network findings from the field of neuroimaging (detailed description of additional streams of evidence in Miyata, 2019). These theories have been particularly instrumental in describing etiological and pathophysiological mechanisms underlying schizophrenia. However, they also provide a framework by which transdiagnostic or non-clinical models of hallucinatory experience can be compared. In accordance with the psychosis continuum hypothesis and the search for possible common etiological factors underlying or interacting with this spectrum of perceptual experience, abnormal salience processing associated with psychotic experience are reviewed below. The aim is to offer an explanation of just how much non-clinical hallucination proneness may be influenced by the salience mechanisms affected in schizophrenia.

4.1 Salience: aetiology at the cellular level?

In terms of psychopharmacology (stream A), the aberrant salience hypothesis of psychosis can be credited to the proposal of a unitary framework underscored by the relationship between mesolimbic dopamine dysregulation and the mediation of salience (Kapur, 2003). This theory was founded on the observation that first generation antipsychotic medication acting as D2 receptor antagonists (e.g., chlorpromazine and haloperidol) are highly effective at alleviating positive symptoms via the dampening of salience processing for external and internal cues (Howes & Nour, 2016; Kapur, 2004; Kapur et al., 2005; Winton-Brown et al., 2014). Correspondingly, the administration of the dopamine agonist amphetamine can lead to psychotic experience similar to that in schizophrenia. Versions of this theory were presented as early as the 1970s when it was first discovered that these drugs operated via dopamine modulation (Carlsson, 1977, 1978, 2001; Meltzer & Stahl, 1976). In the decades since, a body of research across animal models, post-mortem, and in vivo human neuroimaging has corroborated the association between this pathway of action and the relationship to positive symptoms (detailed description of multi-modal findings in McCutcheon et al., 2020). Furthermore, the aberrant salience hypothesis is congruent with inner speech models of hallucinations (Barber et al., 2021), as well as theories of abnormal source monitoring in patients with positive symptoms of psychosis (Kowalski et al., 2021). Additionally, dopamine regulated salience is integral to inference models such as predictive coding for the assessment of epistemic affordance of sensory events (Parr & Friston, 2017), via the balancing of bottom-up sensory and top-down prior belief information (Friston et al., 2012c). Within this Bayesian inference framework, the influence of striatal dopamine regulation on prediction and error signaling underlies the link between inflated salience (strong prior) processing and hallucinatory experience (Howes et al., 2020; Kesby et al., 2021). This divergence has been extended to clinical high-risk groups (Howes et al., 2020), and at-risk mental states (Kowalski et al., 2021), suggesting an effect outside of the clinical range.

Although the link between dopamine, salience, and positive symptoms (such as hallucinations and delusions) has been established, it may not reflect a true etiological relationship. Theories regarding glutamate and serotonin circuits offer alternative and

integrated accounts for neurotransmitter imbalances associated with different (negative, cognitive, affective and other positive) symptoms of psychosis (Corlett et al., 2010; Johnsen et al., 2013; Rolland et al., 2014; Stahl, 2018). Perhaps the most prominent argument revolves around the interaction of glutamate and dopamine systems (early proposals in Coyle, 1996; Olney & Farber, 1995; Olney et al., 1999; Riederer et al., 1992; Tamminga, 1998). Within this glutamate account of psychosis, an upstream hypofunction of the glutamate receptor N-Methyl-D-aspartic acid (NMDA-R) on γ-Aminobutyric acid (GABA) interneurons results in the dopamine dysregulation underlying the aberrant salience hypothesis of psychosis (Howes et al., 2015; Ishimaru & Toru, 2012; Kehrer et al., 2008; Lewis & Sweet, 2009; McCutcheon et al., 2020; Moghaddam & Javitt, 2011; Stahl, 2007; Uno & Coyle, 2019). Abnormal functions of different glutamate pathways account for disparate symptomologies (detailed description of glutamate-GABA-dopamine circuits in Schwartz et al., 2012). For example, the emergence of positive symptoms involves the suboptimal function of NMDA receptors in the frontal lobe, resulting in an increased stimulation of the mesolimbic dopamine pathway by glutamatergic cortical-brainstem signals. Hugdahl and colleagues demonstrated a relationship between increasing levels of cortical (temporal and frontal lobe) glutamate with the increasing frequency and severity of AVH (2015). They proposed that NMDA-R dysfunction not only results in increased stimulation of the mesolimbic dopamine pathway, but also - due to the lack of inhibitory feedback - leaves an excess of glutamate in the temporal cortex imposing an excitatory effect. Glutamate, therefore, may mediate the imbalance of bottom-up/top-down processing associated with AVH at the level of neurotransmission (Hugdahl, 2009, 2017; Hugdahl et al., 2009; Hugdahl & Sommer, 2018), and provide an appropriate system to target for treatment (Sommer et al., 2018). The glutamate account of psychosis is in line with Bayesian inference computational models (see discussion section 3.1.), positioning the excitatory glutamate signaling and NMDA-R hypofunction as the neurochemical inputs underlying the excitatory-inhibitory imbalance leading to false perceptions (Corlett et al., 2011; Heinz et al, 2019; Jardri et al., 2016; Sterzer et al., 2018).

Abnormal glutamate levels may underlie an additional characteristic of schizophrenia: dysconnectivity of dispersed brain areas (Davis et al., 2003; Goff & Coyle, 2001; Mighdoll et al., 2015; Plitman et al., 2014; Suárez-Posos et al., 2020; Takahashi et al.,

2011; Walterfang et al., 2006, 2011). Due to the excitotoxic properties of glutamate (for detailed description see Lau & Tymianski, 2010), the excess of this neurotransmitter in the synaptic cleft due NMDA-R hypofunction has been linked to a breakdown of myelin along white matter pathways (Bakiri et al., 2009; Butt et al., 2014; Luttenbacher et al., 2022; Matute et al., 2007; Stys et al., 2007;). At a structural level, changes to white matter structure detected using DTI may be influenced by myelin thickness and axonal structure or density (Kubicki et al., 2002; O'Donnell & Westin, 2012; Soares et al., 2013). In schizophrenia, DTI studies often indicate a decrease in the directionality of diffusion (fractional anisotropy: FA) of white matter pathways (e.g., Fitzsimmons et al., 2013; Kubicki et al., 2005; Tamnes & Agartz, 2016). Interpretation of white matter changes associated with myelin breakdown and hallucinatory experience are, however, not straightforward.

First, FA data alone cannot confirm demyelination, as other axonal and glial changes may also contribute to the overall decrease in directionality (Alba-Ferrara & Erausquin, 2013; Alexander et al., 2007; Figley et al., 2021; Winklewski et al., 2018). Second, although schizophrenia patients who hallucinate show a lower FA than those who do not, and healthy controls, both positive and negative correlations have been found between FA and severity of positive symptoms (Bopp et al., 2017; Knöchel et al., 2012; Lee et al., 2013; Phillips et al., 2009; Rotarska-Jagiela et al., 2009; Seok et al., 2007; Szeszko et al., 2008; Whitford et al., 2010, 2014) and AVH severity (Bopp et al., 2017; Ćurčić-Blake et al., 2013; Mulert et al., 2012; Psomiades et al., 2016; Rotarska-Jagiela et al., 2009; Seok et al., 2007; Szeszko et al., 2008). Likewise, in the current sample of non-clinical individuals (**chapter 5**), increasing hallucination proneness was associated with higher FA. Third, an effect of antipsychotic medication has been reported in DTI studies on patients with schizophrenia (e.g., Leroux et al., 2017; Seok et al., 2007), indicating the need for caution when interpreting myelin changes in clinical samples. Fourth, although this comprehensive framework accounts for the mechanisms of hallucinatory experience at the level of neurotransmitters, macrostructure, cognition (salience) and perception, the etiological genetic factors for glutamate and NMDA-R dysfunction are specific to schizophrenia (detailed review in Karoutzou et al., 2007). For these reasons, caution is advised when assessing glutamate/NMDA-R salience theories with structural changes to white matter pathways of non-schizophrenia samples.

However, given the evidence reported by the current DTI study (**chapter 5**) indicating HP-related increased directionality, the data suggests the rejection of a common underlying (NMDA-R hypofunction) aetiology for the psychosis continuum ranging from normal experience to subclinical and pathological perceptual abnormalities. Instead, it may be useful to assess salience theories associated with false perceptions at the functional level, in terms of observable patterns in dopaminergic, electrophysiological, or BOLD brain activity. As hallucinations are transdiagnostic and occur in the non-clinical population, it is likely that cascading of various etiological factors in different subpopulations result in the same imbalance of mechanisms which influence perception.

4.2 Salience: an imbalance in functional brain networks?

At roughly the same time as the (stream A) salience theories from the field of psychopharmacology, Downar and colleagues (2000, 2001, 2002) performed studies investigating the role of functional brain networks in the processing of behaviourally salient information. The integration and synchrony (functional connectivity) between networks serving the active sensory processing and an abnormal relationship to default mode network (DMN) processing has been theorized (i.e., stream B) as a characteristic feature of schizophrenia (Buckner, 2022; Calhoun et al., 2009; Hunt et al., 2017; Karbasforoushan & Woodward, 2012; Williamson & Allman, 2012). Abnormal DMN engagement in schizophrenia has further been linked to both changes in dopamine signaling and other functional network activity such as those controlling salience (Hu et al., 2017). This salience network (SN) is essential in the transferring from task-negative to task-positive states (Seeley et al., 2007; Seeley, 2019). When sensory information requires a shift of attention, the engagement of the SN composed of anterior insular and anterior cingulate cortical regions will interrupt DMN processing to facilitate rapid involvement of the central executive network (CEN) and motor systems (Goulden et al., 2014; Menon & Uddin, 2010; Menon, 2011; Sridharan et al., 2008; Uddin, 2015). This triple network model is affected in psychosis, where erroneous activation of the SN triggers a switch from DMN to CEN, allowing for active sensory processing to occur during irrelevant stimuli such as sensory noise (Manoliu et al., 2013; Palaniyappan & Liddle, 2012). It has been suggested that the overactivity of the auditory

cortex may result in the perception of AVH (Hare, 2021; Northoff & Qin, 2011), due to a disconnection between frontal top-down and bottom-up sensory regions (Allen & Modinos, 2012; Petterson-Ye et al., 2011). This desynchrony between functional networks may therefore explain the phenomenon of hypersalient processing of inner speech and abnormal activation of speech processing regions at rest (Alderson-Day et al., 2015, 2016; Ćurčić-Blake et al., 2015; Northoff, 2014).

Although the link between functional connectivity and microstructure of structural pathways of the brain is not fully understood (Bullmore & Sporns, 2009; Friston, 2011b; Honey et al., 2009; Suárez et al., 2020; van den Heuvel & Pol, 2010), abnormal network structure-function associations have been reported in schizophrenia (Cocchi et al., 2014; Fitzsimmons et al., 2013; Hu et al., 2017; Skudlarski et al., 2010). This has been particularly evident for patients who experience AVH (Benetti et al., 2015; Kubera et al., 2019; Roes et al., 2020). Indeed, the disconnection hypothesis is a longstanding perspective for schizophrenia (Coyle et al., 2016; Friston & Frith, 1995; Pettersson-Yeo et al., 2011; Rubinov & Bullmore, 2022; Silverstein et al., 2016). In line with neurochemical salience accounts, disordered brain connectivity has been theorized to arise from abnormal regulation of NMDA-R regulated synaptic plasticity (Brennan et al., 2013; Stephan et al., 2006, 2009). Moreover, this is congruent with predictive coding in which abnormal NMDA-R involvement alters synaptic efficacy and the resulting alterations of gain in pyramidal cells lead to the computational impairments which bring forth false perceptions (Friston, 2016). Furthermore, it has been suggested that patterns of atypical functional network connectivity in schizophrenia may be neurodevelopmental (Fornito & Bullmore, 2015; Nath et al., 2021; Uhlhaas, 2013). According to one perspective of the psychosis continuum, if NMDA-R etiological factors are phenotypically expressed along a spectrum, then the detection of abnormal structure-function coupling or changes to functional network integration may be useful in identifying those at risk for pathology (Schmidt et al., 2015).

In the same vein as the glutamate/NMDA-R account for abnormal salience in schizophrenia (stream A), a functional network imbalance salience theory (stream B) could indeed link hallucinatory experience in schizophrenia to a genetic neurochemical aetiology. However, as genetics play only a partial role in both the emergence of false

perceptions and the risk of illness, the use of abnormal functional network integration alone may not be a strong marker for hereditary factors. Moreover, as hallucinations are also reported in other disorders and non-clinical groups, and HP in the general population recruits the same perceptual processes, it is possible that the same salience mechanisms of the TMN are responsible for a bias in sensory processing seen in all groups, regardless of underlying causes. For example, a positive correlation is reported between HP and the strength of BOLD activation preferential to passively heard voice sounds that may reflect hypersalient processing (**chapter 3**). Future research is encouraged to explore both TNM functional connectivity as well as dopamine concentration associated with increasing HP, as consistent with the aberrant salience hypothesis. Although this would not provide the etiological basis for dysregulated neurotransmitters associated with abnormal, or vivid, sensory experience in the general population, it may assist in differentiating the mechanisms fundamental to the continuum hypothesis of psychosis from those of schizophrenia, and provide a common neurochemical substrate for perceptual abnormalities.

5 Hallucination proneness and the psychosis continuum

Two empirical studies in the dissertation (**chapter 3** and **chapter 5**) included correlational analyses comparing brain function and structure to the LSHS measure of one's proneness for hallucinations. Interpretations of hallucinatory experience in non-clinical samples are theoretically rooted in a psychosis continuum spanning the entire (clinical and otherwise healthy) population. Although psychiatry and diagnostic tools have propagated a categorical approach in determining the presence of clinical disorders involving psychotic symptoms, it has been observed that similar experiences occur in those without mental illness across the lifespan (see meta-analyses in Healy et al., 2019; Kelleher et al., 2012; Linscott & van Os, 2013; Maijer et al., 2018; van Os et al., 2009). Despite initial population-based reports appearing in the late 19th century (reviewed in Le Malefan & Sommer, 2015; Sommer, 2011), it was not until roughly a century later when researchers began to suggest a dimensional or continuous nature of psychosis (Bentall, 1990; Chapman & Chapman, 1980; Claridge, 1994; Strauss, 1969). Arguably, an escalation in large community-based prevalence reports of

psychosis-like experience precipitated the beginning of a paradigm shift at the turn of the millennium (e.g., Eaton et al., 1991; Johns et al., 1998; Kendler et al., 1996; Ohayon, 2000; Tien, 1991; van Os et al., 2000). Likewise, scales were developed to measure variability in the prevalence or predisposition of healthy people to experience hallucinations (e.g., Barrett & Etheridge, 1992; Launay & Slade, 1981; Posey & Losch, 1983). Based on these findings, researchers urged for the disentangling of the dimensionality of these experiences in the general population (Johns & van Os, 2001; Myin-Germeys et al., 2003; Verdoux & van Os, 2002). For example, are hallucinatory experiences in healthy people different from those in psychosis? Are they a predisposition for psychotic illness? Or, do they exist along a continuum ranging from normal to pathological experience? (de Leede-Smith & Barkus, 2013). This *call to refocus*, in parallel with planning for the DSM-V, created the perfect storm for a field-wide debate on how a change from a categorical to a continuity perspective of psychosis may benefit (or deter) both research and clinical practice (reviewed in Allardyce, et al., 2007; David, 2010; Dutta et al., 2007; Esterberg & Compton, 2009; Kaymaz & van Os, 2010; Peralta & Cuesta, 2007; Sommer, 2010). For research into underlying risk (genetic or environmental) or etiological (cognitive or neurological) factors, the message was clear. One should treat the continuum as a null hypothesis and consider how one's sample and tool of measurement contributes to testing the model. In this regard, a discussion is provided below which highlights the bounds of the presented interpretations as well as possible limitations.

For the sake of clarity, the terminology used by David (2010) is applied outlining two forms of dimensionality associated with psychosis-like experiences including hallucinations: A type 1 continuum pertains to population-level distributions useful for determining correlated spectrum of risk (in terms of frequency of experience) or subpopulations more likely to progress to clinical levels of psychosis, while a type 2 continuum pertains to phenomenological-level variability which can be observed as a spectrum of severity. Beginning with an examination of type 1-implications in the current data, the exact section of this continuum that is sampled must be considered. In both **chapter 3** (n = 27) and **chapter 5** (n = 52), participants were recruited from the local university (Faculty of Psychology and Neuroscience, Maastricht University, the Netherlands). Most participants were undergraduate students, capturing only a limited

age range across the general population. Type 1 distributions are not fully one-dimensional (e.g., not determined by a single gene distributed across the population), and therefore random sampling from any given area or age group will not produce a representative sample of the psychosis continuum. The uneven distribution of environmental risk factors associated with psychosis throughout the population, such as urbanicity, ethnicity, or socio-economic status, in interaction with putative genetic risk factors may produce a quasi-dimensional distribution where subpopulations (with higher or lower risk) are grouped (Baumeister et al., 2017; DeRosse & Karlsgodt, 2015; Johns & van Os, 2001). Likewise, the effect of age in the continuum cannot be ignored. Children are more likely to report psychotic-like experiences including hallucinations as compared to adolescents (Kelleher et al., 2012; Maijer et al., 2018; Rubio et al., 2012). Moreover, post-adolescence decreases in both hallucination prevalence (Yates et al., 2021) and proneness (Thompson et al., 2021) continue across the lifespan. By sampling only post-adolescence – or post-developmental risk period – those of higher risk who sought clinical help may be eliminated from the community-based pool. It is apparent that the current sampling, in terms of demographics (e.g., low rates of male, urban, non-white) and age (predominately bachelor program university students), represent only a small, lower risk section of a type 1 population-level continuum. In fact, although this sample did include variability in a predisposition for hallucinations, it did not include any participants who reported chronic hallucinations. Therefore, results pertaining to a hypersensitivity of cortical voice processing (**chapter 3**) or in the increased directionality of white matter diffusion (**chapter 5**) should not be taken as psychosis risk markers indicated by increased frequency of reported experiences, as would be predicted by a type 1 continuum.

Instead, the current findings (**chapter 3** and **chapter 5**) are more in line with the type 2 psychosis continuum. In particular, these studies isolate a single experience associated with the multi-dimensionality of psychosis, hallucinations, which also appear to exist on a continuum (Beavan et al., 2011; de Leede-Smith & Barkus; Johns, 2005; Johns et al., 2014; Larøi et al., 2012; Zhuo et al., 2019). The application of the LSHS provides such a unidimensional (continuous) measurement of perceptual experiences (HP), as a composite of 16 items which range normal experience (e.g., vivid thought and daydreaming) to subclinical perceptual symptoms of psychosis (e.g., auditory and

visual hallucinations) using a 5-point Likert scale assessing "to what extent this statement applies to you" (Larøi & Van der Linden, 2005a). The scale is therefore bounded by two extremes: the absence of proneness and the presence of experiences similar to those in psychosis. It can be determined where one lies on this type 2 continuum by collecting measurements within individuals as opposed to type 1 which requires the measurement of a distribution across a population (Larøi, 2012). The LSHS thus provides a useful tool for correlational analyses in smaller sample sizes, for example, as often found in neuroimaging studies.

The interpretation of type 2 psychosis continua in community samples relies on a number of suppositions: 1) the same cognitive mechanisms underlie normal perceptual experience, unusual non-hallucinatory perceptions, and false perceptions, 2) an increase in unusual perceptions is associated with an increased proneness for true hallucinations, and 3) true hallucinations in healthy and clinical groups are the same thing. These assumptions have been subject to debate, particularly the latter, as phenomenological differences have been observed between hallucinations in non-clinical and clinical groups (Stip & Letourneau, 2009). For example, it has been suggested that this disparity may indicate subtypes of hallucinations rooted in distinct cognitive functions and related brain regions (Johns et al., 2014). However, false perceptions may arise via abnormal salience processing and subsequently be phenomenologically guided by associated cognitive mechanisms that contribute or bias predictions (e.g., traumatic memories or negative emotion) as well as in how the false perceptions are interpreted (e.g., unusual thought content). The guiding and interpretation of the false perceptions may provide the delineation between healthy hallucinators and those who seek (or require) help (Johns et al., 2014). Moreover, these factors may exist as overlapping type 2 continua (e.g., severity of perceptual abnormality versus unusual thought content) that interact to produce clinically relevant hallucinatory experience (Kelleher & Cannon, 2016). The LSHS does not incorporate unusual thought content items, and therefore, if such interacting continua exist, cannot be used to infer upon a proneness for clinical forms of hallucination. Instead, this measure may only be interpreted as a proneness for hallucinations in general, non-continuous with clinical symptomatology. However, follow-up questions were included for those who responded strongly (rating 3-5 out of 5) to each of the primary 16 items

which assessed secondary factors such as frequency, emotional valence, and level of control to stop or avoid the experiences that may assist in disentangling characteristics associated with seeking help and/or diagnosis. Unfortunately, a second limitation of the included studies applying LSHS measures – in addition to, or perhaps related to the homogeneous demographic characteristics mentioned above – is that only a small number of participants responded strongly enough to provide data on these secondary factors. In summary, the findings related to brain function (**chapter 3**) and structure (**chapter 5**) associated with HP should not be interpreted in terms of increased risk for psychosis as related to frequency of psychosis-like experience (type 1) or associated phenomenological factors that demarcate clinical-level manifestations of hallucinations (overlapping or interacting type 2). Instead, the interpretations of these findings relevant to the psychosis continuum should remain in terms of a progressive susceptibility (or bias) of normal cognitive mechanisms underlying perception to false perceptions as proneness to hallucination increases, in other words, of an experiential continuum.

Summary

The aim of the present doctoral dissertation was to extend current knowledge of the putative cognitive mechanisms and neural substrates associated with hallucination proneness. Hallucinations, or the experience of sensory stimuli in the absence of an associated external source, are phenomena often described as a symptom of psychotic disorders, such as schizophrenia. However, they are also seen in different disorders as well as otherwise healthy people. The prevalence across the population, comparable phenomenology, and common risk factors suggests a common aetiology underwriting affecting atypical brain function. It has been theorized that a spectrum of hallucination proneness contributes to a multidimensional psychosis continuum spanning clinical and non-clinical parts of the population. Although multiple patterns of brain activity, structure variability and associated cognitive mechanisms have emerged, there still is much uncertainty in the basic substrates explaining the presence of the many forms of hallucinations. Moreover, most of the existing research has been conducted in clinical samples, predominately in schizophrenia, making aberrations specific to hallucinatory experience difficult to conclude. Through various methodologies in magnetic resonance imaging data analysis, a comprehensive argument across four empirical studies is provided for neural substrates associated with basic mechanisms in theories of hallucination. Importantly, all data analysed across the dissertation has been acquired from non-clinical individuals of the general population.

In **Chapter 1**, a comprehensive review of the history of hallucination research is presented. This section outlines the three main perspective from which the hypotheses across the dissertation are based: 1) sensorimotor feedback processing as the predominate mechanism associated with hallucinatory experience, 2) the cerebellum as the primary structure serving the processing of predicted and unpredicted sensorimotor feedback, and 3) cortical voice processing, as hallucinations are most commonly reported as hearing voices. Additionally, a description is provided of the main methods applied in included research chapters: the Launay-Slade Hallucination Scale (LSHS), the temporal voice area (TVA) localizer task, a novel voice-elicitation task, and a general description of the physics and application of magnetic resonance imaging (MRI).

Chapter 2 provides a meta-analysis on functional neuroimaging research into the neural substrates of prediction error. Specifically, this data represents how the brain responds to unexpected sensory feedback from one's own action when manipulated by an external source. In line with feedback prediction theories of hallucinations, the parts of the brain where typical source attribution is present in the healthy brain were outlined. Multiple separate regions were identified to visual and auditory feedback modalities. Importantly, sensory cortices were more active when self-generated feedback was influenced by an external actor. This finding is congruent with previous hallucination theories, which suggest increased activity in these regions when feedback is incorrectly misattributed to another agent. In addition, although the cerebellum is theorized as a primary actor in this feedback processing system, functional neuroimaging paradigms may be incapable of detecting involvement of this region due to methodological limitations.

Chapter 3 investigated how increased sensory cortex activity may be related to subjective levels of hallucination proneness (collected via the LSHS) when hearing voice stimuli. Using a passive TVA localizer neuroimaging task, a positive correlation between proneness and activation strength was found in specific voice sensitive regions of the temporal cortex engaged in early auditory object processing. Although this finding does not negate propositions of hallucinations as the product of misattributed internally-generated signals such as inner voice and memories, it provides important insight on how a general hypersensitivity of these regions to external input is present.

In **Chapter 4**, a novel voice-elicitation paradigm was conducted to determine if feedback-error and voice identity regions of the brain are differentially as feedback diverges from the expected qualities of one's own voice. A feedback-error region of interest was created using coordinates from the **Chapter 3** meta-analysis study, while the voice identity region was chosen using the localizer task used also in **Chapter 4**. In the novel paradigm, voices were elicited via button-press and the quality ranged from one's own to the voice of another with merged samples at various iterations in between. Findings indicated that during action, the brain suppresses activity in response to one's own voice, but not for that of another person or when uncertain of the source.

Furthermore, as the voice diverges from one's voice, an inferior frontal feedback-error region may engage in monitoring unexpected voice quality. Importantly, the use of the button-press paradigm indicates that specialized processing of one's own voice during action is not contingent on vocal production.

The final empirical study included in **Chapter 5** analyses the relationship between subjective levels of hallucination proneness and the structure of white matter brain pathways. As proneness increases, the directionality of water diffusion rises in right hemisphere tracts and those connecting the halves of the brain. The reported right-hemisphere pathways have been linked with connecting brain regions and networks involved in the salience attribution and processing of sensory stimuli. These findings may signify a structural variability relating to underlying hypersalient processing of irrelevant sensory stimuli subserving hallucinations.

Discussed in **Chapter 6**, the findings are interpreted in comparison with alternative computation models of false perceptions, the theories of neurotransmitter imbalance often reported in schizophrenia explaining positive symptoms such as hallucinations, as well as a detailed exploration into the intricacies of interpreting the continuum of psychosis. In relation to the three perspectives delineated in the introduction, a set of conclusions can be drawn. First, cortical substrates implicated in self-monitoring theories of hallucinations are supported by this research. Premotor and sensory regions identified as feedback-error sensitive - including specific regions differentially responsive to self-other voice identity attribution - correspond to those underlying abnormal self-monitoring and hallucinatory experience. However, these findings refer only to central cognitive mechanisms and are not directly linked to observed false perceptions. Therefore, the cortical regions outlined across this research can be used in future research as regions of interest to delineate differential brain activity associated with the proneness or presence of hallucinations. Second, the current dissertation provides evidence for the role of the cerebellum in self-monitoring pathways associated with hallucinations. This region was implicated in the adaptation to sensorimotor feedback-error, and demonstrated structural changes in white matter tracts carrying putative error-signals to the cerebral cortex associated with increasing levels of hallucination proneness. Third, in terms of cortical voice processing regions, although

sensorimotor feedback influences the attribution of identity, no motor signals are required to elicit an early auditory object sensitivity for voice sounds linked to increased hallucination proneness.

In closing, hallucination proneness - as an indication of increasing abnormality in perceptual experience - is related to both functional and structural changes to the brain. These changes involve both systems incorporating the motor system as well as those that are purely perceptual. It is possible that an underlying abnormality in the attribution of salience to internally- and externally-generated signals biases perceptual processing, leading to a continuum across the population. The aim of the present doctoral dissertation project is to extend current knowledge of the putative cognitive mechanisms and neural substrates associated with hallucination proneness. Hallucinations, or the experience of sensory stimuli in the absence of an associated external source, are phenomena often described as a symptom of psychotic disorders, such as schizophrenia. However, they are also seen in different disorders as well as otherwise healthy people. The prevalence across the population, comparable phenomenology, and common risk factors suggests a common aetiology underwriting affecting atypical brain function. It has been theorized that a spectrum of hallucination proneness contributes to a multidimensional psychosis continuum spanning clinical and non-clinical parts of the population. Although multiple patterns of brain activity, structure variability and associated cognitive mechanisms have emerged, there still is much uncertainty in the basic substrates explaining the presence of the many forms of hallucinations. Moreover, most of the existing research has been conducted in clinical samples, predominately in schizophrenia, making aberrations specific to hallucinatory experience difficult to conclude. Through various methodologies in magnetic resonance imaging data analysis, we attempt to build a comprehensive argument across four empirical studies for neural substrates associated with basic mechanisms in theories of hallucination. Importantly, all data analysed across the dissertation has been acquired from non-clinical individuals of the general population.

Samenvatting

Het doel van dit proefschrift was het vergroten van de huidige kennis van de vermeende cognitieve mechanismen en neurale substraten geassocieerd met de neiging tot hallucinatie. Het zich voordoen van hallucinaties, of het ervaren van zintuiglijke stimuli in afwezigheid van een gelinkte externe bron, is een fenomeen dat vaak beschreven wordt als een symptoom van psychotische stoornissen zoals schizofrenie. Zij worden echter ook waargenomen bij andere stoornissen, en bij verder gezonde individuen. De prevalentie van hallucinaties in de gehele bevolking, vergelijkbare fenomenologie, en gemeenschappelijke risicofactoren suggereren een gemeenschappelijke etiologie die de atypische hersenfunctie beïnvloedt. Er is getheoretiseerd dat een spectrum van hallucinatie-gevoeligheid bijdraagt tot een multidimensioneel psychose-continuüm dat klinische en niet-klinische delen van de bevolking omvat. Hoewel verschillende patronen van hersenactiviteit, structuurvariabiliteit en geassocieerde cognitieve mechanismen naar voren zijn gekomen, is er nog steeds veel onduidelijkheid over de basissubstraten die de aanwezigheid van de vele vormen van hallucinaties verklaren. Bovendien is het meeste bestaande onderzoek uitgevoerd in klinische steekproeven, voornamelijk bij individuen met schizofrenie, waardoor afwijkingen die specifiek zijn aan een hallucinerende ervaring moeilijk te isoleren zijn. Door middel van verschillende methodologieën in magnetische resonantie beeldvorming data analyse, wordt een uitgebreid betoog over vier empirische studies geleverd voor neurale substraten geassocieerd met basismechanismen in theorieën van hallucinatie. Belangrijk is dat alle geanalyseerde data in het proefschrift verkregen zijn van niet-klinische personen uit de algemene bevolking.

In **hoofdstuk 1** wordt een uitgebreid overzicht gegeven van de geschiedenis van het wetenschappelijk onderzoek naar hallucinatie. Dit hoofdstuk schetst de drie perspectieven waarop de hypothesen in het proefschrift zijn gebaseerd: 1) sensorimotorische feedbackverwerking als het overheersende mechanisme geassocieerd met een hallucinerende ervaring, 2) het cerebellum als de primaire structuur die dient voor de verwerking van verwachte en onverwachte sensorimotorische feedback, en 3) corticale stemverwerking, aangezien hallucinaties

het vaakst worden gerapporteerd als het horen van stemmen. Daarnaast wordt een beschrijving gegeven van de belangrijkste methoden die in de onderzoekshoofdstukken zijn toegepast: de Launay-Slade Hallucination Scale (LSHS), de temporal voice area (TVA) localisatietaak, een nieuwe stem-elicitatie taak, en een algemene beschrijving van de fysica en de toepassing van magnetische resonantie imaging (MRI).

Hoofdstuk 2 bevat een meta-analyse van het functioneel neuroimaging onderzoek naar de neurale substraten van voorspellingsfouten. Deze gegevens geven weer hoe de hersenen reageren op onverwachte sensorische feedback van de eigen actie wanneer deze wordt gemanipuleerd door een externe bron. In lijn met feedback voorspellingstheorieën van hallucinaties werden de delen van de hersenen waar typische bronattributie aanwezig is in de gezonde hersenen geschetst. Meerdere afzonderlijke gebieden werden geïdentificeerd voor visuele en auditieve feedback modaliteiten. Belangrijk was dat de sensorische cortex actiever was wanneer zelf-gegenereerde feedback werd beïnvloed door een externe actor. Deze bevinding is congruent met eerdere hallucinatietheorieën, die een verhoogde activiteit in deze regio's suggereren wanneer feedback ten onrechte verkeerd wordt toegeschreven aan een andere actor. Hoewel het is getheoretiseerd dat het cerebellum een primaire actor is in dit feedback verwerkingssysteem zijn functionele neuroimaging paradigma's niet in staat om de betrokkenheid van deze regio te detecteren als gevolg van methodologische beperkingen.

In **hoofdstuk 3** wordt onderzocht hoe een verhoogde activiteit van de sensorische cortex kan samenhangen met subjectieve niveaus van hallucinatie-gevoeligheid (verzameld via de LSHS) bij het horen van stemstimuli. Met behulp van een passieve TVA localizer neuroimaging taak werd een positieve correlatie gevonden tussen neiging tot hallucinatie en de activatiesterkte in specifieke, stem-gevoelige gebieden van de temporale cortex die betrokken zijn bij de vroege verwerking van auditieve objecten. Hoewel deze bevinding niet ontkracht dat hallucinaties het product zijn van verkeerd toegeschreven intern gegenereerde signalen, zoals een innerlijke stem en herinneringen, geeft het een belangrijk inzicht in een algemene overgevoeligheid van deze regio's voor externe input.

In **Hoofdstuk 4** werd een nieuw stem-elicitatie paradigma uitgevoerd om te bepalen of feedback-error en stem-identiteit regio's van de hersenen differentieel zijn als feedback afwijkt van de verwachte kwaliteiten van iemands eigen stem. Een feedback-error gebied van belang werd gecreëerd met behulp van coördinaten uit de meta-analyse van **hoofdstuk 3**, terwijl het stemidentiteitsgebied werd gekozen met behulp van de localisatietaak die ook in **hoofdstuk 4** werd gebruikt. In het nieuwe paradigma werden stemmen opgewekt door op knoppen te drukken; de stemmen varieerden van de eigen stem tot de stem van een ander, met daartussen samengevoegde samples in verschillende iteraties. Uit de bevindingen bleek dat de hersenen tijdens actie de activiteit onderdrukken als reactie op de eigen stem, maar niet als reactie op de stem van een andere persoon of wanneer men niet zeker is van de bron. Bovendien, als de stem afwijkt van de eigen stem kan een inferieure frontale feedback-error regio zich bezighouden met het controleren van onverwachte stemkwaliteit. Belangrijk is dat het gebruik van het knop-druk paradigma aangeeft dat gespecialiseerde verwerking van de eigen stem tijdens actie niet afhankelijk is van vocale productie.

De laatste empirische studie in **Hoofdstuk 5** analyseert de relatie tussen subjectieve niveaus van hallucinatie-gevoeligheid en de structuur van witte stof hersenbanen. Naarmate de gevoeligheid voor hallucinatie toeneemt, neemt de richtingsgevoeligheid van de waterdiffusie toe in de rechterhersenhelft en in de banen die de hersenhelften met elkaar verbinden. De gerapporteerde banen van de rechterhersenhelft zijn in verband gebracht met het verbinden van hersengebieden en netwerken die betrokken zijn bij de opmerkzaamheid en verwerking van zintuiglijke stimuli. Deze bevindingen kunnen wijzen op een structurele variabiliteit met betrekking tot onderliggende hypersalient verwerking van irrelevante zintuiglijke stimuli die hallucinaties veroorzaken.

In **hoofdstuk 6** worden de bevindingen geïnterpreteerd in vergelijking met alternatieve rekenmodellen van valse waarnemingen, de theorieën van neurotransmitter onevenwichtigheid die vaak bij schizofrenie worden gerapporteerd en die positieve symptomen zoals hallucinaties verklaren, alsmede een gedetailleerde verkenning van de fijne kneepjes van het interpreteren van het continuüm van psychose. Met

betrekking tot de drie perspectieven die in de inleiding werden geschetst, kunnen een aantal conclusies worden getrokken.

Ten eerste, corticale substraten die betrokken zijn bij zelf-controlerende theorieën van hallucinaties worden door dit onderzoek ondersteund. Pre-motorische en sensorische regio's die gevoelig zijn voor terugkoppelingsfouten - inclusief specifieke regio's die differentieel reageren op zelf-ander stem identiteitsattributie - komen overeen met de regio's die ten grondslag liggen aan abnormale zelf-monitoring en hallucinerende ervaringen. Deze bevindingen hebben echter alleen betrekking op centrale cognitieve mechanismen en zijn niet direct gekoppeld aan waargenomen valse percepties. Daarom kunnen de corticale regio's die in dit onderzoek geschetst worden in toekomstig onderzoek gebruikt worden als regio's van belang om differentiële hersenactiviteit af te bakenen die geassocieerd wordt met de vatbaarheid of aanwezigheid van hallucinaties. Ten tweede levert het huidige proefschrift bewijs voor de rol van het cerebellum in zelf-controlerende paden geassocieerd met hallucinaties. Dit gebied werd betrokken bij de aanpassing van sensorimotorische feedback-fouten, en toonde structurele veranderingen in witte stof traktaten die vermeende fout-signalen naar de cerebrale cortex stuurden, geassocieerd met toenemende niveaus van hallucinatie vatbaarheid. Ten derde, in termen van corticale stemverwerkingsregio's, hoewel sensorimotorische feedback de toekenning van identiteit beïnvloedt, zijn er geen motorische signalen nodig om een vroege auditieve objectgevoeligheid op te wekken voor stemgeluiden gekoppeld aan een verhoogde neiging tot hallucinatie.

Tot slot, hallucinatie-gevoeligheid - als een indicatie van toenemende abnormaliteit in perceptuele ervaring - is gerelateerd aan zowel functionele als structurele veranderingen in de hersenen. Deze veranderingen betreffen zowel systemen die het motorische systeem bevatten als systemen die zuiver perceptueel zijn. Het is mogelijk dat een onderliggende afwijking in de toeschrijving van opvallendheid aan intern- en extern-gegenereerde signalen de perceptuele verwerking vertekent, wat leidt tot een continuüm in de populatie.

Het doel van dit doctoraatsproject is het vergroten van de huidige kennis van de vermeende cognitieve mechanismen en neurale substraten geassocieerd met de neiging tot hallucinatie. Het zich voordoen van hallucinaties, of de ervaring van

zintuiglijke stimuli in afwezigheid van een gelinkte externe bron, is een fenomeen dat vaak beschreven wordt als een symptoom van psychotische stoornissen zoals schizofrenie. Zij worden echter ook waargenomen bij andere stoornissen en bij verder gezonde individuen. De prevalentie van hallucinaties in de gehele bevolking, vergelijkbare fenomenologie, en gemeenschappelijke risicofactoren suggereren een gemeenschappelijke etiologie die de atypische hersenfunctie beïnvloedt. Er is getheoretiseerd dat een spectrum van hallucinatie-gevoeligheid bijdraagt tot een multidimensioneel psychose-continuüm dat klinische en niet-klinische delen van de bevolking omvat. Hoewel verschillende patronen van hersenactiviteit, structuurvariabiliteit en geassocieerde cognitieve mechanismen naar voren zijn gekomen, is er nog steeds veel onduidelijkheid over de basissubstraten die de aanwezigheid van de vele vormen van hallucinaties verklaren. Bovendien is het meeste bestaande onderzoek uitgevoerd in klinische steekproeven, voornamelijk bij individuen met schizofrenie, waardoor afwijkingen die specifiek zijn aan een hallucinerende ervaring moeilijk te isoleren zijn. Door middel van verschillende methodologieën in magnetische resonantie beeldvorming dataanalyse proberen we een uitgebreid argument op te bouwen over vier empirische studies naar neurale substraten geassocieerd met basismechanismen in theorieën van hallucinatie. Belangrijk is dat alle geanalyseerde gegevens uit dit proefschrift afkomstig zijn van niet-klinische personen uit de algemene bevolking.

Impact Paragraph

Numerous findings reported in this dissertation could offer impactful theoretical or practical contributions to future empirical research, possibly extending to interventions and cross-diagnostics pertaining to a psychosis continuum. Moreover, the included examinations incorporating a spectrum of hallucination proneness may provide a broader value to society itself, in terms of how an advanced knowledge of a psychosis continuum may improve the lives of those affected.

Potential scientific impact. The outcomes suggest both short- and long-term impacts to neuroimaging and translational cognitive neuroscience. Already, three methodological findings may be utilized. In **chapter 3,** findings from a widely-used voice perception task indicate a general variability of brain activations across the general population (Pernet et al., 2015). In healthy participants, a hypersensitivity was indicated in the brain region associated with early stages of voice processing, related to an increasing proneness for abnormal perceptual experience. It would therefore be of interest to studies investigating this region to consider how a general variability in response strength may be distributed in their sample, and how this may affect downstream processing of voice. In **chapter 4**, a behavioural task was the first to show that participants are able to correctly identify their own voice when hearing brief half-second vowel sounds. In theory, this implies that such short, simple, stimuli contain sufficient information for recognizing self-voice identity. In the same study, the first functional magnetic resonance imaging (MRI) evidence for a specific pattern of brain activity associated with self-generated, self-attributed, voice was demonstrated when participants played a sound by pressing a button. Therefore, investigators interested in brain responses to varying expectancy of voice features during action, and not speech itself, can adopt a button-press paradigm avoiding vocal production and reduce the associated head movement which lead to difficulties in MRI processing and analysis (Havsteen et al., 2017; Makowski et al., 2019; Zaitsev et al., 2015). Although these methodological reports are innovative, the lack of comparable findings in the literature implies that replication is necessary before accepting the generalizability of these interpretations.

The current dissertation offers longer-term implications to the scientific field. The meta-analysis in **chapter 2** may provide a farther-reaching, lasting, impact. The brain areas indicated as responsive to unexpected errors in sensory feedback suggest a network of motor and sensory regions with different roles in adaptation motor control. This was intended to isolate brain regions relevant to the mechanisms serving hallucinations. However, due the fundamental nature of this investigation, it was presented as a poster at an established conference (Society for Neuroscience, 2018) within the motor control section. Likewise, it was shared among broader circles, and has resulted in numerous citations, which foreseeably will continue to inform any future research into the brain correlates of sensory feedback and adaptive processes.

Two studies will predictably lead to debate, or possibly a shift in understanding, regarding predominant theories of hallucinations. The abovementioned **chapter 3** finding – of a hypersensitivity to voice sounds – views hallucinations as misperceived externally-produced sensations. This appears to conflict with theories positing hallucinations as misattributed, externalized, internally-produced signals (Ford & Mathalon, 2005; Heinks-Maldonado et al., 2007; van Lutterveld et al., 2011; Whitford et al., 2012). However, as suggested in the **chapter 6** discussion, these positions may not be mutually exclusive, reflecting a general abnormality in salience processing. It is anticipated that future research will determine the contribution of both internally- and externally-generated signals to overactive brain regions serving perception and the emergence of false perceptions. Although schizophrenia research has provided plausible neurochemical aetiology for this salience deficit (Corlett et al., 2011; Heinz et al, 2019; Jardri et al., 2016; Karoutzou et al., 2007; Sterzer et al., 2018), the underlying source of these abnormalities related to false perceptions across the general population remains to be determined. Deeper understanding of the function or imbalance of these inputs may help describe neurochemically how these experiences manifest in the healthy brain and establish an etiological or development distinction specific to schizophrenia.

In **chapter 5**, analyses on the structural pathways connecting brain regions showed a pattern opposite to many reported in schizophrenia compared to healthy participants (e.g., Fitzsimmons et al., 2013; Kubicki et al., 2005; Tamnes & Agartz, 2016). As

hallucinations are a main symptom of schizophrenia, this finding pertaining to hallucination proneness also appears to conflict with contemporary disconnection theories (Coyle et al., 2016; Friston & Frith, 1995; Pettersson-Yeo et al., 2011; Rubinov & Bullmore, 2022; Silverstein et al., 2016). However, there has been a lack of consensus across clinical samples, and the report of effects in both directions when assessing symptom severity, for example, in hallucinated voices (e.g., Bopp et al., 2017; Ćurčić-Blake et al., 2013; Mulert et al., 2012; Psomiades et al., 2016; Rotarska-Jagiela et al., 2009; Seok et al., 2007; Szeszko et al., 2008). It has been suggested that research using non-clinical groups may be of particular help in assessing the underlying cognitive functions and related brain areas associated with symptoms of psychosis. For example, investigations into white matter structure related to hallucinations may be difficult in schizophrenia samples, as changes to these pathways are also susceptible to effects of antipsychotic medication (e.g., Leroux et al., 2017; Seok et al., 2007). Additionally, white matter research may help inform the aforementioned neurochemical associations to false perceptions, and differences between the schizophrenic and healthy brain prone to hallucinations, as these pathways are structurally affected by imbalances in the neurotransmitters implicated in psychotic symptoms (Davis et al., 2003; Goff & Coyle, 2001; Mighdoll et al., 2015; Plitman et al., 2014; Suárez-Posos et al., 2020; Takahashi et al., 2011; Walterfang et al., 2006, 2011).

Potential social impact. A continuum theory of psychosis provides more than theoretical discussion among researchers, as it offers a vital perspective for how variability in these experiences can be perceived within society. All over the world, a proneness for abnormal perceptual experience such as hallucinations has been reported across large population-based samples (e.g., Moseley et al., 2021; Siddi et al., 2019). Likewise, at a higher level of that spectrum, experiences similar to symptoms of psychosis occur at various frequencies in the general population (e.g., Bitta et al., 2022; Bourgin et al., 2020; McGrath et al., 2015; Subramaniam et al., 2016). Finally, some subgroups of the general population experience chronic psychosis-like experience without the need for care or clinical diagnoses (e.g., healthy hallucinators) (Baumeister et al., 2017; Johns, 2014; van Os et al., 2009). Although different cultures provide varying explanations for the source of these experiences (e.g., religious or spiritual) (Larøi et al., 2014; van Elk & Aleman, 2017), it is evident that they are present in the

lives of many different people at many different levels, existing on a continuum with normal human experience. The findings in **chapter 3 and 5** indicate a correlation of brain function and structure with hallucination proneness in healthy people, supporting this theory. However, a dichotomous, categorical, perspective has led to much stigma, further impairing the mental health and help-seeking behaviour or those affected (Corrigan et al., 2017; Lien et al., 2015; Peter et al., 2021). Therefore, this dissertation may contribute to a general shift in perspective to a continuum, allowing for an understanding that abnormal perceptual experiences are nothing to hide or scorn, and allow those who have them to feel more comfortable in sharing with others, including clinicians, when requiring care.

Curriculum Vitae

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Output

Johnson, J. F., Belyk, M., Schwartze, M., Pinheiro, A. P., & Kotz, S. A. (2022). Hypersensitivity to passive voice hearing in hallucination proneness. *Frontiers in Human Neuroscience, 16:859731.* http://doi.org/10.3389/fnhum.2022.859731

Johnson, J. F., Schwartze, M., Belyk, M., Pinheiro, A. P., & Kotz, S. A. (2021). Variability in white matter structure relates to hallucination proneness. *bioRxiv*. https://doi.org/10.1101/2021.06.10.447901 [In revision with Neuroimage: Clinical]

Pinheiro, A. P., **Johnson, J. F.**, Amorim, M., Roberto, M., Schwartze, M., Kotz, S. A., & Shenton, M. E. (2021). The cerebellum links to positive symptoms of psychosis: A systematic review and meta-analysis. *Schizophrenia Bulletin Open, 2*(1), sgab039. https://doi.org/10.1093/schizbullopen/sgab039

Johnson, J. F., Belyk, M., Schwartze, M., Pinheiro, A. P., & Kotz, S. A. (2021). Expectancy changes the self-monitoring of voice identity. *European Journal of Neuroscience, 53*(8), 2681-2695. https://doi.org/10.1111/ejn.15162

Johnson, J. F., Belyk, M., Schwartze, M., Pinheiro, A. P., & Kotz, S. A. (2019). The role of the cerebellum in adaptation: ALE meta-analyses on sensory feedback error. *Human Brain Mapping, 40*(13), 3966-3981. https://doi.org/10.1002/hbm.24681

Johnson, J. F., Belyk, M., Schwartze, M., Pinheiro, A. P., & Kotz, S. A. (2018, November 7). An activation likelihood estimation (ALE) meta-analysis of sensorimotor feedback manipulations [Poster presentation]. Society for Neuroscience, San Diego, CA, United States. https://www.sfn.org/-/media/SfN/Documents/NEW-SfN/Meetings/Neuroscience-2018/Abstracts/Neuroscience-2018-Abstracts/SFN18_Abstract-PDFs---Posters_Wed_5_PM--revised.pdf

Belyk, M., **Johnson, J. F.**, & Kotz, S. A. (2018). Poor neuro-motor tuning of the human larynx: a comparison of sung and whistled pitch imitation. *Royal Society open science, 5*(4), 171544. https://doi.org/10.1098/rsos.171544

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References

Abboud, R., Noronha, C., & Diwadkar, V. A. (2017). Motor system dysfunction in the schizophrenia diathesis: neural systems to neurotransmitters. *European Psychiatry, 44*, 125-133. http://doi.org/10.1016/j.eurpsy.2017.04.004

Abbs, J. H., & **Gracco, V. L.** (1984). Control of complex motor gestures: Orofacial muscle responses to load perturbations of lip during speech. *Journal of Neurophysiology, 51*(4), 705–723. https://doi.org/10.1152/jn.1984.51.4.705

Adams, R. A., Shipp, S., & Friston, K. J. (2013a). Predictions not commands: active inference in the motor system. *Brain Structure and Function, 218*(3), 611-643. https://doi.org/10.1007/s00429-012-0475-5

Adams, R. A., Stephan, K. E., Brown, H. R., Frith, C. D., & Friston, K. J. (2013b). The computational anatomy of psychosis. *Frontiers in psychiatry, 4*, 47. https://doi.org/10.3389/fpsyt.2013.00047

Adank, P. (2012). The neural bases of difficult speech comprehension and speech production: Two Activation Likelihood Estimation (ALE) meta-analyses. *Brain and language, 122*(1), 42-54. https://doi.org/10.1016/j.bandl.2012.04.014

Agnati, L. F., Barlow, P., Ghidoni, R., Borroto-Escuela, D. O., Guidolin, D., & Fuxe, K. (2012). Possible genetic and epigenetic links between human inner speech, schizophrenia and altruism. *Brain research, 1476*, 38-57. https://doi.org/10.1016/j.brainres.2012.02.074

Agnati, L. F., Marcoli, M., Agnati, U., Ferraro, L., Guidolin, D., & Maura, G. (2017). The mis-exaptation of the prediction capability of humans and emergence of intolerant religious beliefs. Neurology, *Psychiatry and Brain Research, 23*, 43-53. https://doi.org/10.1016/j.npbr.2016.12.002

Agnati, L. F., Guidolin, D., Marcoli, M., & Maura, G. (2018). Inner speech mis-exaptation can cause the "Hubris" that speeds up ecosystem over-exploitation. *Neurology, Psychiatry and Brain Research, 30*, 62-73. https://doi.org/10.1016/j.npbr.2018.05.006

Akkal, D., Dum, R. P., & Strick, P. L. (2007). Supplementary motor area and presupplementary motor area: Targets of basal ganglia and cerebellar output. *Journal of Neuroscience, 27*(40), 10659–10673. https://doi.org/10.1523/JNEUROSCI.3134-07.2007

Alba-Ferrara, L., De Erausquin, G. A., Hirnstein, M., Weis, S., & Hausmann, M. (2013). Emotional prosody modulates attention in schizophrenia patients with hallucinations. *Frontiers in human neuroscience, 7*, 59. https://doi.org/10.3389/fnhum.2013.00059

Alba-Ferrara, L. M., & **de Erausquin, G. A.** (2013). What does anisotropy measure? Insights from increased and decreased anisotropy in selective fiber tracts in schizophrenia. *Frontiers in integrative neuroscience, 7*, 9. https://doi.org/10.3389/fnint.2013.00009

Alderson-Day, B., McCarthy-Jones, S., Bedford, S., Collins, H., Dunne, H., Rooke, C., & Fernyhough, C. (2014). Shot through with voices: Dissociation mediates the relationship between varieties of inner speech and auditory hallucination proneness. *Consciousness and Cognition,* 27, 288-296. https://doi.org/10.1016/j.concog.2014.05.010

Alderson-Day, B., & **Fernyhough, C.** (2015). Inner speech: development, cognitive functions, phenomenology, and neurobiology. *Psychological bulletin, 141*(5), 931. https://doi.org/10.1037/bul0000021

Alderson-Day, B., McCarthy-Jones, S., & Fernyhough, C. (2015). Hearing voices in the resting brain: a review of intrinsic functional connectivity research on auditory verbal hallucinations. *Neuroscience & Biobehavioral Reviews, 55*, 78-87. https://doi.org/10.1016/j.neubiorev.2015.04.016

Alderson-Day, B., Diederen, K., Fernyhough, C., Ford, J. M., Horga, G., Margulies, D. S., ... & Jardri, R. (2016). Auditory hallucinations and the brain's resting-state networks: findings and methodological observations. *Schizophrenia bulletin, 42*(5), 1110-1123. https://doi.org/10.1093/schbul/sbw078

Alderson-Day, B., Woods, A., Moseley, P., Common, S., Deamer, F., Dodgson, G., & Fernyhough, C. (2021). Voice-hearing and personification: characterizing social qualities of auditory verbal hallucinations in early psychosis. *Schizophrenia bulletin, 47*(1), 228-236. https://doi.org/10.1093/schbul/sbaa095

Aleman, A., Nieuwenstein, M. R., Böcker, K. B., & De Haan, E. H. (2001). Multi-dimensionality of hallucinatory predisposition: factor structure of the Launay–Slade Hallucination Scale in a normal sample. *Personality and Individual Differences, 30*(2), 287-292. https://doi.org/10.1016/S0191-8869(00)00045-3

Alexander, A. L., Lee, J. E., Lazar, M., & Field, A. S. (2007). Diffusion tensor imaging of the brain. *Neurotherapeutics, 4*(3), 316-329. https://doi.org/10.1016/j.nurt.2007.05.011

Aglieri, V., Chaminade, T., Takerkart, S., & Belin, P. (2018). Functional connectivity within the voice perception network and its behavioural relevance. *NeuroImage, 183*, 356-365. https://doi.org/10.1016/j.neuroimage.2018.08.011

Aliu, S. O., Houde, J. F., & Nagarajan, S. S. (2009). Motor-induced suppression of the auditory cortex. *Journal of Cognitive Neuroscience, 21*(4), 791-802. https://doi.org/10.1162/jocn.2009.21055

Allardyce, J., Suppes, T., & Van Os, J. (2007). Dimensions and the psychosis phenotype. *International journal of methods in psychiatric research, 16*(S1), S34-S40. https://doi.org/10.1002/mpr.214

Allen, P., Johns, L. C., Fu, C. H., Broome, M. R., Vythelingum, G. N., & McGuire, P. K. (2004). Misattribution of external speech in patients with hallucinations and delusions. *Schizophrenia research, 69*(2-3), 277-287. https://doi.org/10.1016/j.schres.2003.09.008

Allen, G., McColl, R., Barnard, H., Ringe, W. K., Fleckenstein, J., & Cullum, C. M. (2005a). Magnetic resonance imaging of cerebellar–prefrontal and cerebellar–parietal functional connectivity. *NeuroImage, 28*(1), 39–48. https://doi.org/10.1016/j.neuroimage.2005.06.013

Allen, P. P., Amaro, E., Fu, C. H., Williams, S. C., Brammer, M., Johns, L. C., & McGuire, P. K. (2005b). Neural correlates of the misattribution of self-generated speech. *Human Brain Mapping, 26*(1), 44-53. https://doi.org/10.1002/hbm.20120

Allen, P., Freeman, D., Johns, L., & McGuire, P. (2006). Misattribution of self-generated speech in relation to hallucinatory proneness and delusional ideation in healthy volunteers. *Schizophrenia Research, 84*(2-3), 281-288. https://doi.org/10.1016/j.schres.2006.01.021

Allen, P., Aleman, A., & McGuire, P. K. (2007a). Inner speech models of auditory verbal hallucinations: evidence from behavioural and neuroimaging studies. *International Review of Psychiatry, 19*(4), 407-415. https://doi.org/10.1080/09540260701486498

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

Allen, P., Larøi, F., McGuire, P. K., & Aleman, A. (2008). The hallucinating brain: a review of structural and functional neuroimaging studies of hallucinations. *Neuroscience & Biobehavioral Reviews, 32*(1), 175-191. https://doi.org/10.1016/j.neubiorev.2007.07.012

Allen, P., & **Modinos, G.** (2012). Structural neuroimaging in psychotic patients with auditory verbal hallucinations. In Hallucinations (pp. 251-265). Springer, New York, NY. https://doi.org/10.1007/978-1-4614-0959-5_19

Amaro Jr, E., & **Barker, G. J.** (2006). Study design in fMRI: basic principles. *Brain and cognition, 60*(3), 220-232. https://doi.org/10.1016/j.bandc.2005.11.009

Amorim, M., Roberto, M. S., Kotz, S. A., & Pinheiro, A. P. (2021). The perceived salience of vocal emotions is dampened in non-clinical auditory verbal hallucinations. *Cognitive Neuropsychiatry*, 1-14. https://doi.org/10.1080/13546805.2021.1949972

Andersson, G., & **Armstrong, D.** (1987). Complex spikes in Purkinje cells in the lateral vermis (b zone) of the cat cerebellum during locomotion. *The Journal of Physiology, 385*(1), 107–134. https://doi.org/10.1113/jphysiol.1987.sp016487

Andersson, J. L., Skare, S., & Ashburner, J. (2003). How to correct susceptibility distortions in spin-echo echo-planar images: application to diffusion tensor imaging. *Neuroimage, 20*(2), 870-888. https://doi.org/10.1016/S1053-8119(03)00336-7

Andersson, J. L., & **Sotiropoulos, S. N.** (2016). An integrated approach to correction for off-resonance effects and subject movement in diffusion MR imaging. *Neuroimage, 125*, 1063-1078. https://doi.org/10.1016/j.neuroimage.2015.10.019

Andics, A., McQueen, J. M., Petersson, K. M., Gál, V., Rudas, G., & Vidnyánszky, Z. (2010). Neural mechanisms for voice recognition. *Neuroimage, 52*(4), 1528-1540. https://doi.org/10.1016/j.neuroimage.2010.05.048

Andics, A., McQueen, J. M., & Petersson, K. M. (2013). Mean-based neural coding of voices. *Neuroimage, 79*, 351-360. https://doi.org/10.1016/j.neuroimage.2013.05.002

Andreasen, N. C., Paradiso, S., & O'leary, D. S. (1998). "Cognitive dysmetria" as an integrative theory of schizophrenia: a dysfunction in cortical-subcortical-cerebellar circuitry?. *Schizophrenia bulletin, 24*(2), 203-218. https://doi.org/10.1093/oxfordjournals.schbul.a033321

Andreasen, N. C., & **Pierson, R.** (2008). The role of the cerebellum in schizophrenia. *Biological psychiatry, 64*(2), 81-88. https://doi.org/10.1016/j.biopsych.2008.01.003

Anguera, J. A., Reuter-Lorenz, P. A., Willingham, D. T., & Seidler, R. D. (2010). Contributions of spatial working memory to visuomotor learning. *Journal of Cognitive Neuroscience, 22*(9), 1917–1930. https://doi.org/10.1162/jocn.2009.21351

Anthony, D. (2004). The cognitive neuropsychiatry of auditory verbal hallucinations: an overview. *Cognitive Neuropsychiatry, 9*(1-2), 107-123. https://doi.org/10.1080/13546800344000183

Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience, 6*(2), 115-116. https://doi.org/10.1038/nn1003

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences, 8*(4), 170-177. https://doi.org/10.1016/j.tics.2004.02.010

Aron, A. R., & Poldrack, R. A. (2006). Cortical and subcortical contributions to stop signal response inhibition: role of the subthalamic nucleus. *Journal of Neuroscience, 26*(9), 2424-2433. https://doi.org/10.1523/JNEUROSCI.4682-05.2006

Aron, A. R. (2007). The neural basis of inhibition in cognitive control. *The Neuroscientist, 13*(3), 214-228. https://doi.org/10.1177/1073858407299288

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014). Inhibition and the right inferior frontal cortex: one decade on. *Trends in Cognitive Sciences, 18*(4), 177-185. https://doi.org/10.1016/j.tics.2013.12.003

Asadi-Eydivand, M., Ebadzadeh, M. M., Solati-Hashjin, M., Darlot, C., & Abu Osman, N. A. (2015). Cerebellum-inspired neural network solution of the inverse kinematics problem. *Biological Cybernetics, 109*(6), 561-574. https://doi.org/10.1007/s00422-015-0661-7

Asai, T., & **Tanno, Y.** (2013). Why must we attribute our own action to ourselves? Auditory hallucination like-experiences as the results both from the explicit self-other attribution and implicit regulation in speech. *Psychiatry Research, 207*(3), 179-188. https://doi.org/10.1016/j.psychres.2012.09.055

Ashby, W. R. (1991). Principles of the self-organizing system. In Facets of systems science (pp. 521-536). Springer, Boston, MA. https://doi.org/10.1007/978-1-4899-0718-9_38

Astafiev, S. V., Stanley, C. M., Shulman, G. L., & Corbetta, M. (2004). Extrastriate body area in human occipital cortex responds to the performance of motor actions. *Nature Neuroscience, 7*(5), 542–548. https://doi.org/10.1038/nn1241

Attwell, D., & **Iadecola, C.** (2002). The neural basis of functional brain imaging signals. *Trends in neurosciences, 25*(12), 621-625. https://doi.org/10.1016/S0166-2236(02)02264-6

Backasch, B., Sommer, J., Klöhn-Saghatolislam, F., Müller, M. J., Kircher, T. T., & Leube, D. T. (2014). Dysconnectivity of the inferior frontal gyrus: Implications for an impaired self-other distinction in patients with schizophrenia. *Psychiatry Research: Neuroimaging, 223*(3), 202–209. https://doi.org/10.1016/j.pscychresns.2014.05.007

Badcock, J. C., & **Chhabra, S.** (2013). Voices to reckon with: perceptions of voice identity in clinical and non-clinical voice hearers. *Frontiers in Human Neuroscience, 7,* 114. https://doi.org/10.3389/fnhum.2013.00114

Badcock, P. B., Friston, K. J., & Ramstead, M. J. (2019a). The hierarchically mechanistic mind: A free-energy formulation of the human psyche. *Physics of life Reviews, 31*, 104-121. https://doi.org/10.1016/j.plrev.2018.10.002

Badcock, P. B., Friston, K. J., Ramstead, M. J., Ploeger, A., & Hohwy, J. (2019b). The hierarchically mechanistic mind: an evolutionary systems theory of the human brain, cognition, and behavior. *Cognitive, Affective, & Behavioral Neuroscience, 19*(6), 1319-1351. https://doi.org/10.3758/s13415-019-00721-3

Baess, P., Widmann, A., Roye, A., Schröger, E., & Jacobsen, T. (2009). Attenuated human auditory middle latency response and evoked 40-Hz response to self-initiated sounds. *European Journal of Neuroscience, 29*(7), 1514-1521. https://doi.org/10.1111/j.1460-9568.2009.06683.x

Baizer, J. S., Kralj-Hans, I., & Glickstein, M. (1999). Cerebellar lesions and prism adaptation in macaque monkeys. *Journal of Neurophysiology, 81* (4), 1960–1965. https://doi.org/10.1152/jn.1999.81.4.1960

Bakiri, Y., Burzomato, V., Frugier, G., Hamilton, N. B., Karadottir, R., & Attwell, D. (2009). Glutamatergic signaling in the brain's white matter. *Neuroscience, 158*(1), 266-274. https://doi.org/10.1016/j.neuroscience.2008.01.015

Balslev, D., Nielsen, F. Å., Lund, T. E., Law, I., & Paulson, O. B. (2006). Similar brain networks for detecting visuo-motor and visuo-proprioceptive synchrony. *NeuroImage, 31*(1), 308–312. https://doi.org/10.1016/j.neuroimage.2005.11.037

Balsters, J. H., Cussans, E., Diedrichsen, J., Phillips, K. A., Preuss, T. M., Rilling, J. K., & Ramnani, N. (2010). Evolution of the cerebellar cortex: The selective expansion of prefrontal-projecting cerebellar lobules. *NeuroImage, 49*(3), 2045–2052. https://doi.org/10.1016/j.neuroimage.2009.10.045

Bandettini, P. A. (2012). Twenty years of functional MRI: the science and the stories. *Neuroimage, 62*(2), 575-588. https://doi.org/10.1016/j.neuroimage.2012.04.026

Banzhaf, W. (2009). Self-organizing Systems. *Encyclopedia of complexity and systems science, 14*, 589. https://doi.org/10.1007/978-0-387-30440-3_475

Barber, L., Reniers, R., & Upthegrove, R. (2021). A review of functional and structural neuroimaging studies to investigate the inner speech model of auditory verbal hallucinations in schizophrenia. *Translational psychiatry, 11*(1), 1-12. https://doi.org/10.1038/s41398-021-01670-7

Barbieri, M. (2010). On the origin of language. *Biosemiotics, 3*(2), 201-223. https://doi.org/10.1007/s12304-010-9088-7

Barrett, T. R., & **Etheridge, J. B.** (1992). Verbal hallucinations in normals, I: People who hear 'voices'. *Applied cognitive psychology, 6*(5), 379-387. https://doi.org/10.1002/acp.2350060503

Barron, H. C., Garvert, M. M., & Behrens, T. E. (2016). Repetition suppression: a means to index neural representations using BOLD?. *Philosophical Transactions of the Royal Society B: Biological Sciences, 371*(1705), 20150355. https://doi.org/10.1098/rstb.2015.0355

Basser, P. J. (1995). Inferring microstructural features and the physiological state of tissues from diffusion-weighted images. *NMR in Biomedicine, 8*(7), 333-344. http://doi.org/10.1002/nbm.1940080707

Bastian, A. J. (2006). Learning to predict the future: The cerebellum adapts feedforward movement control. Current Opinion in *Neurobiology, 16*(6), 645–649. https://doi.org/10.1016/j.conb.2006.08.016

Bastian, A. J. (2008). Understanding sensorimotor adaptation and learning for rehabilitation. *Current opinion in neurology, 21*(6), 628. http://doi.org/10.1097/WCO.0b013e328315a293

Bastian, A. J. (2011). Moving, sensing and learning with cerebellar damage. *Current opinion in neurobiology, 21*(4), 596-601. https://doi.org/10.1016/j.conb.2011.06.007

Bastos, A. M., Usrey, W. M., Adams, R. A., Mangun, G. R., Fries, P., & Friston, K. J. (2012). Canonical microcircuits for predictive coding. *Neuron, 76*(4), 695-711. https://doi.org/10.1016/j.neuron.2012.10.038

Bates, D., Maechler, M., Bolker, B., Walker, S., Christensen, R. H. B., Singmann, H., ... & Bolker, M. B. (2015). Package 'lme4'. *Convergence, 12*(1), 2. http://cran.r-project.org/package=lme4

Bauer, S. M., Schanda, H., Karakula, H., Olajossy-Hilkesberger, L., Rudaleviciene, P., Okribelashvili, N., ... & Stompe, T. (2011). Culture and the prevalence of hallucinations in schizophrenia. *Comprehensive psychiatry, 52(*3), 319-325. https://doi.org/10.1016/j.comppsych.2010.06.008

Baumann, O., & **Belin, P.** (2010). Perceptual scaling of voice identity: common dimensions for different vowels and speakers. *Psychological Research, 74*(1), 110. https://doi.org/10.1007/s00426-008-0185-z

Baumeister, D., Sedgwick, O., Howes, O., & Peters, E. (2017). Auditory verbal hallucinations and continuum models of psychosis: a systematic review of the healthy voice-hearer literature. *Clinical Psychology Review, 51*, 125-141. https://doi.org/10.1016/j.cpr.2016.10.010

Beavan, V., Read, J., & Cartwright, C. (2011). The prevalence of voice-hearers in the general population: a literature review. *Journal of Mental Health, 20*(3), 281-292. https://doi.org/10.3109/09638237.2011.562262

Behrendt, R. P. (1998). Underconstrained perception: A theoretical approach to the nature and function of verbal hallucinations. *Comprehensive Psychiatry, 39*(4), 236-248. https://doi.org/10.1016/S0010-440X(98)90067-0

Behrens, T. E., Woolrich, M. W., Jenkinson, M., Johansen-Berg, H., Nunes, R. G., Clare, S., ... & Smith, S. M. (2003). Characterization and propagation of uncertainty in diffusion-weighted MR imaging. *Magnetic Resonance in Medicine: An Official Journal of the International Society for Magnetic Resonance in Medicine, 50*(5), 1077-1088. https://doi.org/10.1002/mrm.10609

Behrens, T. E., Berg, H. J., Jbabdi, S., Rushworth, M. F., & Woolrich, M. W. (2007). Probabilistic diffusion tractography with multiple fibre orientations: What can we gain?. *Neuroimage, 34*(1), 144-155. http://doi.org/10.1016/j.neuroimage.2006.09.018

Behroozmand, R., & **Larson, C.** (2011). Motor-induced suppression of auditory neural responses to pitch-shifted voice feedback. *The Journal of the Acoustical Society of America, 129*(4), 2454-2454. https://doi.org/10.1121/1.3588063

Behroozmand, R., Shebek, R., Hansen, D. R., Oya, H., Robin, D. A., Howard, M. A., III, & Greenlee, J. D. (2015). Sensory–motor networks involved in speech production and motor control: An fMRI study. *NeuroImage, 109*, 418–428. https://doi.org/10.1016/j.neuroimage.2015.01.040

Belin, P., Zatorre, R. J., Hoge, R., Evans, A. C., & Pike, B. (1999). Event-related fMRI of the auditory cortex. *Neuroimage, 10*(4), 417-429. https://doi.org/10.1006/nimg.1999.0480

Belin, P., & **Zatorre, R. J.** (2000). 'What', 'where' and 'how' in auditory cortex. *Nature neuroscience, 3*(10), 965-966. https://doi.org/10.1038/79890

Belin, P., Zatorre, R. J., Lafaille, P., Ahad, P., & Pike, B. (2000). Voice-selective areas in human auditory cortex. *Nature, 403*(6767), 309-312. https://doi.org/10.1038/35002078

Belin, P., Zatorre, R. J., & Ahad, P. (2002). Human temporal-lobe response to vocal sounds. *Cognitive Brain Research, 13*(1), 17-26. https://doi.org/10.1016/S0926-6410(01)00084-2

Belin, P., & **Zatorre, R. J.** (2003). Adaptation to speaker's voice in right anterior temporal lobe. *Neuroreport, 14(*16), 2105-2109. https://doi.org/10.1097/00001756-200311140-00019

Belin, P., Fecteau, S., & Bedard, C. (2004). Thinking the voice: neural correlates of voice perception. *Trends in Cognitive Sciences, 8*(3), 129-135. https://doi.org/10.1016/j.tics.2004.01.008

Belin, P., Bestelmeyer, P. E., Latinus, M., & Watson, R. (2011). Understanding voice perception. *British Journal of Psychology, 102*(4), 711-725. https://doi.org/10.1111/j.2044-8295.2011.02041.x

Belyk, M., & **Brown, S.** (2017). The origins of the vocal brain in humans. *Neuroscience & Biobehavioral Reviews, 77*, 177-193. https://doi.org/10.1016/j.neubiorev.2017.03.014

Belyk, M., Brown, S., Lim, J., & Kotz, S. A. (2017). Convergence of semantics and emotional expression within the IFG pars orbitalis. *Neuroimage, 156*, 240-248. https://doi.org/10.1016/j.neuroimage.2017.04.020

Benetti, S., Pettersson-Yeo, W., Allen, P., Catani, M., Williams, S., Barsaglini, A., ... & Mechelli, A. (2015). Auditory verbal hallucinations and brain dysconnectivity in the perisylvian language network: a multimodal investigation. *Schizophrenia bulletin, 41*(1), 192-200. https://doi.org/10.1093/schbul/sbt172

Bentall, R. P., & **Slade, P. D.** (1985). Reliability of a scale measuring disposition towards hallucination: a brief report. *Personality and Individual Differences, 6*(4), 527-529. https://doi.org/10.1016/0191-8869(85)90151-5

Bentall, R. P. (1990). The illusion of reality: a review and integration of psychological research on hallucinations. *Psychological bulletin, 107*(1), 82. https://doi.org/10.1037/0033-2909.107.1.82

Bernard, J. A., Seidler, R. D., Hassevoort, K. M., Benson, B. L., Welsh, R. C., Wiggins, J. L., ... & Peltier, S. J. (2012). Resting state cortico-cerebellar functional connectivity networks: a comparison of anatomical and self-organizing map approaches. *Frontiers in neuroanatomy, 6*, 31. https://doi.org/10.3389/fnana.2012.00031

Berry, K., Fleming, P., Wong, S., & Bucci, S. (2018). Associations between trauma, dissociation, adult attachment and proneness to hallucinations. *Behavioural and cognitive psychotherapy, 46*(3), 292-301. https://doi.org/10.1017/S1352465817000716

Bestelmeyer, P. E., Latinus, M., Bruckert, L., Rouger, J., Crabbe, F., & Belin, P. (2012). Implicitly perceived vocal attractiveness modulates prefrontal cortex activity. *Cerebral Cortex, 22*(6), 1263-1270. https://doi.org/10.1093/cercor/bhr204

Bitar, R., Leung, G., Perng, R., Tadros, S., Moody, A. R., Sarrazin, J., ... & Roberts, T. P. (2006). MR pulse sequences: what every radiologist wants to know but is afraid to ask. *Radiographics, 26*(2), 513-537. https://doi.org/10.1148/rg.262055063

Bitta, M., Thungana, Y., Kim, H. H., Denckla, C. A., Ametaj, A., Yared, M., ... & Gelaye, B. (2022). Cross-country variations in the reporting of psychotic symptoms among sub-Saharan African adults: A psychometric evaluation of the Psychosis Screening Questionnaire. *Journal of Affective Disorders, 304*, 85-92. https://doi.org/10.1016/j.jad.2022.02.048

Black, J. W. (1951). The effect of delayed side-tone upon vocal rate and intensity. *Journal of Speech and Hearing Disorders, 16*(1), 56–60. https://doi.org/10.1044/jshd.1601.56

Blakemore, S. J., Wolpert, D. M., & Frith, C. D. (1998). Central cancellation of self-produced tickle sensation. *Nature Neuroscience, 1*(7), 635–640. https://doi.org/10.1038/2870

Blakemore, S. J., Frith, C. D., & Wolpert, D. M. (2001). The cerebellum is involved in predicting the sensory consequences of action. *NeuroReport, 12*(9), 1879–1884. https://doi.org/10.1097/00001756-200107030-00023

Blakemore, S. J., Wolpert, D. M., & Frith, C. D. (2002). Abnormalities in the awareness of action. *Trends in cognitive sciences, 6*(6), 237-242. https://doi.org/10.1016/S1364-6613(02)01907-1

Blank, H., Anwander, A., & Von Kriegstein, K. (2011). Direct structural connections between voice-and face-recognition areas. *Journal of Neuroscience, 31*(36), 12906-12915. https://doi.org/10.1111/j.2044-8295.2011.02041.x

Blank, H., Wieland, N., & von Kriegstein, K. (2014). Person recognition and the brain: merging evidence from patients and healthy individuals. *Neuroscience & Biobehavioral Reviews, 47*, 717-734. https://doi.org/10.1016/j.neubiorev.2014.10.022

Bogacz, R. (2017). A tutorial on the free-energy framework for modelling perception and learning. *Journal of mathematical psychology, 76*, 198-211. https://doi.org/10.1016/j.jmp.2015.11.003

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

Boillat, Y., Bazin, P. L., & van der Zwaag, W. (2020). Whole-body somatotopic maps in the cerebellum revealed with 7T fMRI. *Neuroimage, 211*, 116624. https://doi.org/10.1016/j.neuroimage.2020.116624

Bolt, N. K., & **Loehr, J. D.** (2021). The motor-related brain activity that supports joint action: A review. *Acta Psychologica, 212*, 103218. https://doi.org/10.1016/j.actpsy.2020.103218

Bolton, T. A., Wotruba, D., Buechler, R., Theodoridou, A., Michels, L., Kollias, S., ... & Van De Ville, D. (2020). Triple network model dynamically revisited: lower salience network state switching in pre-psychosis. *Frontiers in physiology, 11*, 66. https://doi.org/10.3389/fphys.2020.00066

Bonte, M., Hausfeld, L., Scharke, W., Valente, G., & Formisano, E. (2014). Task-dependent decoding of speaker and vowel identity from auditory cortical response patterns. *Journal of Neuroscience, 34*(13), 4548-4557. https://doi.org/10.1523/JNEUROSCI.4339-13.2014

Boos, H. B., Mandl, R. C., van Haren, N. E., Cahn, W., van Baal, G. C. M., Kahn, R. S., & Pol, H. E. H. (2013). Tract-based diffusion tensor imaging in patients with schizophrenia and their non-psychotic siblings. *European Neuropsychopharmacology, 23*(4), 295-304. https://doi.org/10.1016/j.euroneuro.2012.05.015

Bopp, M. H., Zöllner, R., Jansen, A., Dietsche, B., Krug, A., & Kircher, T. T. (2017). White matter integrity and symptom dimensions of schizophrenia: a diffusion tensor imaging study. *Schizophrenia research, 184*, 59-68. https://doi.org/10.1016/j.schres.2016.11.045

Bopp, M. H., Zöllner, R., Jansen, A., Dietsche, B., Krug, A., & Kircher, T. T. (2017). White matter integrity and symptom dimensions of schizophrenia: a diffusion tensor imaging study. *Schizophrenia research, 184*, 59-68. https://doi.org/10.1016/j.schres.2016.11.045

Bourgin, J., Tebeka, S., Mallet, J., Mazer, N., Dubertret, C., & Le Strat, Y. (2020). Prevalence and correlates of psychotic-like experiences in the general population. *Schizophrenia research, 215*, 371-377. https://doi.org/10.1016/j.schres.2019.08.024

Bradbury, D. A., Stirling, J., Cavill, J., & Parker, A. (2009). Psychosis-like experiences in the general population: An exploratory factor analysis. *Personality and Individual Differences, 46*(7), 729-734. https://doi.org/10.1016/j.paid.2009.01.035

Brand, J., Michels, L., Bakker, R., Hepp-Reymond, M. C., Kiper, D., Morari, M., & Eng, K. (2017). Neural correlates of visuomotor adjustments during scaling of human finger movements. *European Journal of Neuroscience, 46*(1), 1717–1729. https://doi.org/10.1111/ejn.13606

Breier, A., & **Berg, P. H.** (1999). The psychosis of schizophrenia: prevalence, response to atypical antipsychotics, and prediction of outcome. *Biological Psychiatry, 46*(3), 361-364. https://doi.org/10.1016/S0006-3223(99)00040-2

Brennan, A. M., Harris, A. W., & Williams, L. M. (2013). Functional dysconnectivity in schizophrenia and its relationship to neural synchrony. *Expert review of neurotherapeutics, 13*(7), 755-765. https://doi.org/10.1586/14737175.2013.811899

Brett, M., Anton, J., Valabregue, R., & Poline, J. Region of interest analysis using an SPM toolbox [Abstract] Presented at the 8th International Conference on Functional Mapping of the Human Brain, June 2-6, 2002, Sendai, Japan. Available on CD-ROM in *NeuroImage, 16*(2). https://marsbar-toolbox.github.io/

Briggs, R. G., Rahimi, M., Conner, A. K., Sali, G., Baker, C. M., Burks, J. D., ... & Sughrue, M. E. (2018). A Connectomic Atlas of the Human Cerebrum—Chapter 15: Tractographic Description of the Uncinate Fasciculus. *Operative Neurosurgery, 15*(suppl_1), S450-S455. https://doi.org/10.1093/ons/opy269

Brisch, R., Saniotis, A., Wolf, R., Bielau, H., Bernstein, H. G., Steiner, J., ... & Gos, T. (2014). The role of dopamine in schizophrenia from a neurobiological and evolutionary perspective: old fashioned, but still in vogue. *Frontiers in psychiatry, 5*, 47. https://doi.org/10.3389/fpsyt.2014.00047

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

Broucke, M. E. (2021, July). On the use of regulator theory in neuroscience with implications for robotics. In 18th International Conference on Informatics in Control, Automation, and Robotics. Retrieved from https://www.scitepress.org/Papers/2021/106391/106391.pdf

Brower, R., Wang, H. R., Bansal, S., & Joiner, W. M. (2019). Using Corollary Discharge and Predictive Coding to Understand False Sensations and Beliefs. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 4*(9), 770-772. https://doi.org/10.1016/j.bpsc.2019.07.004

Brown, E. C., & **Brüne, M.** (2012). The role of prediction in social neuroscience. *Frontiers in human neuroscience, 6*, 147. https://doi.org/10.3389/fnhum.2012.00147

Bruckert, L., Bestelmeyer, P., Latinus, M., Rouger, J., Charest, I., Rousselet, G. A., ... & Belin, P. (2010). Vocal attractiveness increases by averaging. *Current Biology, 20*(2), 116-120. https://doi.org/10.1016/j.cub.2009.11.034

Bruineberg, J., & **Rietveld, E.** (2014). Self-organization, free energy minimization, and optimal grip on a field of affordances. *Frontiers in human neuroscience, 8*, 599. https://doi.org/10.3389/fnhum.2014.00599

Bruineberg, J., Kiverstein, J., & Rietveld, E. (2018a). The anticipating brain is not a scientist: the free-energy principle from an ecological-enactive perspective. *Synthese, 195*(6), 2417-2444. https://doi.org/10.1007/s11229-016-1239-1

Bruineberg, J., Rietveld, E., Parr, T., van Maanen, L., & Friston, K. J. (2018b). Free-energy minimization in joint agent-environment systems: A niche construction perspective. *Journal of theoretical biology, 455*, 161-178. https://doi.org/10.1016/j.jtbi.2018.07.002.

Buckley, C. L., Kim, C. S., McGregor, S., & Seth, A. K. (2017). The free energy principle for action and perception: A mathematical review. *Journal of Mathematical Psychology, 81*, 55-79. https://doi.org/10.1016/j.jmp.2017.09.004

Buckner, R. L., Krienen, F. M., Castellanos, A., Diaz, J. C., & Yeo, B. T. (2011). The organization of the human cerebellum estimated by intrinsic functional connectivity. *Journal of Neurophysiology, 106*, 2322–2345. https://doi.org/10.1152/jn.00339.2011

Buckner, R. L. (2013). The cerebellum and cognitive function: 25 years of insight from anatomy and neuroimaging. *Neuron, 80*(3), 807-815. https://doi.org/10.1016/j.neuron.2013.10.044

Buckner, R. L. (2022). The brain's default network: origins and implications for the study of psychosis. *Dialogues in clinical neuroscience*. https://doi.org/10.31887/DCNS.2013.15.3/rbuckner

Bullmore, E., & **Sporns, O.** (2009). Complex brain networks: graph theoretical analysis of structural and functional systems. *Nature reviews neuroscience, 10*(3), 186-198. https://doi.org/10.1038/nrn2575

Burnett, T. A., Freedland, M. B., Larson, C. R., & Hain, T. C. (1998). Voice F0 responses to manipulations in pitch feedback. The *Journal of the Acoustical Society of America, 103*(6), 3153–3161. https://doi.org/10.1121/1.423073

Butt, A. M., Fern, R. F., & Matute, C. (2014). Neurotransmitter signaling in white matter. *Glia, 62*(11), 1762-1779. http://doi.org/10.1002/glia.22674

Buxton, R. B., & **Frank, L. R.** (1997). A model for the coupling between cerebral blood flow and oxygen metabolism during neural stimulation. *Journal of cerebral blood flow & metabolism, 17*(1), 64-72. https://doi.org/10.1097/00004647-199701000-00009

Cai, W., Ryali, S., Chen, T., Li, C. S. R., & Menon, V. (2014). Dissociable roles of right inferior frontal cortex and anterior insula in inhibitory control: evidence from intrinsic and task-related functional parcellation, connectivity, and response profile analyses across multiple datasets. *Journal of Neuroscience, 34*(44), 14652-14667. https://doi.org/10.1523/JNEUROSCI.3048-14.2014

Calhoun, V. D., Eichele, T., & Pearlson, G. (2009). Functional brain networks in schizophrenia: a review. *Frontiers in human neuroscience*, 17. https://doi.org/10.3389/neuro.09.017.2009

Caligiore, D., Pezzulo, G., Baldassarre, G., Bostan, A. C., Strick, P. L., Doya, K., ... & Herreros, I. (2017). Consensus paper: towards a systems-level view of cerebellar function: the interplay between cerebellum, basal ganglia, and cortex. *The Cerebellum, 16*(1), 203-229. https://doi.org/10.1007/s12311-016-0763-3

Campbell, J. O. (2016). Universal Darwinism as a process of Bayesian inference. *Frontiers in Systems Neuroscience, 10*, 49. https://doi.org/10.3389/fnsys.2016.00049

Candini, M., Zamagni, E., Nuzzo, A., Ruotolo, F., Iachini, T., & Frassinetti, F. (2014). Who is speaking? Implicit and explicit self and other voice recognition. *Brain and Cognition, 92*, 112-117. https://doi.org/10.1016/j.bandc.2014.10.001

Cao, H., & **Cannon, T. D.** (2019). Cerebellar dysfunction and schizophrenia: from "cognitive dysmetria" to a potential therapeutic target. *American Journal of Psychiatry, 176*(7), 498-500. https://doi.org/10.1176/appi.ajp.2019.19050480

Carbajal, G. V., & **Malmierca, M. S.** (2018). The neuronal basis of predictive coding along the auditory pathway: from the subcortical roots to cortical deviance detection. *Trends in Hearing, 22,* 2331216518784822. https://doi.org/10.1177/2331216518784822

Carlsson, A. (1977). Does dopamine play a role in schizophrenia?. *Psychological medicine, 7*(4), 583-597. https://doi.org/10.1017/S003329170000622X

Carlsson, A. (1978). Antipsychotic drugs, neurotransmitters, and schizophrenia. *American Journal of Psychiatry, 135*(2), 164-173. https://doi.org/10.1176/ajp.135.2.164

Carlsson, A. (2001). A half-century of neurotransmitter research: impact on neurology and psychiatry (Nobel lecture). *Chembiochem, 2*(7-8), 484-493. https://doi.org/10.1002/1439-7633(20010803)2:7/8<484::AID-CBIC484>3.0.CO;2-5

Carr, H. Y., & **Purcell, E. M.** (1954). Effects of diffusion on free precession in nuclear magnetic resonance experiments. *Physical review, 94*(3), 630. https://doi.org/10.1103/PhysRev.94.630

Carrillo, R. R., Ros, E., Boucheny, C., & Olivier, J. M. C. (2008). A real-time spiking cerebellum model for learning robot control. *Biosystems, 94*(1-2), 18-27. https://doi.org/10.1016/j.biosystems.2008.05.008

Casellato, C., Antonietti, A., Garrido, J. A., Carrillo, R. R., Luque, N. R., Ros, E., ... & D'Angelo, E. (2014). Adaptive robotic control driven by a versatile spiking cerebellar network. *PLoS one, 9*(11), e112265. https://doi.org/10.1371/journal.pone.0112265

Casellato, C., Antonietti, A., Garrido, J. A., Ferrigno, G., D'Angelo, E., & Pedrocchi, A. (2015). Distributed cerebellar plasticity implements generalized multiple-scale memory components in real-robot sensorimotor tasks. *Frontiers in Computational Neuroscience, 9,* 24. https://doi.org/10.3389/fncom.2015.00024.

Castiajo, P., & **Pinheiro, A. P.** (2021). Acoustic salience in emotional voice perception and its relationship with hallucination proneness. *Cognitive, Affective, & Behavioral Neuroscience, 21*(2), 412-425. https://doi.org/10.3758/s13415-021-00864-2

Catani, M., & **De Schotten, M. T.** (2008). A diffusion tensor imaging tractography atlas for virtual in vivo dissections. *Cortex, 44*(8), 1105-1132. https://doi.org/10.1016/j.cortex.2008.05.004

Catani, M., Craig, M. C., Forkel, S. J., Kanaan, R., Picchioni, M., Toulopoulou, T., ... & McGuire, P. (2011). Altered integrity of perisylvian language pathways in schizophrenia: relationship to auditory hallucinations. *Biological psychiatry, 70*(12), 1143-1150. https://doi.org/10.1016/j.biopsych.2011.06.013

Cella, M., Cooper, A., Dymond, S. O., & Reed, P. (2008). The relationship between dysphoria and proneness to hallucination and delusions among young adults. *Comprehensive Psychiatry, 49*(6), 544-550. https://doi.org/10.1016/j.comppsych.2008.02.011

Chambers, C. D., Bellgrove, M. A., Stokes, M. G., Henderson, T. R., Garavan, H., Robertson, I. H., ... & Mattingley, J. B. (2006). Executive "brake failure" following deactivation of human frontal lobe. *Journal of Cognitive Neuroscience, 18*(3), 444-455. https://doi.org/10.1162/jocn.2006.18.3.444

Chang-Yit, R., Pick, H. L., Jr., & Siegel, G. M. (1975). Reliability of sidetone amplification effect in vocal intensity. *Journal of Communication Disorders, 8*(4), 317–324. https://doi.org/10.1016/0021-9924(75)90032-5

Chang, E. F., Niziolek, C. A., Knight, R. T., Nagarajan, S. S., & Houde, J. F. (2013). Human cortical sensorimotor network underlying feedback control of vocal pitch. *Proceedings of the National Academy of Sciences, 110*(7), 2653-2658. https://doi.org/10.1073/pnas.1216827110

Chapman, L. J., & **Chapman, J. P.** (1980). Scales for rating psychotic and psychotic-like experiences as continua. *Schizophrenia Bulletin, 6*(3), 476. https://doi.org/10.1093/schbul/6.3.476

Charest, I., Pernet, C., Latinus, M., Crabbe, F., & Belin, P. (2013). Cerebral processing of voice gender studied using a continuous carryover fMRI design. *Cerebral Cortex, 23*(4), 958-966. https://doi.org/10.1093/cercor/bhs090

Chawla, N., Deep, R., Khandelwal, S. K., & Garg, A. (2019). Reduced integrity of superior longitudinal fasciculus and arcuate fasciculus as a marker for auditory hallucinations in schizophrenia: a DTI tractography study. *Asian journal of psychiatry*, 44, 179-186. https://doi.org/10.1016/j.ajp.2019.07.043

Cheung, C., Hamilton, L. S., Johnson, K., & Chang, E. F. (2016). The auditory representation of speech sounds in human motor cortex. ***elife, 5***, e12577. https://doi.org/10.7554/eLife.12577

Chevrier, A. D., Noseworthy, M. D., & Schachar, R. (2007). Dissociation of response inhibition and performance monitoring in the stop signal task using event-related fMRI. *Human brain mapping, 28*(12), 1347-1358. https://doi.org/10.1002/hbm.20355

Christiansen, M. H., & **Kirby, S.** (2003). Language evolution: Consensus and controversies. *Trends in cognitive sciences, 7*(7), 300-307. https://doi.org/10.1016/S1364-6613(03)00136-0

Christensen, M. S., Lundbye-Jensen, J., Petersen, N., Geertsen, S. S., Paulson, O. B., & Nielsen, J. B. (2006). Watching your foot move—An fMRI study of visuomotor interactions during foot movement. *Cerebral Cortex, 17*(8), 1906–1917. https://doi.org/10.1093/cercor/bhl101

Christoffels, I. K., Formisano, E., & Schiller, N. O. (2007). Neural correlates of verbal feedback processing: an fMRI study employing overt speech. *Human Brain Mapping, 28*(9), 868-879. https://doi.org/10.1002/hbm.20315

Christoffels, I. K., van de Ven, V., Waldorp, L. J., Formisano, E., & Schiller, N. O. (2011). The sensory consequences of speaking: parametric neural cancellation during speech in auditory cortex. *PLoS One, 6*(5). https://doi.org/10.1371/journal.pone.0018307

Cho, R., & **Wu, W.** (2013). Mechanisms of auditory verbal hallucination in schizophrenia. *Frontiers in psychiatry, 4*, 155. https://doi.org/10.3389/fpsyt.2013.00155

Claridge, G. (1994). Single indicator of risk for schizophrenia: probable fact or likely myth?. *Schizophrenia bulletin, 20(*1), 151-168. https://doi.org/10.1093/schbul/20.1.151

Clark, A. (2013). Whatever next? Predictive brains, situated agents, and the future of cognitive science. *Behavioral and brain sciences, 36*(3), 181-204. https://doi.org/10.1017/S0140525X12000477

Clark, A., Friston, K., & Wilkinson, S. (2019). Bayesing qualia: Consciousness as inference, not raw datum. *Journal of Consciousness Studies, 26*(9-10), 19-33. Retrieved from http://hdl.handle.net/10871/39428

Clower, D. M., West, R. A., Lynch, J. C., & Strick, P. L. (2001). The inferior parietal lobule is the target of output from the superior colliculus, hippocampus, and cerebellum. *Journal of Neuroscience, 21*(16), 6283–6291. https://doi.org/10.1523/JNEUROSCI.21-16-06283.2001

Cocchi, L., Harding, I. H., Lord, A., Pantelis, C., Yucel, M., & Zalesky, A. (2014). Disruption of structure–function coupling in the schizophrenia connectome. *NeuroImage: Clinical, 4*, 779-787. https://doi.org/10.1016/j.nicl.2014.05.004

Coenen, O. J., Arnold, M. P., Sejnowski, T. J., & Jabri, M. A. (2001). Parallel fiber coding in the cerebellum for life-long learning. *Autonomous robots, 11(*3), 291-297. https://doi.org/10.1023/A:1012403510221

Collignon, O., Van der Linden, M., & Larøi, F. (2005). Source monitoring for actions in hallucination proneness. *Cognitive Neuropsychiatry, 10*(2), 105-123. https://doi.org/10.1080/13546800344000354

Conant, R. C., & **Ross Ashby, W.** (1970). Every good regulator of a system must be a model of that system. *International journal of systems science, 1*(2), 89-97. https://doi.org/10.1080/00207727008920220

Conde, T., Gonçalves, Ó. F., & Pinheiro, A. P. (2018). Stimulus complexity matters when you hear your own voice: Attention effects on self-generated voice processing. *International Journal of Psychophysiology*, 133, 66-78. https://doi.org/10.1016/j.ijpsycho.2018.08.007

Constant, A., Ramstead, M. J., Veissière, S. P., & Friston, K. (2019). Regimes of expectations: An active inference model of social conformity and human decision making. *Frontiers in psychology, 10*, 679. https://doi.org/10.3389/fpsyg.2019.00679

Copolov, D. L., Seal, M. L., Maruff, P., Ulusoy, R., Wong, M. T., Tochon-Danguy, H. J., & Egan, G. F. (2003). Cortical activation associated with the experience of auditory hallucinations and perception of human speech in schizophrenia: a PET correlation study. *Psychiatry Research: Neuroimaging, 122*(3), 139-152. https://doi.org/10.1016/S0925-4927(02)00121-X

Corbetta, M., & **Shulman, G. L.** (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*(3), 201–215. https://doi.org/10.1038/nrn755

Corlett, P. R., Taylor, J. R., Wang, X. J., Fletcher, P. C., & Krystal, J. H. (2010). Toward a neurobiology of delusions. *Progress in neurobiology, 92*(3), 345-369. https://doi.org/10.1016/j.pneurobio.2010.06.007

Corlett, P. R., Honey, G. D., Krystal, J. H., & Fletcher, P. C. (2011). Glutamatergic model psychoses: prediction error, learning, and inference. *Neuropsychopharmacology, 36*(1), 294-315. https://doi.org/10.1038/npp.2010.163

Corlett, P. R., Horga, G., Fletcher, P. C., Alderson-Day, B., Schmack, K., & Powers III, A. R. (2019). Hallucinations and strong priors. *Trends in cognitive sciences, 23*(2), 114-127. https://doi.org/10.1016/j.tics.2018.12.001

Corrigan, P. W., Schmidt, A., Bink, A. B., Nieweglowski, K., Al-Khouja, M. A., Qin, S., & Discont, S. (2017). Changing public stigma with continuum beliefs. *Journal of Mental Health, 26*(5), 411-418. https://doi.org/10.1080/09638237.2016.1207224

Costafreda, S. G., Brébion, G., Allen, P., McGuire, P. K., & Fu, C. H. (2008). Affective modulation of external misattribution bias in source monitoring in schizophrenia. *Psychological Medicine, 38*(6), 821-824. https://doi.org/10.1017/S0033291708003243

Coyle, J. T. (1996). The glutamatergic dysfunction hypothesis for schizophrenia. *Harvard review of psychiatry, 3*(5), 241-253. http://doi.org/10.3109/10673229609017192

Coyle, J. T., Balu, D., Puhl, M., & Konopaske, G. (2016). A perspective on the history of the concept of "disconnectivity" in schizophrenia. *Harvard review of psychiatry, 24*(2), 80. http://doi.org/10.1097/HRP.0000000000000102

Craik, K. J. W. (1952). The nature of explanation (Vol. 445). CUP Archive. https://doi.org/10.2307/2181361

Crapse, T. B., & **Sommer, M. A.** (2008). Corollary discharge across the animal kingdom. *Nature Reviews Neuroscience, 9*(8), 587-600. https://doi.org/10.1038/nrn2457

Ćurčić-Blake, B., Nanetti, L., van der Meer, L., Cerliani, L., Renken, R., Pijnenborg, G. H., & Aleman, A. (2015). Not on speaking terms: hallucinations and structural network disconnectivity in schizophrenia. *Brain Structure and Function, 220*(1), 407-418. http://doi.org/10.1007/s00429-013-0663-y

Ćurčić-Blake, B., Ford, J. M., Hubl, D., Orlov, N. D., Sommer, I. E., Waters, F., ... & Aleman, A. (2017a). Interaction of language, auditory and memory brain networks in auditory verbal hallucinations. *Progress in neurobiology, 148*, 1-20. https://doi.org/10.1016/j.pneurobio.2016.11.002

Ćurčić-Blake, B., Bais, L., Sibeijn-Kuiper, A., Pijnenborg, H. M., Knegtering, H., Liemburg, E., & Aleman, A. (2017b). Glutamate in dorsolateral prefrontal cortex and auditory verbal hallucinations in patients with schizophrenia: a 1H MRS study. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 78*, 132-139. https://doi.org/10.1016/j.pnpbp.2017.05.020

Ćurčić-Blake B., Houenou J., Jardri R. (2018) Dysconnectivity in Hallucinations. In: Brambilla P., Mauri M., Altamura A. (eds) Hallucinations in Psychoses and Affective Disorders. Springer, Cham. https://doi.org/10.1007/978-3-319-75124-5_10

Curio, G., Neuloh, G., Numminen, J., Jousmäki, V., & Hari, R. (2000). Speaking modifies voice-evoked activity in the human auditory cortex. *Human brain mapping, 9*(4), 183-191. https://doi.org/10.1002/(SICI)1097-0193(200004)9:4<183::AID-HBM1>3.0.CO;2-Z

Currie, S., Hoggard, N., Craven, I. J., Hadjivassiliou, M., & Wilkinson, I. D. (2013). Understanding MRI: basic MR physics for physicians. *Postgraduate medical journal, 89*(1050), 209-223. http://doi.org/10.1136/postgradmedj-2012-131342

Daalman, K., Boks, M. P., Diederen, K. M., de Weijer, A. D., Blom, J. D., Kahn, R. S., & Sommer, I. E. (2011). The same or different? A phenomenological comparison of auditory verbal hallucinations in healthy and psychotic individuals. *The Journal of clinical psychiatry, 72*(3), 0-0. https://doi.org/10.4088/JCP.09m05797yel

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

D'Agostino, A., Limosani, I., & Scarone, S. (2012). The dreaming brain/mind: a role in understanding complex mental disorders?. *Frontiers in psychiatry, 3*, 3. https://doi.org/10.3389/fpsyt.2012.00003

Damiani, S., Donadeo, A., Bassetti, N., Salazar-de-Pablo, G., Guiot, C., Politi, P., & Fusar-Poli, P. (2022). Understanding source monitoring subtypes and their relation to psychosis: a systematic review and meta-analysis. *Psychiatry and Clinical* Neurosciences. https://doi.org/10.1111/pcn.13338

Davies-Thompson, J., Elli, G. V., Rezk, M., Benetti, S., van Ackeren, M., & Collignon, O. (2019). Hierarchical brain network for face and voice integration of emotion expression. *Cerebral Cortex, 29*(9), 3590-3605. https://doi.org/10.1093/cercor/bhy240

David, N., Cohen, M. X., Newen, A., Bewernick, B. H., Shah, N. J., Fink, G. R., & Vogeley, K. (2007). The extrastriate cortex distinguishes between the consequences of one's own and others' behavior. *NeuroImage, 36*(3), 1004–1014. https://doi.org/10.1016/j.neuroimage.2007.03.030

David, N., Newen, A., & Vogeley, K. (2008). The "sense of agency" and its underlying cognitive and neural mechanisms. *Consciousness and cognition, 17*(2), 523-534. https://doi.org/10.1016/j.concog.2008.03.004

Davis, K. L., Stewart, D. G., Friedman, J. I., Buchsbaum, M., Harvey, P. D., Hof, P. R., ... & Haroutunian, V. (2003). White matter changes in schizophrenia: evidence for myelin-related dysfunction. *Archives of general psychiatry, 60*(5), 443-456. http://doi.org/10.1001/archpsyc.60.5.443

Davis, M. H., & **Johnsrude, I. S.** (2007). Hearing speech sounds: top-down influences on the interface between audition and speech perception. *Hearing research, 229*(1-2), 132-147. https://doi.org/10.1016/j.heares.2007.01.014

Dayan, P., Hinton, G. E., Neal, R. M., & Zemel, R. S. (1995). The Helmholtz machine. *Neural computation, 7*(5), 889-904. http://doi.org/10.1162/neco.1995.7.5.889

Dean, P., Porrill, J., Ekerot, C. F., & Jörntell, H. (2010). The cerebellar microcircuit as an adaptive filter: experimental and computational evidence. *Nature Reviews Neuroscience, 11*(1), 30-43. https://doi.org/10.1038/nrn2756

de Boer, J. N., Heringa, S. M., van Dellen, E., Wijnen, F. N. K., & Sommer, I. E. C. (2016). A linguistic comparison between auditory verbal hallucinations in patients with a psychotic disorder and in nonpsychotic individuals: Not just what the voices say, but how they say it. *Brain and Language, 162*, 10–18. https://doi.org/10.1016/j.bandl.2016.07.011

de Bruin, L., & Michael, J. (2021). Prediction error minimization as a framework for social cognition research. *Erkenntnis, 86*(1), 1-20. https://doi.org/10.1007/s10670-018-0090-9

Decety, J., Chaminade, T., Grezes, J., & Meltzoff, A. N. (2002). A PET exploration of the neural mechanisms involved in reciprocal imitation. *NeuroImage, 15*(1), 265–272. https://doi.org/10.1006/nimg.2001.0938

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

Deneve, S., & Jardri, R. (2016). Circular inference: mistaken belief, misplaced trust. *Current Opinion in Behavioral Sciences, 11*, 40-48. https://doi.org/10.1016/j.cobeha.2016.04.001

DeRosse, P., & **Karlsgodt, K. H.** (2015). Examining the psychosis continuum. *Current behavioral neuroscience reports, 2*(2), 80-89. https://doi.org/10.1007/s40473-015-0040-7

Desmurget, M., & **Grafton, S.** (2000). Forward modeling allows feedback control for fast reaching movements. *Trends in Cognitive Sciences, 4*(11), 423–431. https://doi.org/10.1016/S1364-6613(00)01537-0

Desmurget, M., & **Sirigu, A.** (2009). A parietal-premotor network for movement intention and motor awareness. *Trends in Cognitive Sciences*, 13 (10), 411–419. https://doi.org/10.1016/j.tics.2009.08.001

Devlin, J. T., & **Aydelott, J.** (2009). Speech perception: Motoric contributions versus the motor theory. *Current Biology, 19(*5), R198-R200. https://doi.org/10.1016/j.cub.2009.01.005

de Weijer, A. D., Mandl, R. C. W., Diederen, K. M. J., Neggers, S. F. W., Kahn, R. S., Pol, H. H., & Sommer, I. E. C. (2011). Microstructural alterations of the arcuate fasciculus in schizophrenia patients with frequent auditory verbal hallucinations. *Schizophrenia research, 130*(1-3), 68-77. https://doi.org/10.1016/j.schres.2011.05.010

de Weijer, A. D., Neggers, S. F., Diederen, K. M., Mandl, R. C., Kahn, R. S., Hulshoff Pol, H. E., & Sommer, I. E. (2013). Aberrations in the arcuate fasciculus are associated with auditory verbal hallucinations in psychotic and in non-psychotic individuals. *Human brain mapping, 34*(3), 626-634. http://doi.org/10.1002/hbm.21463

Diedrichsen, J., Hashambhoy, Y., Rane, T., & Shadmehr, R. (2005). Neural correlates of reach errors. *Journal of Neuroscience, 25*(43), 9919–9931. https://doi.org/10.1523/JNEUROSCI.1874-05.2005

Diedrichsen, J., Verstynen, T., Schlerf, J., & Wiestler, T. (2010). Advances in functional imaging of the human cerebellum. *Current Opinion in Neurology, 23*(4), 382–387. https://doi.org/10.1097/WCO.0b013e32833be837

Diehl, R. L., Lotto, A. J., & Holt, L. L. (2004). Speech perception. *Annu. Rev. Psychol., 55*, 149-179. https://doi.org/10.1146/annurev.psych.55.090902.142028

Dijkstra, N., Kok, P., & Fleming, S. M. (2022). Perceptual reality monitoring: Neural mechanisms dissociating imagination from reality. *Neuroscience & Biobehavioral Reviews,* 104557. https://doi.org/10.1016/j.neubiorev.2022.104557

Ding, Y., Ou, Y., Pan, P., Shan, X., Chen, J., Liu, F., ... & Guo, W. (2019). Cerebellar structural and functional abnormalities in first-episode and drug-naive patients with schizophrenia: a meta-analysis. *Psychiatry Research: Neuroimaging, 283*, 24-33. https://doi.org/10.1016/j.pscychresns.2018.11.009

Ditman, T., & **Kuperberg, G. R.** (2005). A source-monitoring account of auditory verbal hallucinations in patients with schizophrenia. *Harvard review of psychiatry, 13*(5), 280-299. https://doi.org/10.1080/10673220500326391

Donath, T. M., Natke, U., & Kalveram, K. T. (2002). Effects of frequency-shifted auditory feedback on voice F 0 contours in syllables. *The Journal of the Acoustical Society of America, 111*(1), 357–366. https://doi.org/10.1121/1.1424870

Dosenbach, N. U., Fair, D. A., Miezin, F. M., Cohen, A. L., Wenger, K. K., Dosenbach, R. A., ... & Schlaggar, B. L. (2007). Distinct brain networks for adaptive and stable task control in humans. *Proceedings of the National Academy of Sciences, 104*(26), 11073-11078. https://doi.org/10.1073/pnas.0704320104

Downar, J., Crawley, A. P., Mikulis, D. J., & Davis, K. D. (2000). A multimodal cortical network for the detection of changes in the sensory environment. *Nature neuroscience, 3*(3), 277-283. https://doi.org/10.1038/72991

Downar, J., Crawley, A. P., Mikulis, D. J., & Davis, K. D. (2001). The effect of task relevance on the cortical response to changes in visual and auditory stimuli: An event-related fMRI study. *NeuroImage, 14*(6),1256–1267. https://doi.org/10.1006/nimg.2001.0946

Downar, J., Crawley, A. P., Mikulis, D. J., & Davis, K. D. (2002). A cortical network sensitive to stimulus salience in a neutral behavioral context across multiple sensory modalities. *Journal of neurophysiology, 87*(1), 615-620. https://doi.org/10.1152/jn.00636.2001

Downing, P. E., Jiang, Y., Shuman, M., & Kanwisher, N. (2001). A cortical area selective for visual processing of the human body. *Science, 293*(5539), 2470–2473. https://doi.org/10.1126/science.1063414

Downing, P. E., Peelen, M. V., Wiggett, A. J., & Tew, B. D. (2006). The role of the extrastriate body area in action perception. *Social Neuroscience, 1*(1), 52–62. https://doi.org/10.1080/17470910600668854

Doya, K. (2000). Complementary roles of basal ganglia and cerebellum in learning and motor control. *Current opinion in neurobiology, 10*(6), 732-739. https://doi.org/10.1016/S0959-4388(00)00153-7

Doya, K., Kimura, H., & Kawato, M. (2001). Neural mechanisms of learning and control. *IEEE Control Systems Magazine, 21*(4), 42-54. http://doi.org/10.1109/37.939943

Doyon, J., Penhune, V., & Ungerleider, L. G. (2003). Distinct contribution of the cortico-striatal and cortico-cerebellar systems to motor skill learning. *Neuropsychologia, 41*(3), 252–262. https://doi.org/10.1016/S0028-3932(02)00158-6

Doyon, J., Bellec, P., Amsel, R., Penhune, V., Monchi, O., Carrier, J., ... & Benali, H. (2009). Contributions of the basal ganglia and functionally related brain structures to motor learning. *Behavioural brain research, 199*(1), 61-75. https://doi.org/10.1016/j.bbr.2008.11.012

Dum, R. P., & **Strick, P. L.** (2003). An unfolded map of the cerebellar dentate nucleus and its projections to the cerebral cortex. *Journal of Neurophysiology, 89*(1), 634–639. https://doi.org/10.1152/jn.00626.2002

Dutta, R., Greene, T., Addington, J., McKenzie, K., Phillips, M., & Murray, R. M. (2007). Biological, life course, and cross-cultural studies all point toward the value of dimensional and developmental ratings in the classification of psychosis. *Schizophrenia Bulletin, 33*(4), 868-876. https://doi.org/10.1093/schbul/sbm059

Eaton, W. W., Romanoski, A., Anthony, J. C., & Nestadt, G. (1991). Screening for psychosis in the general population with a self-report interview. *Journal of nervous and mental Disease*. https://doi.org/10.1097/00005053-199111000-00007

Eckert, M. A., Menon, V., Walczak, A., Ahlstrom, J., Denslow, S., Horwitz, A., & Dubno, J. R. (2009). At the heart of the ventral attention system: the right anterior insula. *Human brain mapping, 30*(8), 2530-2541. https://doi.org/10.1002/hbm.20688

Eddy, C. M. (2016). The junction between self and other? Temporo-parietal dysfunction in neuropsychiatry. *Neuropsychologia, 89*, 465-477. https://doi.org/10.1016/j.neuropsychologia.2016.07.030

Eickhoff, S. B., Laird, A. R., Grefkes, C., Wang, L. E., Zilles, K., & Fox, P. T. (2009). Coordinate-based activation likelihood estimation meta-analysis of neuroimaging data: A random-effects approach based on empirical estimates of spatial uncertainty. *Human Brain Mapping, 30*(9), 2907–2926. https://doi.org/10.1002/hbm.20718

Eickhoff, S. B., Bzdok, D., Laird, A. R., Kurth, F., & Fox, P. T. (2012). Activation likelihood estimation meta-analysis revisited. *NeuroImage, 59*(3), 2349–2361. https://doi.org/10.1016/j.neuroimage.2011.09.017

Ekstrom, A. (2010). How and when the fMRI BOLD signal relates to underlying neural activity: The danger in dissociation. *Brain Research Reviews, 62*(2), 233–244. https://doi.org/10.1016/j.brainresrev.2009.12.004

Eliades, S. J., & **Wang, X.** (2008). Neural substrates of vocalization feedback monitoring in primate auditory cortex. *Nature, 453*(7198), 1102-1106. https://doi.org/10.1038/nature06910

Ellaway, P. H., Prochazka, A., Chan, M., & Gauthier, M. J. (2004). The sense of movement elicited by transcranial magnetic stimulation in humans is due to sensory feedback. *The Journal of Physiology, 556*(2), 651–660. https://doi.org/10.1113/jphysiol.2003.060483

Elman, J. L. (1981). Effects of frequency-shifted feedback on the pitch of vocal productions. *The Journal of the Acoustical Society of America, 70*(1), 45–50. https://doi.org/10.1121/1.386580

Escartí, M. J., de la Iglesia-Vayá, M., Martí-Bonmatí, L., Robles, M., Carbonell, J., Lull, J. J., ... & Sanjuán, J. (2010). Increased amygdala and parahippocampal gyrus activation in schizophrenic patients with auditory hallucinations: an fMRI study using independent component analysis. *Schizophrenia research, 117*(1), 31-41. https://doi.org/10.1016/j.schres.2009.12.028

Esterberg, M. L., & **Compton, M. T.** (2009). The psychosis continuum and categorical versus dimensional diagnostic approaches. *Current psychiatry reports, 11*(3), 179-184. https://doi.org/10.1007/s11920-009-0028-7

Ethofer, T., Anders, S., Erb, M., Herbert, C., Wiethoff, S., Kissler, J., ... & Wildgruber, D. (2006). Cerebral pathways in processing of affective prosody: a dynamic causal modeling study. *Neuroimage, 30*(2), 580-587. https://doi.org/10.1016/j.neuroimage.2005.09.059

Ethofer, T., Wiethoff, S., Anders, S., Kreifelts, B., Grodd, W., & Wildgruber, D. (2007). The voices of seduction: cross-gender effects in processing of erotic prosody. *Social Cognitive and Affective Neuroscience, 2*(4), 334-337. https://doi.org/10.1093/scan/nsm028

Ethofer, T., Bretscher, J., Wiethoff, S., Bisch, J., Schlipf, S., Wildgruber, D., & Kreifelts, B. (2013). Functional responses and structural connections of cortical areas for processing faces and voices in the superior temporal sulcus. *Neuroimage, 76*, 45-56. https://doi.org/10.1016/j.neuroimage.2013.02.064

Fairbanks, G., & Guttman, N. (1958). Effects of delayed auditory feedback upon articulation. Journal of Speech, Language, and Hearing Research, 1, 12–22. https://doi.org/10.1044/jshr.0101.12

Farrer, C., Frey, S. H., Van Horn, J. D., Tunik, E., Turk, D., Inati, S., & Grafton, S. T. (2008). The angular gyrus computes action awareness representations. *Cerebral Cortex, 18*(2), 254–261. https://doi.org/10.1093/cercor/bhm050

Farrer, C., & **Frith, C. D.** (2002). Experiencing oneself vs another person as being the cause of an action: The neural correlates of the experience of agency. *NeuroImage, 15*(3), 596–603. https://doi.org/10.1006/nimg.2001.1009

Fecteau, S., Armony, J. L., Joanette, Y., & Belin, P. (2004). Is voice processing species-specific in human auditory cortex? An fMRI study. *Neuroimage, 23*(3), 840-848. https://doi.org/10.1016/j.neuroimage.2004.09.019

Feinberg, I. (1978). Efference copy and corollary discharge: implications for thinking and its disorders. *Schizophrenia bulletin, 4*(4), 636. https://doi.org/10.1093/schbul/4.4.636

Feinberg, I. (2011). Corollary discharge, hallucinations, and dreaming. *Schizophrenia bulletin, 37*(1), 1-3. https://doi.org/10.1093/schbul/sbq115

Feldman, H., & **Friston, K.** (2010). Attention, uncertainty, and free-energy. *Frontiers in human neuroscience, 4*, 215. https://doi.org/10.3389/fnhum.2010.00215

Figley, C. R., Uddin, M. N., Wong, K., Kornelsen, J., Puig, J., & Figley, T. D. (2021). Potential pitfalls of using fractional anisotropy, axial diffusivity, and radial diffusivity as biomarkers of cerebral white matter microstructure. *Frontiers in Neuroscience*, 1855. https://doi.org/10.3389/fnins.2021.799576

Filippi, M., Canu, E., Gasparotti, R., Agosta, F., Valsecchi, P., Lodoli, G., ... & Sacchetti, E. (2014). Patterns of brain structural changes in first-contact, antipsychotic drug-naive patients with schizophrenia. *American Journal of Neuroradiology, 35*(1), 30-37. https://doi.org/10.3174/ajnr.A3583

Fink, G. R., Marshall, J. C., Halligan, P. W., Frith, C. D., Driver, J., Frackowiak, R. S., & Dolan, R. J. (1999). The neural consequences of conflict between intention and the senses. *Brain, 122*(3), 497–512. https://doi.org/10.1093/brain/122.3.497

Fitch, R. H., Miller, S., & Tallal, P. (1997). Neurobiology of speech perception. *Annual review of neuroscience, 20*(1), 331-353. http://doi.org/10.1146/annurev.neuro.20.1.331

Fitzsimmons, J., Kubicki, M., & Shenton, M. E. (2013). Review of functional and anatomical brain connectivity findings in schizophrenia. *Current opinion in psychiatry, 26*(2), 172-187. http://doi.org/10.1097/YCO.0b013e32835d9e6a

Flament, D., Ellermann, J. M., Kim, S. G., Ugurbil, K., & Ebner, T. J. (1996). Functional magnetic resonance imaging of cerebellar activation during the learning of a visuomotor dissociation task. *Human Brain Mapping, 4*(3), 210–226. https://doi.org/10.1002/hbm.460040302

Fletcher, P. C., & **Frith, C. D.** (2009). Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. *Nature Reviews Neuroscience, 10*(1), 48-58. https://doi.org/10.1038/nrn2536

Floreano, D., Ijspeert, A. J., & Schaal, S. (2014). Robotics and neuroscience. *Current Biology, 24*(18), R910-R920. https://doi.org/10.1016/j.cub.2014.07.058

Fonov, V. S., Evans, A. C., McKinstry, R. C., Almli, C. R., & Collins, D. L. (2009). Unbiased nonlinear average age-appropriate brain templates from birth to adulthood. *NeuroImage, 47*, S102. https://doi.org/10.1016/S1053-8119(09)70884-5

Ford, J. M., Mathalon, D. H., Heinks, T., Kalba, S., Faustman, W. O., & Roth, W. T. (2001a). Neurophysiological evidence of corollary discharge dysfunction in schizophrenia. *American Journal of Psychiatry, 158*(12), 2069-2071. https://doi.org/10.1176/appi.ajp.158.12.2069

Ford, J. M., Mathalon, D. H., Kalba, S., Whitfield, S., Faustman, W. O., & Roth, W. T. (2001b). Cortical responsiveness during talking and listening in schizophrenia: an event-related brain potential study. *Biological psychiatry, 50*(7), 540-549. https://doi.org/10.1016/S0006-3223(01)01166-0

Ford, J. M., & **Mathalon, D. H.** (2004). Electrophysiological evidence of corollary discharge dysfunction in schizophrenia during talking and thinking. *Journal of psychiatric research, 38*(1), 37-46. https://doi.org/10.1016/S0022-3956(03)00095-5

Ford, J. M., & **Mathalon, D. H.** (2005). Corollary discharge dysfunction in schizophrenia: can it explain auditory hallucinations?. *International Journal of Psychophysiology, 58*(2-3), 179-189. https://doi.org/10.1016/j.ijpsycho.2005.01.014

Ford, J. M., Gray, M., Faustman, W. O., Roach, B. J., & Mathalon, D. H. (2007). Dissecting corollary discharge dysfunction in schizophrenia. *Psychophysiology, 44*(4), 522-529. https://doi.org/10.1111/j.1469-8986.2007.00533.x

Ford, J. M., Roach, B. J., Faustman, W. O., & Mathalon, D. H. (2007). Synch before you speak: auditory hallucinations in schizophrenia. *American Journal of Psychiatry, 164*(3), 458-466. https://doi.org/10.1176/appi.ajp.164.3.458

Ford, J. M., Mathalon, D. H., Roach, B. J., Keedy, S. K., Reilly, J. L., Gershon, E. S., & Sweeney, J. A. (2013). Neurophysiological evidence of corollary discharge function during vocalization in psychotic patients and their nonpsychotic first-degree relatives. *Schizophrenia bulletin, 39*(6), 1272-1280. https://doi.org/10.1093/schbul/sbs129

Ford, J. M., Palzes, V. A., Roach, B. J., & Mathalon, D. H. (2014). Did I do that? Abnormal predictive processes in schizophrenia when button pressing to deliver a tone. *Schizophrenia bulletin, 40*(4), 804-812. https://doi.org/10.1093/schbul/sbt072

Ford, J. M., & **Mathalon, D. H.** (2019). Efference copy, corollary discharge, predictive coding, and psychosis. Biological Psychiatry: *Cognitive Neuroscience and Neuroimaging, 4*(9), 764-767. https://doi.org/10.1016/j.bpsc.2019.07.005

Formisano, E., De Martino, F., Bonte, M., & Goebel, R. (2008). " Who" is saying" what"? Brain-based decoding of human voice and speech. *Science, 322*(5903), 970-973. https://doi.org/10.1126/science.1164318

Fornito, A., & **Bullmore, E. T.** (2015). Reconciling abnormalities of brain network structure and function in schizophrenia. *Current opinion in neurobiology, 30*, 44-50. https://doi.org/10.1016/j.conb.2014.08.006

Fotopoulou, A., & **Tsakiris, M.** (2017). Mentalizing homeostasis: The social origins of interoceptive inference. *Neuropsychoanalysis, 19*(1), 3-28. https://doi.org/10.3389/fpsyg.2016.01272

Fox, M. D., Corbetta, M., Snyder, A. Z., Vincent, J. L., & Raichel, M. E. (2006). Spontaneous neuronal activity distinguishes human dorsal and ventral attention systems. *Proceedings of the National Academy of Sciences, 103*(26), 10046-10051. https://doi.org/10.1073/pnas.0604187103

Friedrich, P., Fraenz, C., Schlüter, C., Ocklenburg, S., Mädler, B., Güntürkün, O., & Genç, E. (2020). The relationship between axon density, myelination, and fractional anisotropy in the human corpus callosum. *Cerebral Cortex, 30*(4), 2042-2056. https://doi.org/10.1093/cercor/bhz221

Friston, K. J., Frith, C. D., Passingham, R. E., Liddle, P. F., & Frackowiak, R. S. J. (1992). Motor practice and neurophysiological adaptation in the cerebellum: A positron tomography study. *Proceedings of the Royal Society of London B, 248*(1323), 223–228. https://doi.org/10.1098/rspb.1992.0065

Friston, K. J., & Frith, C. D. (1995). Schizophrenia: a disconnection syndrome. Clin Neurosci, 3(2), 89-97.

Friston, K. (2002). Functional integration and inference in the brain. *Progress in neurobiology, 68*(2), 113-143. https://www.fil.ion.ucl.ac.uk/~karl/Schizophrenia%20a%20disconnection%20syndrome.pdf

Friston, K. (2003). Learning and inference in the brain. *Neural Networks, 16*(9), 1325-1352. https://doi.org/10.1016/j.neunet.2003.06.005

Friston, K. (2005a). A theory of cortical responses. *Philosophical transactions of the Royal Society B: Biological sciences, 360*(1456), 815-836. https://dx.doi.org/10.1098%2Frstb.2005.1622

Friston, K. J. (2005b). Hallucinations and perceptual inference. *Behav Brain Sci, 28*(6), 764-766. https://doi.org/10.1017/S0140525X05290131

Friston, K., Kilner, J., & Harrison, L. (2006). A free energy principle for the brain. *Journal of physiology-Paris, 100*(1-3), 70-87. https://doi.org/10.1016/j.jphysparis.2006.10.001

Friston, K. J., & **Stephan, K. E.** (2007). Free-energy and the brain. *Synthese, 159*(3), 417-458. https://doi.org/10.1007/s11229-007-9237-y

Friston, K. J., Daunizeau, J., & Kiebel, S. J. (2009). Reinforcement learning or active inference?. *PloS one, 4*(7), e6421. https://doi.org/10.1371/journal.pone.0006421

Friston, K. (2010). The free-energy principle: a unified brain theory?. *Nature reviews neuroscience, 11*(2), 127-138. https://doi.org/10.1038/nrn2787

Friston, K. (2011a). Embodied inference: or "I think therefore I am, if I am what I think" In W. Tschacher & C. Bergomi (Eds.), *The implications of embodiment: Cognition and communication* (pp. 89–125). Imprint Academic. https://www.fil.ion.ucl.ac.uk/~karl/Embodied%20Inference.pdf

Friston, K. J. (2011b). Functional and effective connectivity: a review. *Brain connectivity, 1*(1), 13-36. https://doi.org/10.1089/brain.2011.0008

Friston, K., Mattout, J., & Kilner, J. (2011). Action understanding and active inference. *Biological cybernetics, 104*(1), 137-160. https://doi.org/10.1007/s00422-011-0424-z

Friston, K. (2012a). The history of the future of the Bayesian brain. *NeuroImage, 62*(2), 1230-1233. https://doi.org/10.1016/j.neuroimage.2011.10.004

Friston, K. (2012b). Prediction, perception and agency. *International Journal of Psychophysiology, 83*(2), 248-252. https://doi.org/10.1016/j.ijpsycho.2011.11.014

Friston, K. J., Shiner, T., FitzGerald, T., Galea, J. M., Adams, R., Brown, H., ... & Bestmann, S. (2012c). Dopamine, affordance and active inference. *PLoS computational biology, 8*(1), e1002327. https://doi.org/10.1371/journal.pcbi.1002327

Friston, K., Schwartenbeck, P., FitzGerald, T., Moutoussis, M., Behrens, T., & Dolan, R. J. (2013). The anatomy of choice: active inference and agency. *Frontiers in human neuroscience, 7*, 598. https://doi.org/10.3389/fnhum.2013.00598

Friston, K., & **Frith, C.** (2015). A duet for one. *Consciousness and cognition*, 36, 390-405. https://doi.org/10.1016/j.concog.2014.12.003

Friston, K., Brown, H. R., Siemerkus, J., & Stephan, K. E. (2016). The dysconnection hypothesis (2016). *Schizophrenia research, 176*(2-3), 83-94. https://doi.org/10.1016/j.schres.2016.07.014

Friston, K. J., Lin, M., Frith, C. D., Pezzulo, G., Hobson, J. A., & Ondobaka, S. (2017). Active inference, curiosity and insight. *Neural computation, 29*(10), 2633-2683. https://doi.org/10.1162/neco_a_00999

Frith, C. D. (1979). Consciousness, information processing and schizophrenia. *The British Journal of Psychiatry, 134*(3), 225-235. http://doi.org/10.1192/bjp.134.3.225

Frith, C. D. (1987). The positive and negative symptoms of schizophrenia reflect impairments in the perception and initiation of action. *Psychological medicine, 17*(3), 631-648. https://doi.org/10.1017/S0033291700025873

Frith, C. D., & **Done, D. J.** (1988). Towards a neuropsychology of schizophrenia. *The British Journal of Psychiatry, 153*(4), 437-443. https://doi.org/10.1192/bjp.153.4.437

Frith, C. D. (1992). The cognitive neuropsychology of schizophrenia. Psychology press. https://doi.org/10.4324/9781315785011

Frith, C. D., Blakemore, S. J., & Wolpert, D. M. (2000). Abnormalities in the awareness and control of action. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 355*(1404), 1771-1788. http://doi.org/10.1098/rstb.2000.0734

Fu, C. H., Vythelingum, G. N., Brammer, M. J., Williams, S. C., Amaro, E., Jr., Andrew, C. M., … McGuire, P. K. (2005). An fMRI study of verbal self monitoring: Neural correlates of auditory verbal feedback. *Cerebral Cortex, 16*(7), 969–977. https://doi.org/10.1093/cercor/bhj039

Fukawa, T., Yoshioka, H., Ozawa, E., & Yoshida, S. (1988). Difference of susceptibility to delayed auditory feedback between stutterers and nonstutterers. *Journal of Speech, Language, and Hearing Research, 31*(3), 475–479. https://doi.org/10.1044/jshr.3103.475

Gainotti, G., Ferraccioli, M., & Marra, C. (2010). The relation between person identity nodes, familiarity judgment and biographical information. Evidence from two patients with right and left anterior temporal atrophy. *Brain Research, 1307*, 103-114. https://doi.org/10.1016/j.brainres.2009.10.009

Gainotti, G., & **Marra, C.** (2011). Differential contribution of right and left temporo-occipital and anterior temporal lesions to face recognition disorders. *Frontiers in Human Neuroscience, 5*, 55. https://doi.org/10.3389/fnhum.2011.00055

Galantucci, B., Fowler, C. A., & Turvey, M. T. (2006). The motor theory of speech perception reviewed. *Psychonomic bulletin & review, 13*(3), 361-377. https://doi.org/10.3758/BF03193857

Galea, J. M., Vazquez, A., Pasricha, N., Orban de Xivry, J. J., & Celnik, P. (2010). Dissociating the roles of the cerebellum and motor cortex during adaptive learning: the motor cortex retains what the cerebellum learns. *Cerebral cortex, 21*(8), 1761-1770. https://doi.org/10.1093/cercor/bhq246

Gaudrain, E., Li, S., Ban, V. S., & Patterson, R. (2009, September). The role of glottal pulse rate and vocal tract length in the perception of speaker identity. In Interspeech 2009. https://www.isca-speech.org/archive_v0/archive_papers/interspeech_2009/papers/i09_0148.pdf

Gauthier, C. J., & **Fan, A. P.** (2019). BOLD signal physiology: models and applications. *Neuroimage, 187*, 116-127. https://doi.org/10.1016/j.neuroimage.2018.03.018

Gershman, S. J. (2019). The generative adversarial brain. *Frontiers in Artificial Intelligence*, 18. https://doi.org/10.3389/frai.2019.00018

Geva, S., & **Fernyhough, C.** (2019). A penny for your thoughts: children's inner speech and its neuro-development. *Frontiers in Psychology, 10*, 1708. https://doi.org/10.3389/fpsyg.2019.01708

Ghazanfar, A. A., & **Hauser, M. D.** (1999). The neuroethology of primate vocal communication: substrates for the evolution of speech. *Trends in cognitive sciences, 3*(10), 377-384. https://doi.org/10.1016/S1364-6613(99)01379-0

Gilbert, P. F. C., & **Thach, W. T.** (1977). Purkinje cell activity during motor learning. *Brain Research, 128*(2), 309–328. https://doi.org/10.1016/0006-8993(77)90997-0

Giovannucci, A., Badura, A., Deverett, B., Najafi, F., Pereira, T. D., Gao, Z., … De Zeeuw, C. I. (2017). Cerebellar granule cells acquire a widespread predictive feedback signal during motor learning. *Nature Neuroscience, 20*(5), 727–734. https://doi.org/10.1038/nn.4531

Glickstein, M., Sultan, F., & Voogd, J. (2011). Functional localization in the cerebellum. *Cortex, 1*(47), 59-80. https://doi.org/10.1016/j.cortex.2009.09.001

Goff, D. C., & **Coyle, J. T.** (2001). The emerging role of glutamate in the pathophysiology and treatment of schizophrenia. *American Journal of Psychiatry, 158*(9), 1367-1377. https://doi.org/10.1176/appi.ajp.158.9.1367

Goldberg, G. (1985). Supplementary motor area structure and function: review and hypotheses. *Behavioral and brain Sciences, 8*(4), 567-588. https://doi.org/10.1017/S0140525X00045167

Golfinopoulos, E., Tourville, J. A., & Guenther, F. H. (2010). The integration of large-scale neural network modeling and functional brain imaging in speech motor control. *Neuroimage, 52*(3), 862-874. https://doi.org/10.1016/j.neuroimage.2009.10.023

Golfinopoulos, E., Tourville, J. A., Bohland, J. W., Ghosh, S. S., Nieto-Castanon, A., & Guenther, F. H. (2011). fMRI investigation of unexpected somatosensory feedback perturbation during speech. *Neuroimage, 55*(3), 1324-1338. https://doi.org/10.1016/j.neuroimage.2010.12.065

Gomi, H., & **Kawato, M.** (1993). Neural network control for a closed-loop system using feedback-error-learning. *Neural Networks, 6*(7), 933-946. https://doi.org/10.1016/S0893-6080(09)80004-X

Gomi, H., Honda, M., Ito, T., & Murano, E. Z. (2002). Compensatory articulation during bilabial fricative production by regulating muscle stiffness. *Journal of Phonetics, 30*(3), 261–279. https://doi.org/10.1006/jpho.2002.0173

Goulden, N., Khusnulina, A., Davis, N. J., Bracewell, R. M., Bokde, A. L., McNulty, J. P., & Mullins, P. G. (2014). The salience network is responsible for switching between the default mode network and the central executive network: replication from DCM. *Neuroimage, 99*, 180-190. https://doi.org/10.1016/j.neuroimage.2014.05.052

Gouvêa-Junqueira, D., Falvella, A. C. B., Antunes, A. S. L. M., Seabra, G., Brandão-Teles, C., Martins-de-Souza, D., & Crunfli, F. (2020). Novel treatment strategies targeting myelin and oligodendrocyte dysfunction in schizophrenia. *Frontiers in Psychiatry, 11*, 379. https://doi.org/10.3389/fpsyt.2020.00379

Grafton, S. T., & **Hamilton, A. F. D. C.** (2007). Evidence for a distributed hierarchy of action representation in the brain. *Human movement science, 26*(4), 590-616. https://doi.org/10.1016/j.humov.2007.05.009

Grafton, S. T., Schmitt, P., Van Horn, J., & Diedrichsen, J. (2008). Neural substrates of visuomotor learning based on improved feedback control and prediction. *NeuroImage, 39*(3), 1383–1395. https://doi.org/10.1016/j.neuroimage.2007.09.062

Grandjean, D., Sander, D., Pourtois, G., Schwartz, S., Seghier, M. L., Scherer, K. R., & Vuilleumier, P. (2005). The voices of wrath: brain responses to angry prosody in meaningless speech. *Nature neuroscience, 8*(2), 145-146. https://doi.org/10.1038/nn1392

Grandjean, D. (2021). Brain networks of emotional prosody processing. *Emotion Review, 13*(1), 34-43. https://doi.org/10.1177/1754073919898522

Graydon, F. X., Friston, K. J., Thomas, C. G., Brooks, V. B., & Menon, R. S. (2005). Learning-related fMRI activation associated with a rotational visuo-motor transformation. *Cognitive Brain Research, 22*(3), 373–383. https://doi.org/10.1016/j.cogbrainres.2004.09.007

Greenlee, J. D., Jackson, A. W., Chen, F., Larson, C. R., Oya, H., Kawasaki, H., ... & Howard III, M. A. (2011). Human auditory cortical activation during self-vocalization. *PloS One, 6*(3). https://doi.org/10.1371/journal.pone.0014744

Gregory, R. L. (1980). Perceptions as hypotheses. *Philosophical Transactions of the Royal Society of London. B, Biological Sciences, 290*(1038), 181-197. https://doi.org/10.1098/rstb.1980.0090

Gregory, D. (2016). Inner speech, imagined speech, and auditory verbal hallucinations. *Review of Philosophy and Psychology, 7*(3), 653-673. https://doi.org/10.1007/s13164-015-0274-z

Griffin, J. D., & **Fletcher, P. C.** (2017). Predictive processing, source monitoring, and psychosis. *Annual Review of Clinical Psychology, 13*, 265-289. https://doi.org/10.1146/annurev-clinpsy-032816-045145

Griffiths, T. D., & **Warren, J. D.** (2004). What is an auditory object?. *Nature Reviews Neuroscience, 5*(11), 887-892. https://doi.org/10.1038/nrn1538

Grill-Spector, K., Henson, R., & Martin, A. (2006). Repetition and the brain: neural models of stimulus-specific effects. *Trends in cognitive sciences, 10*(1), 14-23. https://doi.org/10.1016/j.tics.2005.11.006

Grodd, W., Hülsmann, E., Lotze, M., Wildgruber, D., & Erb, M. (2001). Sensorimotor mapping of the human cerebellum: fMRI evidence of somatotopic organization. *Human brain mapping, 13*(2), 55-73. https://doi.org/10.1002/hbm.1025

Grover, S., Dual, D., Chakrabarti, S., & Avasthi, A. (2018). Factor analysis of symptom dimensions (psychotic, affective and obsessive compulsive symptoms) in schizophrenia. *Asian journal of psychiatry, 38*, 72-77. https://doi.org/10.1016/j.ajp.2017.10.028

Grube, B. S., Bilder, R. M., & Goldman, R. S. (1998). Meta-analysis of symptom factors in schizophrenia. *Schizophrenia Research, 31*(2-3), 113-120. https://doi.org/10.1016/S0920-9964(98)00011-5

Griffiths, T. D., & **Warren, J. D.** (2004). What is an auditory object?. *Nature Reviews Neuroscience, 5*(11), 887-892. https://doi.org/10.1038/nrn1538

Guell, X., & **Schmahmann, J.** (2020). Cerebellar functional anatomy: a didactic summary based on human fMRI evidence. *The Cerebellum, 19*(1), 1-5. https://doi.org/10.1007/s12311-019-01083-9

Guenther, F. H. (1994). A neural network model of speech acquisition and motor equivalent speech production. *Biological cybernetics, 72*(1), 43-53. https://doi.org/10.1007/BF00206237

Guenther, F. H. (1995). Speech sound acquisition, coarticulation, and rate effects in a neural network model of speech production. *Psychological review, 102*(3), 594. https://doi.org/10.1037/0033-295X.102.3.594

Guenther, F. H. (2006). Cortical interactions underlying the production of speech sounds. *Journal of communication disorders, 39*(5), 350-365. https://doi.org/10.1016/j.jcomdis.2006.06.013

Guenther, F. H., Ghosh, S. S., & Tourville, J. A. (2006). Neural modeling and imaging of the cortical interactions underlying syllable production. *Brain and language, 96*(3), 280-301. https://doi.org/10.1016/j.bandl.2005.06.001

Guenther, F. H., & **Vladusich, T.** (2012). A neural theory of speech acquisition and production. *Journal of neurolinguistics, 25*(5), 408-422. https://doi.org/10.1016/j.jneuroling.2009.08.006

Guo, Z., Huang, X., Wang, M., Jones, J. A., Dai, Z., Li, W., ... & Liu, H. (2016). Regional homogeneity of intrinsic brain activity correlates with auditory-motor processing of vocal pitch errors. *Neuroimage, 142*, 565-575. https://doi.org/10.1016/j.neuroimage.2016.08.005

Habas, C., Kamdar, N., Nguyen, D., Prater, K., Beckmann, C. F. Menon, V., & Greicius, M. D. (2009). Distinct cerebellar contributions to intrinsic connectivity networks. *Journal of Neuroscience, 29*(26), 8586–8594. https://doi.org/10.1523/JNEUROSCI.1868-09.2009

Haggard, P., & **Whitford, B.** (2004). Supplementary motor area provides an efferent signal for sensory suppression. *Cognitive Brain Research, 19*(1), 52–58. https://doi.org/10.1016/j.cogbrainres.2003.10.018

Haggard, P. (2005). Conscious intention and motor cognition. *Trends in cognitive sciences, 9*(6), 290-295. https://doi.org/10.1016/j.tics.2005.04.012

Haggard, P. (2017). Sense of agency in the human brain. *Nature Reviews Neuroscience, 18*(4), 196-207. https://doi.org/10.1038/nrn.2017.14

Hall, D. A., Haggard, M. P., Akeroyd, M. A., Palmer, A. R., Summerfield, A. Q., Elliott, M. R., ... & Bowtell, R. W. (1999). "Sparse" temporal sampling in auditory fMRI. *Human Brain Mapping, 7*(3), 213-223. https://doi.org/10.1002/(SICI)1097-0193(1999)7:3<213::AID-HBM5>3.0.CO;2-N

Halsband, U., & **Lange, R. K.** (2006). Motor learning in man: a review of functional and clinical studies. *Journal of Physiology-Paris, 99*(4-6), 414-424. https://doi.org/10.1016/j.jphysparis.2006.03.007

Hailstone, J. C., Ridgway, G. R., Bartlett, J. W., Goll, J. C., Buckley, A. H., Crutch, S. J., & Warren, J. D. (2011). Voice processing in dementia: a neuropsychological and neuroanatomical analysis. *Brain, 134*(9), 2535-2547. https://doi.org/10.1093/brain/awr205

Hain, T. C., Burnett, T. A., Kiran, S., Larson, C. R., Singh, S., & Kenney, M. K. (2000). Instructing subjects to make a voluntary response reveals the presence of two components to the audio-vocal reflex. *Experimental Brain Research, 130*(2), 133–141. https://doi.org/10.1007/s002219900237

Hampshire, A., Chamberlain, S. R., Monti, M. M., Duncan, J., & Owen, A. M. (2010). The role of the right inferior frontal gyrus: Inhibition and attentional control. *NeuroImage, 50*(3), 1313–1319. https://doi.org/10.1016/j.neuroimage.2009.12.109

Hardwick, R. M., Rottschy, C., Miall, R. C., & Eickhoff, S. B. (2013). A quantitative meta-analysis and review of motor learning in the human brain. *Neuroimage, 67*, 283-297. https://doi.org/10.1016/j.neuroimage.2012.11.020

Hare, S. M. (2021). Hallucinations: A Functional Network Model of How Sensory Representations Become Selected for Conscious Awareness in Schizophrenia. *Frontiers in Neuroscience, 15*, 733038. http://doi.org/10.3389/fnins.2021.733038

Haruno, M., Wolpert, D. M., & Kawato, M. (2001). Mosaic model for sensorimotor learning and control. *Neural computation, 13*(10), 2201-2220. http://doi.org/10.1162/089976601750541778

Haruno, M., Wolpert, D. M., & Kawato, M. (2003). Hierarchical MOSAIC for movement generation. *In International Congress Series, 1250*, 575-590. https://doi.org/10.1016/S0531-5131(03)00190-0

Hashimoto, Y., & **Sakai, K. L.** (2003). Brain activations during conscious self-monitoring of speech production with delayed auditory feedback: An fMRI study. *Human Brain Mapping, 20*(1), 22–28. https://doi.org/10.1002/hbm.10119

Hau, J., Sarubbo, S., Perchey, G., Crivello, F., Zago, L., Mellet, E., ... & Petit, L. (2016). Cortical terminations of the inferior fronto-occipital and uncinate fasciculi: anatomical stem-based virtual dissection. *Frontiers in neuroanatomy, 10*, 58. https://doi.org/10.3389/fnana.2016.00058

Hau, J., Sarubbo, S., Houde, J. C., Corsini, F., Girard, G., Deledalle, C., ... & Petit, L. (2017). Revisiting the human uncinate fasciculus, its subcomponents and asymmetries with stem-based tractography and microdissection validation. *Brain Structure and Function, 222*(4), 1645-1662. http://doi.org/10.1007/s00429-016-1298-6

Hauser, M. D., Chomsky, N., & Fitch, W. T. (2002). The faculty of language: what is it, who has it, and how did it evolve? *Science, 298*(5598), 1569-1579. http://doi.org/10.1126/science.298.5598.1569

Havlicek, L. L. (1968). Effect of delayed auditory feedback on musical performance. *Journal of Research in Music Education, 16*(4), 308–318. https://doi.org/10.2307/3344070

Havsteen, I., Ohlhues, A., Madsen, K. H., Nybing, J. D., Christensen, H., & Christensen, A. (2017). Are movement artifacts in magnetic resonance imaging a real problem?—a narrative review. *Frontiers in neurology, 8*, 232. https://doi.org/10.3389/fneur.2017.00232

Healy, C., Brannigan, R., Dooley, N., Coughlan, H., Clarke, M., Kelleher, I., & Cannon, M. (2019). Childhood and adolescent psychotic experiences and risk of mental disorder: a systematic review and meta-analysis. *Psychological Medicine, 49*(10), 1589-1599. https://doi.org/10.1017/S0033291719000485

Healy, C., & **Cannon, M.** (2020). Psychotic-like experiences in the general population. In Risk Factors for Psychosis (pp. 119-141). Academic Press. https://doi.org/10.1016/B978-0-12-813201-2.00007-7

Heeger, D. J., & **Ress, D.** (2002). What does fMRI tell us about neuronal activity?. *Nature reviews neuroscience, 3*(2), 142-151. https://doi.org/10.1038/nrn730

Heilbron, M., & **Chait, M.** (2018). Great expectations: is there evidence for predictive coding in auditory cortex?. *Neuroscience, 389*, 54-73. https://doi.org/10.1016/j.neuroscience.2017.07.061

Heinks-Maldonado, T. H., Mathalon, D. H., Gray, M., & Ford, J. M. (2005). Fine-tuning of auditory cortex during speech production. *Psychophysiology, 42*(2), 180–190. https://doi.org/10.1111/j.1469-8986.2005.00272.x

Heinks-Maldonado, T. H., Mathalon, D. H., Houde, J. F., Gray, M., Faustman, W. O., & Ford, J. M. (2007). Relationship of imprecise corollary discharge in schizophrenia to auditory hallucinations. *Archives of General Psychiatry, 64*(3), 286-296. https://doi.org/10.1001/archpsyc.64.3.286

Heinz, A., Murray, G. K., Schlagenhauf, F., Sterzer, P., Grace, A. A., & Waltz, J. A. (2019). Towards a unifying cognitive, neurophysiological, and computational neuroscience account of schizophrenia. *Schizophrenia bulletin, 45*(5), 1092-1100. https://doi.org/10.1093/schbul/sby154

Hernandez-Fernandez, M., Reguly, I., Jbabdi, S., Giles, M., Smith, S., & Sotiropoulos, S. N. (2019). Using GPUs to accelerate computational diffusion MRI: From microstructure estimation to tractography and connectomes. *Neuroimage, 188*, 598-615. https://doi.org/10.1016/j.neuroimage.2018.12.015

Hickok, G., Houde, J., & Rong, F. (2011). Sensorimotor integration in speech processing: computational basis and neural organization. *Neuron, 69*(3), 407-422. https://doi.org/10.1016/j.neuron.2011.01.019

Hickok, G. (2012). Computational neuroanatomy of speech production. *Nature reviews neuroscience, 13*(2), 135-145. https://doi.org/10.1038/nrn3158

Hobson, J. A., & **Friston, K. J.** (2014). Consciousness, dreams, and inference: The Cartesian theatre revisited. *Journal of Consciousness Studies, 21*(1-2), 6-32. https://www.ingentaconnect.com/contentone/imp/jcs/2014/00000021/f0020001/art00001

Hofer, S., & **Frahm, J.** (2006). Topography of the human corpus callosum revisited—comprehensive fiber tractography using diffusion tensor magnetic resonance imaging. *Neuroimage, 32*(3), 989-994. https://doi.org/10.1016/j.neuroimage.2006.05.044

Hoffman, K. L., & **Logothetis, N. K.** (2009). Cortical mechanisms of sensory learning and object recognition. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*(1515), 321-329. https://doi.org/10.1098/rstb.2008.0271

Hofstötter, C., Mintz, M., & Verschure, P. F. (2002). The cerebellum in action: a simulation and robotics study. *The European journal of neuroscience, 16*(7), 1361-1376. https://doi.org/10.1046/j.1460-9568.2002.02182.x

Hohwy, J. (2014). The neural organ explains the mind. In Open mind. Open MIND. Frankfurt am Main: MIND Group. http://doi.org/10.15502/9783958570016

Hohwy, J. (2016). The self-evidencing brain. *Noûs, 50*(2), 259-285. http://doi.org/10.1111/nous.12062

Hohwy, J. (2017). Priors in perception: Top-down modulation, Bayesian perceptual learning rate, and prediction error minimization. *Consciousness and Cognition, 47*, 75-85. https://doi.org/10.1016/j.concog.2016.09.004

Horga, G., & **Abi-Dargham, A.** (2019). An integrative framework for perceptual disturbances in psychosis. *Nature Reviews Neuroscience, 20*(12), 763-778. https://doi.org/10.1038/s41583-019-0234-1

Holmes, C. J., Hoge, R., Collins, L., Woods, R., Toga, A. W., & Evans, A. C.(1998). Enhancement of MR images using registration for signal averaging. *Journal of Computer Assisted Tomography, 22*(2), 324–333. https://doi.org/10.1097/00004728-199803000-00032

Honda, M., Fujino, A., & Kaburagi, T. (2002). Compensatory responses of articulators to unexpected perturbation of the palate shape. *Journal of Phonetics, 30*(3), 281–302. https://doi.org/10.1006/jpho.2002.0172

Honey, C. J., Sporns, O., Cammoun, L., Gigandet, X., Thiran, J. P., Meuli, R., & Hagmann, P. (2009). Predicting human resting-state functional connectivity from structural connectivity. *Proceedings of the National Academy of Sciences, 106*(6), 2035-2040. https://doi.org/10.1073/pnas.0811168106

Horn, K. M., Pong, M., & Gibson, A. R. (2004). Discharge of inferior olive cells during reaching errors and perturbations. *Brain Research, 996*(2), 148–158. https://doi.org/10.1016/j.brainres.2003.10.021

Houde, J. F., & **Jordan, M. I.** (1998). Sensorimotor adaptation in speech production. *Science, 279*(5354), 1213–1216. https://doi.org/10.1126/science.279.5354.1213

Houde, J. F., & **Jordan, M. I.** (2002). Sensorimotor adaptation of speech I: Compensation and adaptation. *Journal of Speech, Language, and Hearing Research, 45*(2), 295–310. https://doi.org/10.1044/1092-4388(2002/023)

Houde, J. F., Nagarajan, S. S., Sekihara, K., & Merzenich, M. M. (2002). Modulation of the auditory cortex during speech: An MEG study. *Journal of Cognitive Neuroscience, 14*(8), 1125–1138. https://doi.org/10.1162/089892902760807140

Houde, J. F., & **Nagarajan, S. S.** (2011). Speech production as state feedback control. *Frontiers in human neuroscience, 5*, 82. https://doi.org/10.3389/fnhum.2011.00082

Howell, P., & **Powell, D. J.** (1987). Delayed auditory feedback with delayed sounds varying in duration. *Perception & Psychophysics, 42*(2), 166–172. https://doi.org/10.3758/BF03210505

Howes, O., McCutcheon, R., & Stone, J. (2015). Glutamate and dopamine in schizophrenia: an update for the 21st century. *Journal of psychopharmacology, 29*(2), 97-115. https://doi.org/10.1177/0269881114563634

Howes, O. D., & **Nour, M. M.** (2016). Dopamine and the aberrant salience hypothesis of schizophrenia. *World Psychiatry, 15*(1), 3. http://doi.org/10.1002/wps.20276

Howes, O. D., McCutcheon, R., Owen, M. J., & Murray, R. M. (2017). The role of genes, stress, and dopamine in the development of schizophrenia. *Biological psychiatry, 81*(1), 9-20. https://doi.org/10.1016/j.biopsych.2016.07.014

Howes, O. D., Hird, E. J., Adams, R. A., Corlett, P. R., & McGuire, P. (2020). Aberrant salience, information processing, and dopaminergic signaling in people at clinical high risk for psychosis. *Biological psychiatry, 88*(4), 304-314. https://doi.org/10.1016/j.biopsych.2020.03.012

Hoxha, E., Tempia, F., Lippiello, P., & Miniaci, M. C. (2016). Modulation, plasticity and pathophysiology of the parallel fiber-purkinje cell synapse. *Frontiers in synaptic neuroscience, 8*, 35. https://doi.org/10.3389/fnsyn.2016.00035

Hickok, G., & **Poeppel, D.** (2000). Towards a functional neuroanatomy of speech perception. *Trends in cognitive sciences, 4*(4), 131-138. https://doi.org/10.1016/S1364-6613(00)01463-7

Hickok, G., & **Poeppel, D.** (2004). Dorsal and ventral streams: a framework for understanding aspects of the functional anatomy of language. *Cognition, 92*(1-2), 67-99. https://doi.org/10.1016/j.cognition.2003.10.011

Hickok, G., & **Poeppel, D.** (2007). The cortical organization of speech processing. *Nature reviews neuroscience, 8*(5), 393-402. https://doi.org/10.1038/nrn2113

Hickok, G., Holt, L. L., & Lotto, A. J. (2009). Response to Wilson: What does motor cortex contribute to speech perception?. *Trends in Cognitive Sciences, 13*(8), 330-331. http://doi.org/10.1016/j.tics.2009.05.002

Hickok, G., Houde, J., & Rong, F. (2011). Sensorimotor integration in speech processing: computational basis and neural organization. *Neuron, 69*(3), 407-422. https://doi.org/10.1016/j.neuron.2011.01.019

Hinton, G. E. (2007). Learning multiple layers of representation. *Trends in cognitive sciences, 11*(10), 428-434. https://doi.org/10.1016/j.tics.2007.09.004

Hu, M. L., Zong, X. F., Mann, J. J., Zheng, J. J., Liao, Y. H., Li, Z. C., ... & Tang, J. S. (2017). A review of the functional and anatomical default mode network in schizophrenia. *Neuroscience bulletin, 33*(1), 73-84. http://doi.org/10.1007/s12264-016-0090-1

Huang, Y., & **Rao, R. P.** (2011). Predictive coding. Wiley Interdisciplinary Reviews: *Cognitive Science, 2*(5), 580-593. https://doi-org.mu.idm.oclc.org/10.1002/wcs.142

Hubl, D., Koenig, T., Strik, W., Federspiel, A., Kreis, R., Boesch, C., ... & Dierks, T. (2004). Pathways that make voices: white matter changes in auditory hallucinations. *Archives of general psychiatry, 61*(7), 658-668. http://doi.org/10.1001/archpsyc.61.7.658

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

Hugdahl, K., Løberg, E. M., & Nygård, M. (2009). Left temporal lobe structural and functional abnormality underlying auditory hallucinations. *Frontiers in neuroscience, 3*, 1. https://doi.org/10.3389/neuro.01.001.2009

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

Hugdahl, K., Craven, A. R., Nygård, M., Løberg, E. M., Berle, J. Ø., Johnsen, E., ... & Ersland, L. (2015). Glutamate as a mediating transmitter for auditory hallucinations in schizophrenia: A 1H MRS study. *Schizophrenia research, 161*(2-3), 252-260. https://doi.org/10.1016/j.schres.2014.11.015

Hugdahl, K. (2017). Auditory hallucinations as translational psychiatry: Evidence from magnetic resonance imaging. *Balkan medical journal, 34*(6), 504. http://doi.org/10.4274/balkanmedj.2017.1226

Hugdahl, K., & **Sommer, I. E.** (2018). Auditory verbal hallucinations in schizophrenia from a levels of explanation perspective. *Schizophrenia bulletin, 44*(2), 234-241. https://doi.org/10.1093/schbul/sbx142

Hughes, S. M., & **Nicholson, S. E.** (2010). The processing of auditory and visual recognition of self-stimuli. *Consciousness and Cognition, 19*(4), 1124-1134. https://doi.org/10.1016/j.concog.2010.03.001

Hunt, M. J., Kopell, N. J., Traub, R. D., & Whittington, M. A. (2017). Aberrant network activity in schizophrenia. *Trends in neurosciences, 40*(6), 371-382. https://doi.org/10.1016/j.tins.2017.04.003

Imamizu, H., Miyauchi, S., Tamada, T., Sasaki, Y., Takino, R., Pütz, B., … Kawato, M. (2000). Human cerebellar activity reflecting an acquired internal model of a new tool. *Nature, 403*(6766), 192–195. https://doi.org/10.1038/35003194

Inoue, K., Kawashima, R., Satoh, K., Kinomura, S., Sugiura, M., Goto, R., … Fukuda, H. (2000). A PET study of visuomotor learning under optical rotation. *NeuroImage, 11*(5), 505–516. https://doi.org/10.1006/nimg.2000.0554

Ishikawa, T., Tomatsu, S., Izawa, J., & Kakei, S. (2016). The cerebro-cerebellum: Could it be loci of forward models? *Neuroscience Research, 104*, 72–79. https://doi.org/10.1016/j.neures.2015.12.003

Ishimaru, M. J., & **Toru, M.** (1997). The glutamate hypothesis of schizophrenia. *Cns Drugs, 7*(1), 47-67. https://doi.org/10.2165/00023210-199707010-00006

Ito, M., Sakurai, M., & Tongroach, P. (1982). Climbing fibre induced depression of both mossy fibre responsiveness and glutamate sensitivity of cerebellar Purkinje cells. *The Journal of Physiology, 324*(1), 113–134. https://doi.org/10.1113/jphysiol.1982.sp014103

Ito, M. (1984a). The cerebellum and neural control. New York, NY: Raven Press.

Ito, M. (1984b). The modifiable neuronal network of the cerebellum. *The Japanese Journal of Physiology, 34*(5), 781–792. https://doi.org/10.2170/jjphysiol.34.781

Ito, M. (2000). Mechanisms of motor learning in the cerebellum. *Brain Research, 886*(1–2), 237–245. https://doi.org/10.1016/S0006-8993(00)03142-5

Ito, M. (2005). Bases and implications of learning in the cerebellum—Adaptive control and internal model mechanism. *Progress in Brain Research*, 148, 95–109. https://doi.org/10.1016/S0079-6123(04)48009-1

Ito, M. (2006). Cerebellar circuitry as a neuronal machine. *Progress in neurobiology, 78*(3-5), 272-303. https://doi.org/10.1016/j.pneurobio.2006.02.006

Ito, M. (2008). Control of mental activities by internal models in the cerebellum. *Nature Reviews Neuroscience, 9*(4), 304-313. https://doi.org/10.1038/nrn2332

Iwabuchi, S. J., Liddle, P. F., & Palaniyappan, L. (2015). Structural connectivity of the salience-executive loop in schizophrenia. *European archives of psychiatry and clinical neuroscience, 265*(2), 163-166. http://doi.org/10.1007/s00406-014-0547-z

Izawa, J., Rane, T., Donchin, O., & Shadmehr, R. (2008). Motor adaptation as a process of reoptimization. *Journal of Neuroscience, 28*(11), 2883–2891. https://doi.org/10.1523/JNEUROSCI.5359-07.2008

James, W. (1890/1950). The principles of psychology. New York: Dover http://infomotions.com/sandbox/great-books-redux/corpus/html/principles.html

Jardri, R., Pins, D., Bubrovszky, M., Despretz, P., Pruvo, J. P., Steinling, M., & Thomas, P. (2007). Self awareness and speech processing: An fMRI study. *NeuroImage, 35*(4), 1645–1653. https://doi.org/10.1016/j.neuroimage.2007.02.002

Jardri, R., Pouchet, A., Pins, D., & Thomas, P. (2011). Cortical activations during auditory verbal hallucinations in schizophrenia: a coordinate-based meta-analysis. *American Journal of Psychiatry, 168*(1), 73-81. https://doi.org/10.1176/appi.ajp.2010.09101522

Jardri, R., & **Denève, S.** (2013). Circular inferences in schizophrenia. *Brain, 136*(11), 3227-3241. https://doi.org/10.1093/brain/awt257

Jardri, R., & **Sommer, I.** (2013). Functional brain imaging of hallucinations: symptom capture studies. *The Neuroscience of Hallucinations*, 375-391. https://doi.org/10.1007/978-1-4614-4121-2_20

Jardri, R., Hugdahl, K., Hughes, M., Brunelin, J., Waters, F., Alderson-Day, B., ... & Denève, S. (2016). Are hallucinations due to an imbalance between excitatory and inhibitory influences on the brain?. *Schizophrenia bulletin, 42*(5), 1124-1134. https://doi.org/10.1093/schbul/sbw075

Jayaram, G., Tang, B., Pallegadda, R., Vasudevan, E. V., Celnik, P., & Bastian, A. (2012). Modulating locomotor adaptation with cerebellar stimulation. *Journal of neurophysiology, 107*(11), 2950-2957. https://doi.org/10.1152/jn.00645.2011

Jeannerod, M. (1994). The representing brain: Neural correlates of motor intention and imagery. *Behavioral and Brain sciences, 17*(2), 187-202. https://doi.org/10.1017/S0140525X00034026

Jeannerod, M., & **Pacherie, E.** (2004). Agency, simulation and self-identification. *Mind & language, 19*(2), 113-146 https://doi.org/10.1111/j.1468-0017.2004.00251.x

Jenkinson, M., Bannister, P., Brady, M., & Smith, S. (2002). Improved optimization for the robust and accurate linear registration and motion correction of brain images. *Neuroimage, 17*(2), 825-841. https://doi.org/10.1006/nimg.2002.1132

Johns, L. C., Nazroo, J. Y., Bebbington, P., & Kuipers, E. (1998). Occurrence of hallucinations in a community sample. Schizophrenia Research, 1(29), 23.

Johns, L. C., & **van Os, J.** (2001). The continuity of psychotic experiences in the general population. *Clinical psychology review, 21*(8), 1125-1141. https://doi.org/10.1016/S0272-7358(01)00103-9

Johns, L. C., Rossell, S., Frith, C., Ahmad, F., Hemsley, D., Kuipers, E., & McGuire, P. K. (2001). Verbal self-monitoring and auditory verbal hallucinations in patients with schizophrenia. *Psychological medicine, 31*(4), 705-715. https://doi.org/10.1017/S0033291701003774

Johns, L. C., Gregg, L., Vythelingum, N., & McGuire, P. K. (2003). Establishing the reliability of a verbal self-monitoring paradigm. *Psychopathology, 36*(6), 299-303. https://doi.org/10.1159/000075188

Johns, L. C. (2005). Hallucinations in the general population. *Current psychiatry reports, 7*(3), 162-167. https://doi.org/10.1006/nimg.2002.1132

Johns, L. C., Gregg, L., Allen, P., & McGuire, P. K. (2006). Impaired verbal self-monitoring in psychosis: effects of state, trait and diagnosis. *Psychological Medicine, 36*(4), 465-474. https://doi.org/10.1017/S0033291705006628

Johns, L. C., Kompus, K., Connell, M., Humpston, C., Lincoln, T. M., Longden, E., ... & Larøi, F. (2014). Auditory verbal hallucinations in persons with and without a need for care. *Schizophrenia bulletin, 40*(Suppl_4), S255-S264. https://doi.org/10.1093/schbul/sbu005

Johnsen, E., Hugdahl, K., Fusar-Poli, P., Kroken, R. A., & Kompus, K. (2013). Neuropsychopharmacology of auditory hallucinations: insights from pharmacological functional MRI and perspectives for future research. *Expert review of neurotherapeutics, 13*(1), 23-36. https://doi.org/10.1586/ern.12.147

Johnson, J. F., Belyk, M., Schwartze, M., Pinheiro, A. P., & Kotz, S. A. (2019). The role of the cerebellum in adaptation: ALE meta-analyses on sensory feedback error. *Human Brain Mapping, 40*(13), 3966-3981. https://doi.org/10.1002/hbm.24681

Johnson J.F., Belyk M., Schwartze M., Pinheiro A.P., Kotz S.A. (2021). Expectancy changes the self-monitoring of voice identity. *European Journal of Neuroscience, 53*(8), 2681-2695. https://doi.org/10.1111/ejn.15162

Johnstone, T., Van Reekum, C. M., Oakes, T. R., & Davidson, R. J. (2006). The voice of emotion: an FMRI study of neural responses to angry and happy vocal expressions. *Social cognitive and affective neuroscience, 1*(3), 242-249. https://doi.org/10.1093/scan/nsl027

Jones, J. A., & **Munhall, K. G.** (2000). Perceptual calibration of F0 production: Evidence from feedback perturbation. *The Journal of the Acoustical Society of America, 108*(3), 1246–1251. https://doi.org/10.1121/1.1288414

Jones, S. R., & **Fernyhough, C.** (2007a). Thought as action: Inner speech, self-monitoring, and auditory verbal hallucinations. *Consciousness and cognition, 16*(2), 391-399. https://doi.org/10.1016/j.concog.2005.12.003

Jones, S. R., & **Fernyhough, C.** (2007b). Neural correlates of inner speech and auditory verbal hallucinations: a critical review and theoretical integration. *Clinical psychology review, 27*(2), 140-154. https://doi.org/10.1016/j.cpr.2006.10.001

Jones, S. R., & **Fernyhough, C.** (2009). Rumination, reflection, intrusive thoughts, and hallucination-proneness: Towards a new model. *Behaviour Research and Therapy, 47*(1), 54-59. https://doi.org/10.1016/j.brat.2008.09.008

Jones, S. R. (2010). Do we need multiple models of auditory verbal hallucinations? Examining the phenomenological fit of cognitive and neurological models. *Schizophrenia bulletin, 36*(3), 566-575. https://doi.org/10.1093/schbul/sbn129

Jones, D. K., Knösche, T. R., & Turner, R. (2013). White matter integrity, fiber count, and other fallacies: the do's and don'ts of diffusion MRI. *Neuroimage, 73*, 239-254. https://doi.org/10.1016/j.neuroimage.2012.06.081

Jones, A. B., Farrall, A. J., Belin, P., & Pernet, C. R. (2015). Hemispheric association and dissociation of voice and speech information processing in stroke. *cortex, 71*, 232-239. https://doi.org/10.1016/j.cortex.2015.07.004

Jordan, M. I., & **Rumelhart, D. E.** (1992). Forward models: Supervised learning with a distal teacher. *Cognitive Science, 16*(3), 307–354. https://doi.org/10.1207/s15516709cog1603_1

Jordan, M.I. (1995). Computational motor control. In M. S. Gazzaniga (Eds.), The cognitive neurosciences. (pp. 597–609). Cambridge, MA: MIT Press. https://psycnet.apa.org/record/1994-98810-038

Jueptner, M., & **Weiller, C.** (1995). does measurement of regional cerebral blood flow reflect synaptic activity? Implications for PET and fMRI. *Neuroimage, 2*(2), 148-156. http://doi.org/10.1006/nimg.1995.1017

Jung, S., Kim, J. H., Sung, G., Ko, Y. G., Bang, M., Park, C. I., & Lee, S. H. (2020). Uncinate fasciculus white matter connectivity related to impaired social perception and cross-sectional and longitudinal symptoms in patients with schizophrenia spectrum psychosis. *Neuroscience Letters, 737*, 135144. https://doi.org/10.1016/j.neulet.2020.135144

Kalidindi, H. T., Thuruthel, T. G., Laschi, C., & Falotico, E. (2019, April). Cerebellum-inspired approach for adaptive kinematic control of soft robots. In 2019 2nd IEEE International Conference on Soft Robotics (RoboSoft) (pp. 684-689). IEEE. http://doi.org/10.1109/ROBOSOFT.2019.8722735

Kaplan, J. T., Aziz-Zadeh, L., Uddin, L. Q., & Iacoboni, M. (2008). The self across the senses: an fMRI study of self-face and self-voice recognition. *Social Cognitive and Affective Neuroscience, 3*(3), 218-223. https://doi.org/10.1093/scan/nsn014

Kaplan, R., & **Friston, K. J.** (2018). Planning and navigation as active inference. *Biological cybernetics, 112*(4), 323-343. https://doi.org/10.1007/s00422-018-0753-2

Kapur, S. (2003). Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *American journal of Psychiatry, 160*(1), 13-23. https://doi-org.mu.idm.oclc.org/10.1176/appi.ajp.160.1.13

Kapur, S. (2004). How antipsychotics become anti-'psychotic'–from dopamine to salience to psychosis. *Trends in Pharmacological Sciences, 25*(8), 402-406. https://doi.org/10.1016/j.tips.2004.06.005

Kapur, S., Mizrahi, R., & Li, M. (2005). From dopamine to salience to psychosis—linking biology, pharmacology and phenomenology of psychosis. *Schizophrenia research, 79*(1), 59-68. https://doi.org/10.1016/j.schres.2005.01.003

Karbasforoushan, H., & **Woodward, N. D.** (2012). Resting-state networks in schizophrenia. *Current topics in medicinal chemistry, 12*(21), 2404-2414. https://doi.org/10.2174/156802612805289863

Karoutzou, G., Emrich, H. M., & Dietrich, D. E. (2008). The myelin-pathogenesis puzzle in schizophrenia: a literature review. *Molecular psychiatry, 13*(3), 245-260. https://doi.org/10.1038/sj.mp.4002096

Kawahara, H. (2003). Exemplar-based Voice Quality Analysis and Control using a High Quality Auditory Morphing Procedure based on STRAIGHT. *Parameters, 4*(5), 2. Retrieved from https://www.isca-speech.org/archive_open/archive_papers/voqual03/voq3_109.pdf

Kawahara, H. (2006). STRAIGHT, exploitation of the other aspect of VOCODER: Perceptually isomorphic decomposition of speech sounds. *Acoustical Science and Technology, 27*(6), 349-353. https://doi.org/10.1250/ast.27.349

Kawato, M., Furukawa, K., & Suzuki, R. (1987). A hierarchical neural-network model for control and learning of voluntary movement. *Biological Cybernetics, 57*(3), 169–185. https://doi.org/10.1007/BF00364149

Kawato, M., & **Gomi, H.** (1992). A computational model of four regions of the cerebellum based on feedback-error learning. *Biological Cybernetics, 68*(2), 95–103. https://doi.org/10.1007/BF00201431

Kawato, M. (1999). Internal models for motor control and trajectory planning. *Current opinion in neurobiology, 9*(6), 718-727. https://doi.org/10.1016/S0959-4388(99)00028-8

Kawato, M., Ohmae, S., Hoang, H., & Sanger, T. (2021). 50 years since the Marr, Ito, and Albus models of the cerebellum. *Neuroscience, 462*, 151-174. https://doi.org/10.1016/j.neuroscience.2020.06.019

Kaymaz, N., & **Van Os, J.** (2010). Extended psychosis phenotype–yes: single continuum–unlikely: a commentary on 'Why we need more debate on whether psychotic symptoms lie on a continuum with normality'by David (2010). *Psychological medicine, 40*(12), 1963-1966. https://doi.org/10.1017/S0033291710000358

Kearney, E., & **Guenther, F. H.** (2019). Articulating: the neural mechanisms of speech production. *Language, Cognition and Neuroscience, 34*(9), 1214-1229. https://doi.org/10.1080/23273798.2019.1589541

Kehrer, C., Maziashvili, N., Dugladze, T., & Gloveli, T. (2008). Altered excitatory-inhibitory balance in the NMDA-hypofunction model of schizophrenia. *Frontiers in molecular neuroscience, 1*, 6. https://doi.org/10.3389/neuro.02.006.2008

Kelleher, I., Jenner, J. A., & Cannon, M. (2010). Psychotic symptoms in the general population–an evolutionary perspective. *The British Journal of Psychiatry, 197*(3), 167-169. https://doi.org/10.1192/bjp.bp.109.076018

Kelleher, I., & **Cannon, M.** (2011). Psychotic-like experiences in the general population: characterizing a high-risk group for psychosis. *Psychological medicine, 41*(1), 1-6. https://doi.org/10.1017/S0033291710001005

Kelleher, I., Connor, D., Clarke, M. C., Devlin, N., Harley, M., & Cannon, M. (2012). Prevalence of psychotic symptoms in childhood and adolescence: a systematic review and meta-analysis of population-based studies. *Psychological medicine, 42*(9), 1857-1863. http://doi.org/10.1017/S0033291711002960

Kelly, R. M., & **Strick, P. L.** (2003). Cerebellar loops with motor cortex and prefrontal cortex of a nonhuman primate. *Journal of Neuroscience, 23* (23), 8432–8444. https://doi.org/10.1523/JNEUROSCI.23-23-08432.2003

Kendler, K. S., Gallagher, T. J., Abelson, J. M., & Kessler, R. C. (1996). Lifetime prevalence, demographic risk factors, and diagnostic validity of nonaffective psychosis as assessed in a US community sample: the National Comorbidity Survey. *Archives of general psychiatry, 53*(11), 1022-1031. http://doi.org/10.1001/archpsyc.1996.01830110060007

Kerr, G. K., Miall, R. C., & Stein, J. F. (1993). Visuo-motor adaptation during inactivation of the cerebellar nuclei: A preliminary report. *Human Movement Science, 12*(1-2), 71-83. https://doi.org/10.1016/0167-9457(93)90037-P

Kesby, J. P., Murray, G. K., & Knolle, F. (2021). Neural circuitry of salience and reward processing in psychosis. *Biological Psychiatry Global Open Science*. https://doi.org/10.1016/j.bpsgos.2021.12.003

Kilner, J. M., Friston, K. J., & Frith, C. D. (2007). Predictive coding: an account of the mirror neuron system. *Cognitive processing, 8*(3), 159-166. https://doi.org/10.1007/s10339-007-0170-2

Kim, S. G., & **Ogawa, S.** (2012). Biophysical and physiological origins of blood oxygenation level-dependent fMRI signals. *Journal of Cerebral Blood Flow & Metabolism, 32*(7), 1188-1206. https://doi.org/10.1038/jcbfm.2012.23

Kim, D. J., Kent, J. S., Bolbecker, A. R., Sporns, O., Cheng, H., Newman, S. D., ... & Hetrick, W. P. (2014). Disrupted modular architecture of cerebellum in schizophrenia: a graph theoretic analysis. *Schizophrenia bulletin, 40*(6), 1216-1226. https://doi.org/10.1093/schbul/sbu059

Kim, J., & **Augustine, G. J.** (2021). Molecular layer interneurons: key elements of cerebellar network computation and behavior. *Neuroscience, 462*, 22-35. https://doi.org/10.1016/j.neuroscience.2020.10.008

Kimura M., & **Yotsumoto Y.** (2018). Auditory traits of "own voice". *PLoS ONE 13*(6): e0199443. https://doi.org/10.1371/journal.pone.0199443

Kitis, O., Ozalay, O., Zengin, E. B., Haznedaroglu, D., Eker, M. C., Yalvac, D., ... & Gonul, A. S. (2012). Reduced left uncinate fasciculus fractional anisotropy in deficit schizophrenia but not in non-deficit schizophrenia. *Psychiatry and clinical neurosciences, 66*(1), 34-43. https://doi.org/10.1111/j.1440-1819.2011.02293.x

Kleber, B., Zeitouni, A. G., Friberg, A., & Zatorre, R. J. (2013). Experience dependent modulation of feedback integration during singing: Role of the right anterior insula. *Journal of Neuroscience, 33*(14), 6070–6080. https://doi.org/10.1523/JNEUROSCI.4418-12.2013

Knill, D. C., & **Pouget, A.** (2004). The Bayesian brain: the role of uncertainty in neural coding and computation. *TRENDS in Neurosciences, 27*(12), 712-719. https://doi.org/10.1016/j.tins.2004.10.007

Knöchel, C., O'Dwyer, L., Alves, G., Reinke, B., Magerkurth, J., Rotarska-Jagiela, A., ... & Oertel-Knöchel, V. (2012). Association between white matter fiber integrity and subclinical psychotic symptoms in schizophrenia patients and unaffected relatives. *Schizophrenia research, 140*(1-3), 129-135. https://doi.org/10.1016/j.schres.2012.06.001

Knolle, F., Schröger, E., Baess, P., & Kotz, S. A. (2012). The cerebellum generates motor-to-auditory predictions: ERP lesion evidence. *Journal of Cognitive Neuroscience, 24*(3), 698–706. https://doi.org/10.1162/jocn_a_00167

Knolle, F., Schröger, E., & Kotz, S. A. (2013). Prediction errors in self-and externally-generated deviants. *Biological Psychology, 92*(2), 410-416. https://doi.org/10.1016/j.biopsycho.2012.11.017

Knolle, F., Schwartze, M., Schröger, E., & Kotz, S. A. (2019). Auditory predictions and prediction errors in response to self-initiated vowels. *Frontiers in Neuroscience, 13*, 1146. https://doi.org/10.3389/fnins.2019.01146

Koenigsberger, L. (1906). Hermann von Helmholtz (F. A. Welby, Trans.). Oxford, England: Clarendon Press.

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

Kompus, K., Løberg, E. M., Posserud, M. B., & Lundervold, A. J. (2015). Prevalence of auditory hallucinations in Norwegian adolescents: Results from a population-based study. *Scandinavian journal of psychology, 56*(4), 391-396. https://doi.org/10.1111/sjop.12219

Kowalski, J., Aleksandrowicz, A., Dąbkowska, M., & Gawęda, Ł. (2021). Neural Correlates of Aberrant Salience and Source Monitoring in Schizophrenia and At-Risk Mental States—A Systematic Review of fMRI Studies. *Journal of clinical medicine, 10*(18), 4126. https://doi.org/10.3390/jcm10184126

Kotz, S. A., Meyer, M., Alter, K., Besson, M., von Cramon, D. Y., & Friederici, A. D. (2003). On the lateralization of emotional prosody: an event-related functional MR investigation. *Brain and Language, 86*(3), 366-376. https://doi.org/10.1016/S0093-934X(02)00532-1

Koziol, L. F., Budding, D., Andreasen, N., D'Arrigo, S., Bulgheroni, S., Imamizu, H., ... & Yamazaki, T. (2014). Consensus paper: the cerebellum's role in movement and cognition. *The Cerebellum, 13*(1), 151-177. http://doi.org/10.1007/s12311-013-0511-x

Krakauer, J. W., Ghilardi, M. F., Mentis, M., Barnes, A., Veytsman, M., Eidelberg, D., & Ghez, C. (2004). Differential cortical and subcortical activations in learning rotations and gains for reaching: A PET study. *Journal of Neurophysiology, 91*(2), 924–933. https://doi.org/10.1152/jn.00675.2003

Krakauer, J. W., Hadjiosif, A. M., Xu, J., Wong, A. L., & Haith, A. M. (2019). Motor learning. *Compr Physiol, 9*(2), 613-663. http://doi.org/10.1002/cphy.c170043

Kreiman, J., Gerratt, B. R., Precoda, K., & Berke, G. S. (1992). Individual differences in voice quality perception. *Journal of Speech, Language, and Hearing Research, 35*(3), 512-520. https://doi.org/10.1044/jshr.3503.512

Kreiman, J., & **Sidtis, D.** (2011). Foundations of voice studies: An interdisciplinary approach to voice production and perception. John Wiley & Sons. https://doi.org/10.1002/9781444395068

Kreitewolf, J., Gaudrain, E., & von Kriegstein, K. (2014). A neural mechanism for recognizing speech spoken by different speakers. *Neuroimage, 91*, 375-385. https://doi.org/10.1016/j.neuroimage.2014.01.005

Krienen, F. M., & **Buckner, R. L.** (2009). Segregated fronto-cerebellar circuits revealed by intrinsic functional connectivity. *Cerebral Cortex, 19*(10), 2485–2497. https://doi.org/10.1093/cercor/bhp135

Kroken, R. A., Løberg, E. M., Drønen, T., Gruner, R., Hugdahl, K., Kompus, K., ... & Johnsen, E. (2014). A critical review of pro-cognitive drug targets in psychosis: convergence on myelination and inflammation. *Frontiers in psychiatry, 5*, 11. https://doi.org/10.3389/fpsyt.2014.00011

Kubera, K. M., Rashidi, M., Schmitgen, M. M., Barth, A., Hirjak, D., Sambataro, F., ... & Wolf, R. C. (2019). Structure/function interrelationships in patients with schizophrenia who have persistent auditory verbal hallucinations: A multimodal MRI study using parallel ICA. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 93*, 114-121. https://doi.org/10.1016/j.pnpbp.2019.03.007

Kubicki, M., Westin, C. F., Maier, S. E., Mamata, H., Frumin, M., Ersner-Hershfield, H., ... & Shenton, M. E. (2002). Diffusion tensor imaging and its application to neuropsychiatric disorders. *Harvard review of psychiatry, 10*(6), 324-336. http://doi.org/10.1080/10673220216231

Kubicki, M., Park, H., Westin, C. F., Nestor, P. G., Mulkern, R. V., Maier, S. E., ... & Shenton, M. E. (2005). DTI and MTR abnormalities in schizophrenia: analysis of white matter integrity. *Neuroimage, 26*(4), 1109-1118. https://doi.org/10.1016/j.neuroimage.2005.03.026

Kühn, S., & **Gallinat, J.** (2012). Quantitative meta-analysis on state and trait aspects of auditory verbal hallucinations in schizophrenia. *Schizophrenia bulletin, 38*(4), 779-786. https://doi.org/10.1093/schbul/sbq152

Kumari, V., Fannon, D., Ffytche, D. H., Raveendran, V., Antonova, E., Premkumar, P., ... & Johns, L. C. (2010). Functional MRI of verbal self-monitoring in schizophrenia: performance and illness-specific effects. *Schizophrenia bulletin, 36*(4), 740-755. https://doi.org/10.1093/schbul/sbn148

Kurata, K., Tsuji, T., Naraki, S., Seino, M., & Abe, Y. (2000). Activation of the dorsal premotor cortex and pre-supplementary motor area of humans during an auditory conditional motor task. *Journal of Neurophysiology, 84*(3), 1667–1672. https://doi.org/10.1152/jn.2000.84.3.1667

Laird, A. R., Fox, P. M., Price, C. J., Glahn, D. C., Uecker, A. M., Lancaster, J. L., … Fox, P. T. (2005). ALE meta-analysis: Controlling the false discovery rate and performing statistical contrasts. *Human Brain Mapping, 25*(1), 155–164. https://doi.org/10.1002/hbm.20136

Lancaster, J. L., Tordesillas-Gutiérrez, D., Martinez, M., Salinas, F., Evans, A., Zilles, K., … Fox, P. T. (2007). Bias between MNI and Talairach coordinates analyzed using the ICBM-152 brain template. *Human Brain Mapping, 28*(11), 1194–1205. https://doi.org/10.1002/hbm.20345

Lane, H., & **Tranel, B.** (1971). The Lombard sign and the role of hearing in speech. *Journal of Speech, Language, and Hearing Research, 14*(4), 677–709. https://doi.org/10.1044/jshr.1404.677

Larøi, F., Marczewski, P., & Van der Linden, M. (2004). Further evidence of the multi-dimensionality of hallucinatory predisposition: factor structure of a modified version of the Launay-Slade Hallucinations Scale in a normal sample. *European Psychiatry, 19*(1), 15-20 https://doi.org/10.1016/S0924-9338(03)00028-2

Larøi, F., & **Van Der Linden, M.** (2005a). Nonclinical Participants' Reports of Hallucinatory Experiences. *Canadian Journal of Behavioural Science/Revue canadienne des sciences du comportement, 37*(1), 33. https://doi.org/10.1037/h0087243

Larøi, F., & **Van der Linden, M.** (2005b). Metacognitions in proneness towards hallucinations and delusions. *Behaviour research and Therapy, 43*(11), 1425-1441. https://doi.org/10.1016/j.brat.2004.10.008

Larøi, F., & **Woodward, T. S.** (2007). Hallucinations from a cognitive perspective. *Harvard Review of Psychiatry, 15*(3), 109-117. https://doi.org/10.1080/10673220701401993

Larøi, F., Sommer, I. E., Blom, J. D., Fernyhough, C., Ffytche, D. H., Hugdahl, K., ... & Waters, F. (2012). The characteristic features of auditory verbal hallucinations in clinical and nonclinical groups: state-of-the-art overview and future directions. *Schizophrenia bulletin, 38*(4), 724-733. https://doi.org/10.1093/schbul/sbs061

Larøi, F., Luhrmann, T. M., Bell, V., Christian Jr, W. A., Deshpande, S., Fernyhough, C., ... & Woods, A. (2014). Culture and hallucinations: overview and future directions. *Schizophrenia bulletin, 40*(Suppl_4), S213-S220. https://doi.org/10.1093/schbul/sbu012

Larson, C. R., Burnett, T. A., Kiran, S., & Hain, T. C. (2000). Effects of pitch-shift velocity on voice F0 responses. *The Journal of the Acoustical Society of America, 107*(1), 559–564. https://doi.org/10.1121/1.428323

Larsson, J., Solomon, S. G., & Kohn, A. (2016). fMRI adaptation revisited. *Cortex, 80*, 154-160. https://doi.org/10.1016/j.cortex.2015.10.026

Latinus, M., & **Belin, P.** (2011). Human voice perception. *Current Biology, 21*(4), R143-R145. https://doi.org/10.1016/j.cub.2010.12.033

Latinus, M., & **Belin, P.** (2012). Perceptual auditory aftereffects on voice identity using brief vowel stimuli. *PLoS One, 7*(7). https://doi.org/10.1371/journal.pone.0041384

Latinus, M., McAleer, P., Bestelmeyer, P. E., & Belin, P. (2013). Norm-based coding of voice identity in human auditory cortex. *Current Biology, 23*(12), 1075-1080. https://doi.org/10.1016/j.cub.2013.04.055

Lau, A., & **Tymianski, M.** (2010). Glutamate receptors, neurotoxicity and neurodegeneration. *Pflügers Archiv-European Journal of Physiology, 460*(2), 525-542. https://doi.org/10.1007/s00424-010-0809-1

Launay, G., & **Slade, P.** (1981). The measurement of hallucinatory predisposition in male and female prisoners. *Personality and Individual Differences, 2*(3), 221-234. https://doi.org/10.1016/0191-8869(81)90027-1

Lavan, N., Burton, A. M., Scott, S. K., & McGettigan, C. (2019). Flexible voices: Identity perception from variable vocal signals. *Psychonomic bulletin & review, 26*(1), 90-102. https://doi.org/10.3758/s13423-018-1497-7

Lavner, Y., Gath, I., & Rosenhouse, J. (2000). The effects of acoustic modifications on the identification of familiar voices speaking isolated vowels. *Speech Communication, 30*(1), 9-26. https://doi.org/10.1016/S0167-6393(99)00028-X

Lavner, Y., Rosenhouse, J., & Gath, I. (2001). The prototype model in speaker identification by human listeners. *International Journal of Speech Technology, 4(*1), 63-74. https://doi.org/10.1023/A:1009656816383

Lee, B. S. (1950). Effects of delayed speech feedback. *The Journal of the Acoustical Society of America, 22*(6), 824–826. https://doi.org/10.1121/1.1906696

Lehoux, C., Gobeil, M. H., Lefèbvre, A. A., Maziade, M., & Roy, M. A. (2009). The five-factor structure of the PANSS: a critical review of its consistency across studies. *Clinical schizophrenia & related psychoses, 3*(2), 103-110. https://www.clinicalschizophrenia.net/articles/the-fivefactor-structure-of-the-panss-a-critical-review-of-its-consistency-across-studies.pdf

Lei, W., Li, N., Deng, W., Li, M., Huang, C., Ma, X., ... & Li, T. (2015). White matter alterations in first episode treatment-naive patients with deficit schizophrenia: a combined VBM and DTI study. *Scientific reports, 5*(1), 1-11. https://doi.org/10.1038/srep12994

Le Maléfan, P., & **Sommer, A.** (2015). Léon Marillier and the veridical hallucination in late-nineteenth-and early-twentieth-century French psychology and psychopathology. *History of Psychiatry, 26*(4), 418-432. https://doi.org/10.1177/0957154X14562756

Leng, B., Han, S., Bao, Y., Zhang, H., Wang, Y., Wu, Y., & Wang, Y. (2016). The uncinate fasciculus as observed using diffusion spectrum imaging in the human brain. *Neuroradiology, 58*(6), 595-606. https://doi.org/10.1007/s00234-016-1650-9

Lenth, R., Singmann, H., Love, J., Buerkner, P., & Herve, M. (2020). emmeans: estimated marginal means. R package version 1.4. 4. Retrieved from https://CRAN.R-project.org/package=emmeans

Leptourgos, P., Denève, S., & Jardri, R. (2017). Can circular inference relate the neuropathological and behavioral aspects of schizophrenia?. *Current opinion in neurobiology, 46*, 154-161. https://doi.org/10.1016/j.conb.2017.08.012

Leptourgos, P., & **Corlett, P. R.** (2020). Embodied predictions, agency, and psychosis. *Frontiers in big Data, 3*, 27. https://doi.org/10.3389/fdata.2020.00027

Leroux, E., Delcroix, N., & Dollfus, S. (2015). Left-hemisphere lateralization for language and interhemispheric fiber tracking in patients with schizophrenia. *Schizophrenia research, 165*(1), 30-37. https://doi.org/10.1016/j.schres.2015.03.028

Leroux, E., Delcroix, N., & Dollfus, S. (2017). Abnormalities of language pathways in schizophrenia patients with and without a lifetime history of auditory verbal hallucinations: A DTI-based tractography study. *The World Journal of Biological Psychiatry, 18*(7), 528-538. https://doi.org/10.1080/15622975.2016.1274053

Leube, D. T., Knoblich, G., Erb, M., Grodd, W., Bartels, M., & Kircher, T. T. (2003). The neural correlates of perceiving one's own movements. *NeuroImage, 20*(4), 2084–2090. https://doi.org/10.1016/j.neuroimage.2003.07.033

Levitan, C., Ward, P. B., Catts, S. V., & Hemsley, D. R. (1996). Predisposition toward auditory hallucinations: The utility of the Launay-Slade Hallucination Scale in psychiatric patients. *Personality and Individual Differences, 21*(2), 287-289. https://doi.org/10.1016/0191-8869(96)00052-9

Levy, B. J., & **Wagner, A. D.** (2011). Cognitive control and right ventrolateral prefrontal cortex: Reflexive reorienting, motor inhibition, and action updating. *Annals of the New York Academy of Sciences, 1224*(1), 40–62. https://doi.org/10.1111/j.1749-6632.2011.05958.x

Lewis, D. A., & **Sweet, R. A.** (2009). Schizophrenia from a neural circuitry perspective: advancing toward rational pharmacological therapies. *The Journal of clinical investigation, 119*(4), 706-716. http://doi.org/10.1172/JCI37335

Liberman, A. M. (1957). Some results of research on speech perception. *The Journal of the Acoustical Society of America, 29*(1), 117-123. https://doi.org/10.1121/1.1908635

Liberman, A. M., Cooper, F. S., Shankweiler, D. P., & Studdert-Kennedy, M. (1967). Perception of the speech code. *Psychological review, 74*(6), 431. https://doi.org/10.1037/h0020279

Liberman, A. M., & **Whalen, D. H.** (2000). On the relation of speech to language. *Trends in cognitive sciences, 4*(5), 187-196. https://doi.org/10.1016/S1364-6613(00)01471-6

Liebenthal, E., & **Möttönen, R.** (2018). An interactive model of auditory-motor speech perception. *Brain and language, 187*, 33-40. https://doi.org/10.1016/j.bandl.2017.12.004

Lien, Y. J., Kao, Y. C., Liu, Y. P., Chang, H. A., Tzeng, N. S., Lu, C. W., ... & Loh, C. H. (2015). Relationships of perceived public stigma of mental illness and psychosis-like experiences in a non-clinical population sample. *Social psychiatry and psychiatric epidemiology, 50*(2), 289-298. https://doi.org/10.1007/s00127-014-0929-6

Lillicrap, T. P., Santoro, A., Marris, L., Akerman, C. J., & Hinton, G. (2020). Backpropagation and the brain. *Nature Reviews Neuroscience, 21*(6), 335-346. https://doi.org/10.1038/s41583-020-0277-3

Linscott, R. J., & **van Os, J.** (2013). An updated and conservative systematic review and meta-analysis of epidemiological evidence on psychotic experiences in children and adults: on the pathway from proneness to persistence to dimensional expression across mental disorders. *Psychological medicine, 43*(6), 1133. https://doi.org/10.1017/S0033291712001626

Lim, A., Hoek, H. W., Deen, M. L., Blom, J. D., Bruggeman, R., Cahn, W., ... & Wiersma, D. (2016). Prevalence and classification of hallucinations in multiple sensory modalities in schizophrenia spectrum disorders. *Schizophrenia research, 176*(2-3), 493-499. https://doi.org/10.1016/j.schres.2016.06.010

Lima, C. F., Krishnan, S., & Scott, S. K. (2016). Roles of supplementary motor areas in auditory processing and auditory imagery. *Trends in neurosciences, 39*(8), 527-542. https://doi.org/10.1016/j.tins.2016.06.003

Limanowski, J., Kirilina, E., & Blankenburg, F. (2017). Neuronal correlates of continuous manual tracking under varying visual movement feedback in a virtual reality environment. *NeuroImage, 146*, 81–89. https://doi.org/10.1016/j.neuroimage.2016.11.009

Loehr, J. D. (2022). The sense of agency in joint action: An integrative review. *Psychonomic Bulletin & Review*, 1-29. https://doi.org/10.3758/s13423-021-02051-3

Logothetis, N. K., & **Wandell, B. A.** (2004). Interpreting the BOLD signal. *Annu. Rev. Physiol., 66,* 735-769. https://doi.org/10.1146/annurev.physiol.66.082602.092845

Logothetis, N. K. (2008). What we can do and what we cannot do with fMRI. *Nature, 453*(7197), 869-878. https://doi.org/10.1038/nature06976

Lombard, E. (1911). Le signe de l'elevation de la voix. *Ann Mal de L'Oreille et du Larynx, 37*, 101–119.

Lotto, A. J., Hickok, G. S., & Holt, L. L. (2009). Reflections on mirror neurons and speech perception. *Trends in cognitive sciences, 13*(3), 110-114. https://doi.org/10.1016/j.tics.2008.11.008

Lu, X., Miyachi, S., & Takada, M. (2012). Anatomical evidence for the involvement of medial cerebellar output from the interpositus nuclei in cognitive functions. *Proceedings of the National Academy of Sciences of the United States of America, 109*(46), 18980–18984. https://doi.org/10.1073/pnas.1211168109

Luck, D., Buchy, L., Czechowska, Y., Bodnar, M., Pike, G. B., Campbell, J. S., ... & Lepage, M. (2011). Fronto-temporal disconnectivity and clinical short-term outcome in first episode psychosis: a DTI-tractography study. *Journal of psychiatric research, 45*(3), 369-377. https://doi.org/10.1016/j.jpsychires.2010.07.007

Luttenbacher, I., Philips, A., Kazemi, R., Hadipour, A. L., Sanghvi, I., Martinez, J., & Adamson, M. M. (2021). Transdiagnostic role of glutamate and white matter damage in neuropsychiatric disorders: A Systematic Review. Journal of Psychiatric Research. https://doi.org/10.1016/j.jpsychires.2021.12.042

MacKay, D. G. (1968). Metamorphosis of a critical interval: Age-linked changes in the delay in auditory feedback that produces maximal disruption of speech. *The Journal of the Acoustical Society of America, 43*(4), 811–821. https://doi.org/10.1121/1.1910900

Maguinness, C., Roswandowitz, C., & von Kriegstein, K. (2018). Understanding the mechanisms of familiar voice-identity recognition in the human brain. *Neuropsychologia, 116*, 179-193. https://doi.org/10.1016/j.neuropsychologia.2018.03.039

Maijer, K., Begemann, M. J., Palmen, S. J., Leucht, S., & Sommer, I. E. (2018). Auditory hallucinations across the lifespan: a systematic review and meta-analysis. *Psychological medicine, 48*(6), 879-888. http://doi.org/10.1017/S0033291717002367

Makowski, C., Lepage, M., & Evans, A. C. (2019). Head motion: the dirty little secret of neuroimaging in psychiatry. *Journal of Psychiatry and Neuroscience, 44*(1), 62-68. http://doi.org/10.1503/jpn.180022

Mandl, R. C., Schnack, H. G., Luigjes, J., Van Den Heuvel, M. P., Cahn, W., Kahn, R. S., & Hulshoff Pol, H. E. (2010). Tract-based analysis of magnetization transfer ratio and diffusion tensor imaging of the frontal and frontotemporal connections in schizophrenia. *Schizophrenia bulletin, 36*(4), 778-787. https://doi.org/10.1093/schbul/sbn161

Manni, E., & **Petrosini, L.** (2004). A century of cerebellar somatotopy: a debated representation. *Nature Reviews Neuroscience, 5*(3), 241-249. https://doi.org/10.1038/nrn1347

Manoliu, A., Riedl, V., Zherdin, A., Mühlau, M., Schwerthöffer, D., Scherr, M., ... & Sorg, C. (2014). Aberrant dependence of default mode/central executive network interactions on anterior insular salience network activity in schizophrenia. *Schizophrenia bulletin, 40*(2), 428-437. https://doi.org/10.1093/schbul/sbt037

Manto, M., Bower, J. M., Conforto, A. B., Delgado-García, J. M., Da Guarda, S. N. F., Gerwig, M., ... & Timmann, D. (2012). Consensus paper: roles of the cerebellum in motor control—the diversity of ideas on cerebellar involvement in movement. *The Cerebellum, 11*(2), 457-487. https://doi.org/10.1007/s12311-011-0331-9

Martí-Bonmatí, L., Lull, J. J., García-Martí, G., Aguilar, E. J., Moratal-Pérez, D., Poyatos, C., ... & Sanjuán, J. (2007). Chronic auditory hallucinations in schizophrenic patients: MR analysis of the coincidence between functional and morphologic abnormalities. *Radiology, 244*(2), 549-556. https://doi.org/10.1148/radiol.2442060727

Martin, P., & **Albers, M.** (1995). Cerebellum and schizophrenia: a selective review. *Schizophrenia Bulletin, 21*(2), 241-250. https://doi.org/10.1093/schbul/21.2.241

Maschke, M., Gomez, C. M., Ebner, T. J., & Konczak, J. (2004). Hereditary cerebellar ataxia progressively impairs force adaptation during goal-directed arm movements. *Journal of Neurophysiology, 91*(1), 230–238. https://doi.org/10.1152/jn.00557.2003

Mathalon, D. H., & **Ford, J. M.** (2008). Corollary discharge dysfunction in schizophrenia: evidence for an elemental deficit. *Clinical EEG and neuroscience, 39*(2), 82-86. https://doi.org/10.1177/155005940803900212

Matute, C., Alberdi, E., Domercq, M., Sánchez-Gómez, M. V., Pérez-Samartín, A., Rodríguez-Antigüedad, A., & Pérez-Cerdá, F. (2007). Excitotoxic damage to white matter. *Journal of anatomy, 210*(6), 693-702. http://doi.org/10.1111/j.1469-7580.2007.00733.x

McCarthy-Jones, S., & **Fernyhough, C.** (2011). The varieties of inner speech: Links between quality of inner speech and psychopathological variables in a sample of young adults. *Consciousness and cognition, 20(*4), 1586-1593. https://doi.org/10.1016/j.concog.2011.08.005

McCarthy-Jones, S., Trauer, T., Mackinnon, A., Sims, E., Thomas, N., & Copolov, D. L. (2014). A new phenomenological survey of auditory hallucinations: evidence for subtypes and implications for theory and practice. *Schizophrenia bulletin, 40*(1), 231-235. https://doi.org/10.1093/schbul/sbs156

McCarthy-Jones, S., Oestreich, L. K., Bank, A. S. R., & Whitford, T. J. (2015). Reduced integrity of the left arcuate fasciculus is specifically associated with auditory verbal hallucinations in schizophrenia. *Schizophrenia research, 162*(1-3), 1-6. https://doi.org/10.1016/j.schres.2014.12.041

McCarthy-Jones, S., Smailes, D., Corvin, A., Gill, M., Morris, D. W., Dinan, T. G., ... & Dudley, R. (2017). Occurrence and co-occurrence of hallucinations by modality in schizophrenia-spectrum disorders. *Psychiatry research, 252*, 154-160. https://doi.org/10.1016/j.psychres.2017.01.102

McCutcheon, R. A., Krystal, J. H., & Howes, O. D. (2020). Dopamine and glutamate in schizophrenia: biology, symptoms and treatment. *World Psychiatry, 19*(1), 15-33. https://doi.org/10.1002/wps.20693

McGettigan, C., & **Scott, S. K.** (2012). Cortical asymmetries in speech perception: what's wrong, what's right and what's left?. *Trends in cognitive sciences, 16*(5), 269-276. https://doi.org/10.1016/j.tics.2012.04.006

McGrath, J. J., Saha, S., Al-Hamzawi, A., Alonso, J., Bromet, E. J., Bruffaerts, R., ... & Kessler, R. C. (2015). Psychotic experiences in the general population: a cross-national analysis based on 31 261 respondents from 18 countries. *JAMA psychiatry, 72*(7), 697-705. https://doi.org/10.1001/jamapsychiatry.2015.0575

McGuire, P. K., Silbersweig, D. A., & Frith, C. D. (1996). Functional neuroanatomy of verbal self-monitoring. *Brain, 119*(3), 907–917. https://doi.org/10.1093/brain/119.3.907

McNamee, D., & **Wolpert, D. M.** (2019). Internal models in biological control. *Annual review of control, robotics, and autonomous systems, 2*, 339-364. http://doi.org/10.1146/annurev-control-060117-105206

Mechelli, A., Allen, P., Amaro Jr., E., Fu, C. H., Williams, S. C., Brammer, M. J., ... & McGuire, P. K. (2007). Misattribution of speech and impaired connectivity in patients with auditory verbal hallucinations. *Human brain mapping, 28*(11), 1213-1222. https://doi.org/10.1002/hbm.20341

Meltzer, H. Y., & **Stahl, S. M.** (1976). The dopamine hypothesis of schizophrenia: a review. *Schizophrenia bulletin, 2*(1), 19. https://doi.org/10.1093/schbul/2.1.19

Menon, V., & **Uddin, L. Q.** (2010). Saliency, switching, attention and control: a network model of insula function. *Brain structure and function, 214*(5-6), 655-667. https://doi.org/10.1007/s00429-010-0262-0

Menon, V. (2011). Large-scale brain networks and psychopathology: a unifying triple network model. *Trends in cognitive sciences, 15*(10), 483-506. https://doi.org/10.1016/j.tics.2011.08.003

Miall, R. C., Weir, D. J., Wolpert, D. M., & Stein, J. F. (1993). Is the cerebellum a smith predictor? *Journal of Motor Behavior, 25*(3), 203–216. https://doi.org/10.1080/00222895.1993.9942050

Miall, R. C., & **Wolpert, D. M.** (1996). Forward models for physiological motor control. *Neural Networks, 9*(8), 1265–1279. https://doi.org/10.1016/S0893-6080(96)00035-4

Middleton, F. A., & **Strick, P. L.** (1997). Cerebellar output channels. *International review of neurobiology, 41*, 61-82. https://doi.org/10.1016/S0074-7742(08)60347-5

Middleton, F. A., & **Strick, P. L.** (2001). Cerebellar projections to the prefrontal cortex of the primate. *Journal of Neuroscience, 21*(2), 700–712. https://doi.org/10.1523/JNEUROSCI.21-02-00700.2001

Mighdoll, M. I., Tao, R., Kleinman, J. E., & Hyde, T. M. (2015). Myelin, myelin-related disorders, and psychosis. *Schizophrenia research, 161*(1), 85-93. https://doi.org/10.1016/j.schres.2014.09.040

Miyata, J. (2019). Toward integrated understanding of salience in psychosis. *Neurobiology of disease, 131*, 104414. https://doi.org/10.1016/j.nbd.2019.03.002

Moberget, T., Gullesen, E. H., Andersson, S., Ivry, R. B., & Endestad, T. (2014). Generalized role for the cerebellum in encoding internal models: Evidence from semantic processing. *Journal of Neuroscience, 34*(8), 2871–2878. https://doi.org/10.1523/JNEUROSCI.2264-13.2014

Moberget, T., Doan, N. T., Alnæs, D., Kaufmann, T., Córdova-Palomera, A., Lagerberg, T. V., ... & Westlye, L. T. (2018). Cerebellar volume and cerebellocerebral structural covariance in schizophrenia: a multisite mega-analysis of 983 patients and 1349 healthy controls. *Molecular psychiatry, 23*(6), 1512-1520. https://doi.org/10.1038/mp.2017.106

Moberget, T., & **Ivry, R. B.** (2019). Prediction, psychosis, and the cerebellum. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 4*(9), 820-831. https://doi.org/10.1016/j.bpsc.2019.06.001

Moerel, M., De Martino, F., & Formisano, E. (2012). Processing of natural sounds in human auditory cortex: tonotopy, spectral tuning, and relation to voice sensitivity. *Journal of Neuroscience, 32*(41), 14205-14216. https://doi.org/10.1523/JNEUROSCI.1388-12.2012

Moghaddam, B., & **Javitt, D.** (2012). From revolution to evolution: the glutamate hypothesis of schizophrenia and its implication for treatment. *Neuropsychopharmacology, 37*(1), 4-15. https://doi.org/10.1038/npp.2011.181

Moore, J. W. (2016). What is the sense of agency and why does it matter?. *Frontiers in psychology, 7*, 1272. https://doi.org/10.3389/fpsyg.2016.01272

Morrison, A. P., Wells, A., & Nothard, S. (2000). Cognitive factors in predisposition to auditory and visual hallucinations. *British Journal of Clinical Psychology, 39*(1), 67-78. https://doi.org/10.1348/014466500163112

Morton, S. M., & **Bastian, A. J.** (2006). Cerebellar contributions to locomotor adaptations during splitbelt treadmill walking. *Journal of Neuroscience, 26*(36), 9107–9116. https://doi.org/10.1523/JNEUROSCI.2622-06.2006

Moseley, P., Aleman, A., Allen, P., Bell, V., Bless, J., Bortolon, C., ... & Fernyhough, C. (2021). Correlates of hallucinatory experiences in the general population: An international multisite replication study. *Psychological Science, 32*(7), 1024-1037. https://doi.org/10.1177/0956797620985832

Mottolese, C., Richard, N., Harquel, S., Szathmari, A., Sirigu, A., &Desmurget, M. (2013). Mapping motor representations in the human cerebellum. *Brain, 136*(1), 330–342. https://doi.org/10.1093/brain/aws186

Mulert, C., Kirsch, V., Whitford, T. J., Alvarado, J., Pelavin, P., McCarley, R. W., ... & Shenton, M. E. (2012). Hearing voices: a role of interhemispheric auditory connectivity? *The World Journal of Biological Psychiatry, 13*(2), 153-158. https://doi.org/10.3109/15622975.2011.570789

Mullennix, J. W., Ross, A., Smith, C., Kuykendall, K., Conard, J., & Barb, S. (2011). Typicality effects on memory for voice: Implications for earwitness testimony. *Applied Cognitive Psychology, 25*(1), 29-34. https://doi.org/10.1002/acp.1635

Müller, V. I., Cieslik, E. C., Laird, A. R., Fox, P. T., Radua, J., Mataix-Cols, D., … Wager, T. D. (2017). Ten simple rules for neuroimaging meta-analysis. *Neuroscience and Biobehavioral Reviews, 84*, 151–161. https://doi.org/10.1016/j.neubiorev.2017.11.012

Müller-Preuss, P., & **Ploog, D.** (1981). Inhibition of auditory cortical neurons during phonation. *Brain research, 215*(1-2), 61-76. https://doi.org/10.1016/0006-8993(81)90491-1

Myin-Germeys, I., Krabbendam, L., & van Os, J. (2003). Continuity of psychotic symptoms in the community. *Current Opinion in Psychiatry, 16*(4), 443-449. http://doi.org/10.1097/01.yco.0000079208.36371.cd

Mumford, D. (1992). On the computational architecture of the neocortex. *Biological cybernetics, 66*(3), 241-251. http://doi.org/10.1007/BF00198477

Nahab, F. B., Kundu, P., Gallea, C., Kakareka, J., Pursley, R., Pohida, T., … Hallett, M. (2010). The neural processes underlying self-agency. *Cerebral Cortex, 21*(1), 48–55. https://doi.org/10.1093/cercor/bhq059

Nakamura, K., Kawashima, R., Sugiura, M., Kato, T., Nakamura, A., Hatano, K., ... & Kojima, S. (2001). Neural substrates for recognition of familiar voices: a PET study. *Neuropsychologia, 39*(10), 1047-1054. https://doi.org/10.1016/S0028-3932(01)00037-9

Nath, M., Wong, T. P., & Srivastava, L. K. (2021). Neurodevelopmental insights into circuit dysconnectivity in schizophrenia. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 104*, 110047. https://doi.org/10.1016/j.pnpbp.2020.110047

Natke, U., Donath, T. M., & Kalveram, K. T. (2003). Control of voice fundamental frequency in speaking versus singing. *The Journal of the Acoustical Society of America, 113*(3), 1587–1593. https://doi.org/10.1121/1.1543928

Nazimek, J. M., Hunter, M. D., & Woodruff, P. W. R. (2012). Auditory hallucinations: Expectation–perception model. *Medical hypotheses, 78*(6), 802-810. https://doi.org/10.1016/j.mehy.2012.03.014

Nezafat, R., Shadmehr, R., & Holcomb, H. H. (2001). Long-term adaptation to dynamics of reaching movements: A PET study. *Experimental Brain Research, 140*(1), 66–76. https://doi.org/10.1007/s002210100787

Nitschke, M. F., Kleinschmidt, A., Wessel, K., & Frahm, J. (1996). Somatotopic motor representation in the human anterior cerebellum. *Brain, 119*, 1023-1029. http://doi.org/10.1093/brain/119.3.1023

Nolan, F., McDougall, K., & Hudson, T. (2011). Some acoustic correlates of perceived (dis)similarity between same-accent voices. *ICPhS* (17), 1506-1509. http://icphs2011.hk.lt.cityu.edu.hk/resources/OnlineProceedings/RegularSession/Nolan/Nolan.pdf

Northoff, G., & **Qin, P.** (2011). How can the brain's resting state activity generate hallucinations? A 'resting state hypothesis' of auditory verbal hallucinations. *Schizophrenia research, 127*(1-3), 202-214. https://doi.org/10.1016/j.schres.2010.11.009

Northoff, G. (2014). Are auditory hallucinations related to the brain's resting state activity? A 'neurophenomenal resting state hypothesis'. *Clinical Psychopharmacology and Neuroscience, 12*(3), 189. https://doi.org/10.9758/cpn.2014.12.3.189

Numminen, J., & **Curio, G.** (1999). Differential effects of overt, covert and replayed speech on vowel-evoked responses of the human auditory cortex. *Neuroscience Letters, 272*(1), 29–32. https://doi.org/10.1016/S0304-3940(99)00573-X

Numminen, J., Salmelin, R., & Hari, R. (1999). Subject's own speech reduces reactivity of the human auditory cortex. *Neuroscience Letters, 265*(2), 119-122. https://doi.org/10.1016/S0304-3940(99)00218-9

O'Connor, S. (2020). The natural selection of private and inner speech. *Frontiers in Psychology, 11*, 163. https://doi.org/10.3389/fpsyg.2020.00163

Odeh, F., Ackerley, R., Bjaalie, J. G., & Apps, R. (2005). Pontine maps linking somatosensory and cerebellar cortices are in register with climbing fiber somatotopy. *Journal of Neuroscience, 25*(24), 5680–5690. https://doi.org/10.1523/JNEUROSCI.0558-05.2005

O'Donnell, L. J., Rigolo, L., Norton, I., Wells III, W. M., Westin, C. F., & Golby, A. J. (2012). fMRI-DTI modeling via landmark distance atlases for prediction and detection of fiber tracts. *NeuroImage, 60*(1), 456-470. https://doi.org/10.1016/j.neuroimage.2011.11.014

Oestreich, L. K., Mifsud, N. G., Ford, J. M., Roach, B. J., Mathalon, D. H., & Whitford, T. J. (2016). Cortical suppression to delayed self-initiated auditory stimuli in schizotypy: neurophysiological evidence for a continuum of psychosis. *Clinical EEG and Neuroscience, 47*(1), 3-10. https://doi.org/10.1177/1550059415581708

Ogawa, S., Menon, R. S., Kim, S. G., & Ugurbil, K. (1998). On the characteristics of functional magnetic resonance imaging of the brain. *Annual review of biophysics and biomolecular structure, 27*(1), 447-474. https://doi.org/10.1146/annurev.biophys.27.1.447

Ogawa, K., Inui, T., & Sugio, T. (2007). Neural correlates of state estimation in visually guided movements: An event-related fMRI study. *Cortex, 43*(3), 289–300. https://doi.org/10.1016/S0010-9452(08)70455-6

O'Hanlon, E., Leemans, A., Kelleher, I., Clarke, M. C., Roddy, S., Coughlan, H., ... & Cannon, M. (2015). White matter differences among adolescents reporting psychotic experiences: a population-based diffusion magnetic resonance imaging study. *JAMA psychiatry, 72*(7), 668-677. http://doi.org/10.1001/jamapsychiatry.2015.0137

Ohayon, M. M. (2000). Prevalence of hallucinations and their pathological associations in the general population. *Psychiatry research, 97*(2-3), 153-164. https://doi.org/10.1016/S0165-1781(00)00227-4

Olney, J. W., & **Farber, N. B.** (1995). Glutamate receptor dysfunction and schizophrenia. *Archives of general psychiatry, 52*(12), 998-1007. http://doi.org/10.1001/archpsyc.1995.03950240016004

Olney, J. W., Newcomer, J. W., & Farber, N. B. (1999). NMDA receptor hypofunction model of schizophrenia. *Journal of psychiatric research, 33*(6), 523-533. https://doi.org/10.1016/S0022-3956(99)00029-1

Olson, I. R., Von Der Heide, R. J., Alm, K. H., & Vyas, G. (2015). Development of the uncinate fasciculus: Implications for theory and developmental disorders. *Developmental cognitive neuroscience, 14*, 50-61. https://doi.org/10.1016/j.dcn.2015.06.003

O'Reilly, J. X., Beckmann, C. F., Tomassini, V., Ramnani, N., & Johansen- Berg, H. (2009). Distinct and overlapping functional zones in the cerebellum defined by resting state functional connectivity. *Cerebral Cortex, 20*(4), 953–965. https://doi.org/10.1093/cercor/bhp157

Palacios, E. R., Houghton, C., & Chadderton, P. (2021). Accounting for uncertainty: inhibition for neural inference in the cerebellum. *Proceedings of the Royal Society B, 288*(1947), 20210276. https://doi.org/10.1098/rspb.2021.0276

Palaniyappan, L., & **Liddle, P. F.** (2012). Does the salience network play a cardinal role in psychosis? An emerging hypothesis of insular dysfunction. *Journal of Psychiatry & Neuroscience, 37*(1), 17–27. https://doi.org/10.1503/jpn.100176

Palesi, F., Rinaldis, A., Castellazzi, G., Calamante, F., Muhlert, N., Chard, D., … Wheeler-Kingshott, C. A. G. (2017). Contralateral cortico-pontocerebellar pathways reconstruction in humans in vivo: Implications for reciprocal cerebro-cerebellar structural connectivity in motor and nonmotor areas. *Scientific Reports, 7*(1), 12841. https://doi.org/10.1038/s41598-017-13079-8

Parellada, E., Lomena, F., Font, M., Pareto, D., Gutierrez, F., Simo, M., ... & Bernardo, M. (2008). Fluordeoxyglucose-PET study in first-episode schizophrenic patients during the hallucinatory state, after remission and during linguistic–auditory activation. *Nuclear medicine communications, 29*(10), 894-900. https://10.1097/MNM.0b013e328302cd10

Parkinson, A. L., Flagmeier, S. G., Manes, J. L., Larson, C. R., Rogers, B., & Robin, D. A. (2012). Understanding the neural mechanisms involved in sensory control of voice production. *NeuroImage, 61*(1), 314–322. https://doi.org/10.1016/j.neuroimage.2012.02.068

Parr, T., & **Friston, K. J.** (2017). Working memory, attention, and salience in active inference. *Scientific reports, 7*(1), 1-21. https://doi.org/10.1038/s41598-017-15249-0

Parr, T., & **Friston, K. J.** (2018). The anatomy of inference: generative models and brain structure. *Frontiers in computational neuroscience, 12*, 90. https://doi.org/10.3389/fncom.2018.00090

Parr, T., & **Friston, K. J.** (2019). Attention or salience?. *Current opinion in psychology, 29*, 1-5. https://doi.org/10.1016/j.copsyc.2018.10.006

Pauling, L., & **Coryell, C. D.** (1936). The magnetic properties and structure of hemoglobin, oxyhemoglobin and carbonmonoxyhemoglobin. *Proceedings of the National Academy of Sciences, 22*(4), 210-216. https://doi.org/10.1073/pnas.22.4.210

Paulin, M. G. (2005). Evolution of the cerebellum as a neuronal machine for Bayesian state estimation. *Journal of neural engineering, 2*(3), S219. https://doi.org/10.1088/1741-2560/2/3/S06

Peelen, M. V., & **Downing, P. E.** (2007). The neural basis of visual body perception. *Nature Reviews Neuroscience, 8*(8), 636. https://doi.org/10.1038/nrn2195

Peralta, V., & **Cuesta, M. J.** (1999). Diagnostic significance of Schneider's first-rank symptoms in schizophrenia: Comparative study between schizophrenic and non-schizophrenic psychotic disorders. *The British Journal of Psychiatry, 174*(3), 243-248. https://doi.org/10.1192/bjp.174.3.243

Peralta, V., & **Cuesta, M. J.** (2007). A dimensional and categorical architecture for the classification of psychotic disorders. *World Psychiatry, 6*(2), 100. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2219908/

Perez, V. B., Ford, J. M., Roach, B. J., Loewy, R. L., Stuart, B. K., Vinogradov, S., & Mathalon, D. H. (2012). Auditory cortex responsiveness during talking and listening: early illness schizophrenia and patients at clinical high-risk for psychosis. *Schizophrenia bulletin, 38*(6), 1216-1224. https://doi.org/10.1093/schbul/sbr124

Pernet, C., Schyns, P. G., & Demonet, J. F. (2007). Specific, selective or preferential: comments on category specificity in neuroimaging. *Neuroimage, 35*(3), 991-997. https://doi.org/10.1016/j.neuroimage.2007.01.017

Pernet, C. R., McAleer, P., Latinus, M., Gorgolewski, K. J., Charest, I., Bestelmeyer, P. E., ... & Belin, P. (2015). The human voice areas: Spatial organization and inter-individual variability in temporal and extra-temporal cortices. *Neuroimage, 119*, 164-174. https://doi.org/10.1016/j.neuroimage.2015.06.050

Perona-Garcelán, S., García-Montes, J. M., Rodríguez-Testal, J. F., Ruiz-Veguilla, M., Benítez-Hernández, M. D. M., López-Jiménez, A. M., ... & Pérez-Álvarez, M. (2013). Relationship of absorption, depersonalisation, and self-focused attention in subjects with and without hallucination proneness. *Cognitive Neuropsychiatry, 18*(5), 422-436. https://doi.org/10.1080/13546805.2012.728133

Perrone-Bertolotti, M., Rapin, L., Lachaux, J. P., Baciu, M., & Loevenbruck, H. (2014). What is that little voice inside my head? Inner speech phenomenology, its role in cognitive performance, and its relation to self-monitoring. *Behavioural brain research, 261*, 220-239. https://doi.org/10.1016/j.bbr.2013.12.034

Petacchi, A., Laird, A. R., Fox, P. T., & Bower, J. M. (2005). Cerebellum and auditory function: An ALE meta-analysis of functional neuroimaging studies. *Human Brain Mapping, 25*(1), 118–128. https://doi.org/10.1002/hbm.20137

Peter, L. J., Schindler, S., Sander, C., Schmidt, S., Muehlan, H., McLaren, T., ... & Schomerus, G. (2021). Continuum beliefs and mental illness stigma: a systematic review and meta-analysis of correlation and intervention studies. *Psychological medicine*, 1-11. https://doi.org/10.1017/S0033291721000854

Petkov, C. I., & **Vuong, Q. C.** (2013). Neuronal Coding: The Value in Having an Average Voice. *Current Biology, 23*(12), R521-R523. https://doi.org/10.1016/j.cub.2013.04.077

Pettersson-Yeo, W., Allen, P., Benetti, S., McGuire, P., & Mechelli, A. (2011). Dysconnectivity in schizophrenia: where are we now?. *Neuroscience & Biobehavioral Reviews, 35*(5), 1110-1124. https://doi.org/10.1016/j.neubiorev.2010.11.004

Pesquita, A., Whitwell, R. L., & Enns, J. T. (2018). Predictive joint-action model: A hierarchical predictive approach to human cooperation. *Psychonomic bulletin & review, 25*(5), 1751-1769. https://doi.org/10.3758/s13423-017-1393-6

Pfordresher, P. Q., Mantell, J. T., Brown, S., Zivadinov, R., & Cox, J. L. (2014). Brain responses to altered auditory feedback during musical keyboard production: An fMRI study. *Brain Research, 1556*, 28–37. https://doi.org/10.1016/j.brainres.2014.02.004

Phillips, O. R., Nuechterlein, K. H., Clark, K. A., Hamilton, L. S., Asarnow, R. F., Hageman, N. S., ... & Narr, K. L. (2009). Fiber tractography reveals disruption of temporal lobe white matter tracts in schizophrenia. *Schizophrenia research, 107*(1), 30-38. https://doi.org/10.1016/j.schres.2008.10.019

Picard, H., Amado, I., Mouchet-Mages, S., Olié, J. P., & Krebs, M. O. (2008). The role of the cerebellum in schizophrenia: an update of clinical, cognitive, and functional evidences. *Schizophrenia bulletin, 34*(1), 155-172. https://doi.org/10.1093/schbul/sbm049

Picardi, A., Viroli, C., Tarsitani, L., Miglio, R., de Girolamo, G., Dell'Acqua, G., & Biondi, M. (2012). Heterogeneity and symptom structure of schizophrenia. *Psychiatry research, 198*(3), 386-394. https://doi.org/10.1016/j.psychres.2011.12.051

Picard, F., & **Friston, K.** (2014). Predictions, perception, and a sense of self. *Neurology, 83*(12), 1112-1118. http://doi.org/10.1212/WNL.0000000000000798

Pickering, M. J., & **Garrod, S.** (2007). Do people use language production to make predictions during comprehension?. *Trends in cognitive sciences, 11*(3), 105-110. https://doi.org/10.1016/j.tics.2006.12.002

Pilton, M., Varese, F., Berry, K., & Bucci, S. (2015). The relationship between dissociation and voices: a systematic literature review and meta-analysis. *Clinical Psychology Review, 40*, 138-155. https://doi.org/10.1016/j.cpr.2015.06.004

Pinheiro, A. P., Rezaii, N., Rauber, A., & Niznikiewicz, M. (2016). Is this my voice or yours? The role of emotion and acoustic quality in self-other voice discrimination in schizophrenia. *Cognitive neuropsychiatry, 21*(4), 335-353. https://doi.org/10.1080/13546805.2016.1208611

Pinheiro, A. P., Rezaii, N., Nestor, P. G., Rauber, A., Spencer, K. M., & Niznikiewicz, M. (2016b). Did you or I say pretty, rude or brief? An ERP study of the effects of speaker's identity on emotional word processing. *Brain and Language, 153*, 38-49. https://doi.org/10.1016/j.bandl.2015.12.003

Pinheiro, A. P., Rezaii, N., Rauber, A., Nestor, P. G., Spencer, K. M., & Niznikiewicz, M. (2017). Emotional self–other voice processing in schizophrenia and its relationship with hallucinations: ERP evidence. *Psychophysiology, 54*(9), 1252-1265. https://doi.org/10.1111/psyp.12880

Pinheiro, A. P., Schwartze, M., & Kotz, S. A. (2018). Voice-selective prediction alterations in nonclinical voice hearers. *Scientific Reports, 8*(1), 1-10. https://doi.org/10.1038/s41598-018-32614-9

Pinheiro, A. P., Farinha-Fernandes, A., Roberto, M. S., & Kotz, S. A. (2019). Self-voice perception and its relationship with hallucination predisposition. *Cognitive neuropsychiatry, 24*(4), 237-255. https://doi.org/10.1080/13546805.2019.1621159

Pinheiro, A. P., Schwartze, M., & Kotz, S. A. (2020a). Cerebellar circuitry and auditory verbal hallucinations: An integrative synthesis and perspective. *Neuroscience & Biobehavioral Reviews, 118*, 485-503. https://doi.org/10.1016/j.neubiorev.2020.08.004

Pinheiro, A. P., Schwartze, M., Amorim, M., Coentre, R., Levy, P., & Kotz, S. A. (2020b). Changes in motor preparation affect the sensory consequences of voice production in voice hearers. *Neuropsychologia, 146*, 107531. https://doi.org/10.1016/j.neuropsychologia.2020.107531

Pinheiro, A. P., Johnson, J. F., Amorim, M., Roberto, M., Schwartze, M., Kotz, S. A., & Shenton, M. E. (2021). The cerebellum links to positive symptoms of psychosis: A systematic review and meta-analysis. *Schizophrenia Bulletin Open, 2*(1), sgab039. https://doi.org/10.1093/schizbullopen/sgab039

Plitman, E., Nakajima, S., de la Fuente-Sandoval, C., Gerretsen, P., Chakravarty, M. M., Kobylianskii, J., ... & Graff-Guerrero, A. (2014). Glutamate-mediated excitotoxicity in schizophrenia: a review. *European Neuropsychopharmacology, 24*(10), 1591-1605. https://doi.org/10.1016/j.euroneuro.2014.07.015

Plato. (2000). The Republic (T. Griffith, Trans. & G.R.F. Ferrari, Ed.). Cambridge University Press. https://books.google.ca/books?id=aPwPjVIxbGQC&dq=plato%27s+republic&source=gbs_navlinks_s (Original work published ca. 370 B.C.E)

Plewes, D. B., & **Kucharczyk, W.** (2012). Physics of MRI: a primer. *Journal of magnetic resonance imaging, 35*(5), 1038-1054. https://doi.org/10.1002/jmri.23642

Popa, L. S., & **Ebner, T. J.** (2019). Cerebellum, predictions and errors. *Frontiers in cellular neuroscience, 12*, 524. https://doi.org/10.3389/fncel.2018.00524

Porrill, J., Dean, P., & Stone, J. V. (2004). Recurrent cerebellar architecture solves the motor-error problem. Proceedings of the Royal Society of London. *Series B: Biological Sciences, 271*(1541), 789-796. https://doi.org/10.1098/rspb.2003.2658

Posey, T. B., & **Losch, M. E.** (1983). Auditory hallucinations of hearing voices in 375 normal subjects. *Imagination, cognition and personality, 3*(2), 99-113. https://doi.org/10.2190/74V5-HNXN-JEY5-DG7W

Pouget, A., & **Snyder, L. H.** (2000). Computational approaches to sensorimotor transformations. *Nature neuroscience, 3*(11), 1192-1198. https://doi.org/10.1038/81469

Power, J. D., Cohen, A. L., Nelson, S. M., Wig, G. S., Barnes, K. A., Church, J. A., ... & Petersen, S. E. (2011). Functional network organization of the human brain. *Neuron, 72*(4), 665-678. https://doi.org/10.1016/j.neuron.2011.09.006

Powers III, A. R., Kelley, M., & Corlett, P. R. (2016). Hallucinations as top-down effects on perception. Biological Psychiatry: *Cognitive Neuroscience and Neuroimaging, 1*(5), 393-400. https://doi.org/10.1016/j.bpsc.2016.04.003

Prevosto, V., Graf, W., & Ugolini, G. (2009). Cerebellar inputs to intraparietal cortex areas LIP and MIP: Functional frameworks for adaptive control of eye movements, reaching, and arm/eye/head movement coordination. *Cerebral Cortex, 20*(1), 214–228. https://doi.org/10.1093/cercor/bhp091

Prinz, W. (1997). Perception and action planning. *European journal of cognitive psychology, 9*(2), 129-154. https://doi.org/10.1080/713752551

Psomiades, M., Fonteneau, C., Mondino, M., Luck, D., Haesebaert, F., Suaud-Chagny, M. F., & Brunelin, J. (2016). Integrity of the arcuate fasciculus in patients with schizophrenia with auditory verbal hallucinations: A DTI-tractography study. *NeuroImage: Clinical, 12*, 970-975. https://doi.org/10.1016/j.nicl.2016.04.013

Pulvermüller, F., Huss, M., Kherif, F., Moscoso del Prado Martin, F., Hauk, O., & Shtyrov, Y. (2006). Motor cortex maps articulatory features of speech sounds. *Proceedings of the National Academy of Sciences, 103*(20), 7865-7870. https://doi.org/10.1073/pnas.0509989103

Purcell, D. W., & **Munhall, K. G.** (2006). Compensation following real-time manipulation of formants in isolated vowels. *The Journal of the Acoustical Society of America, 119*(4), 2288–2297. https://doi.org/10.1121/1.2173514

Quesque, F., & **Brass, M.** (2019). The role of the temporoparietal junction in self-other distinction. *Brain topography, 32*(6), 943-955. https://doi.org/10.1007/s10548-019-00737-5

Raichle, M. E., & **Mintun, M. A.** (2006). Brain work and brain imaging. *Annu. Rev. Neurosci., 29*, 449-476. https://doi-org.mu.idm.oclc.org/10.1146/annurev.neuro.29.051605.112819

Rao, R. P., & **Ballard, D. H.** (1999). Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. *Nature neuroscience, 2*(1), 79-87. https://doi.org/10.1038/4580

Ramnani, N. (2006). The primate cortico-cerebellar system: anatomy and function. *Nature reviews neuroscience, 7*(7), 511-522. https://doi.org/10.1038/nrn1953

Ramnani, N. (2012). Frontal lobe and posterior parietal contributions to the cortico-cerebellar system. *The Cerebellum, 11*(2), 366–383. https://doi.org/10.1007/s12311-011-0272-3

Rauschecker, J. P. (1998). Cortical processing of complex sounds. *Current opinion in neurobiology, 8*(4), 516-521. https://doi.org/10.1016/S0959-4388(98)80040-8

Rauschecker, J. P., & **Scott, S. K.** (2009). Maps and streams in the auditory cortex: nonhuman primates illuminate human speech processing. *Nature Neuroscience, 12*(6), 718-724. https://doi.org/10.1038/nn.2331

Rauschecker, J. P. (2011). An expanded role for the dorsal auditory pathway in sensorimotor control and integration. *Hearing research, 271*(1-2), 16-25. https://doi.org/10.1016/j.heares.2010.09.001

Rauschecker, J. P. (2018). Where, When, and How: Are they all sensorimotor? Towards a unified view of the dorsal pathway in vision and audition. *Cortex, 98*, 262-268. https://doi.org/10.1016/j.cortex.2017.10.020

Raymond, J. L., Lisberger, S. G., & Mauk, M. D. (1996). The cerebellum: A neuronal learning machine? *Science, 272*(5265), 1126–1131. https://doi.org/10.1126/science.272.5265.1126

Reininghaus, U., Kempton, M. J., Valmaggia, L., Craig, T. K., Garety, P., Onyejiaka, A., ... & Morgan, C. (2016). Stress sensitivity, aberrant salience, and threat anticipation in early psychosis: an experience sampling study. *Schizophrenia bulletin, 42*(3), 712-722. https://doi.org/10.1093/schbul/sbv190

Riecker, A., Ackermann, H., Wildgruber, D., Dogil, G., & Grodd, W. (2000). Opposite hemispheric lateralization effects during speaking and singing at motor cortex, insula and cerebellum. *Neuroreport, 11*(9), 1997-2000. https://doi.org/10.1097/00001756-200006260-00038

Riederer, P., Lange, K. W., Kornhuber, J., & Danielczyk, W. (1992). Glutamatergic-dopaminergic balance in the brain. Its importance in motor disorders and schizophrenia. *Arzneimittel-Forschung, 42*(2A), 265-268. http://doi.org/10.5283/epub.25433

Rijntjes, M., Buechel, C., Kiebel, S., & Weiller, C. (1999). Multiple somatotopic representations in the human cerebellum. *Neuroreport, 10(*17), 3653-3658. http://doi.org/10.1097/00001756-199911260-00035

Rizzolatti, G., & **Craighero, L.** (2004). The mirror-neuron system. *Annu. Rev. Neurosci., 27*, 169-192. https://doi.org/10.1146/annurev.neuro.27.070203.144230

Robinson, J. D., Wagner, N. F., & Northoff, G. (2016). Is the sense of agency in schizophrenia influenced by resting-state variation in self-referential regions of the brain? *Schizophrenia bulletin, 42*(2), 270-276. https://doi.org/10.1093/schbul/sbv102

Roes, M. M., Yin, J., Taylor, L., Metzak, P. D., Lavigne, K. M., Chinchani, A., ... & Woodward, T. S. (2020). Hallucination-Specific structure-function associations in schizophrenia. *Psychiatry Research: Neuroimaging, 305*, 111171. https://doi.org/10.1016/j.pscychresns.2020.111171

Rolland, B., Jardri, R., Amad, A., Thomas, P., Cottencin, O., & Bordet, R. (2014). Pharmacology of hallucinations: several mechanisms for one single symptom?. *BioMed Research International*, 2014. https://doi.org/10.1155/2014/307106

Rollins, C. P., Garrison, J. R., Simons, J. S., Rowe, J. B., O'Callaghan, C., Murray, G. K., & Suckling, J. (2019). Meta-analytic evidence for the plurality of mechanisms in transdiagnostic structural MRI studies of hallucination status. *EClinicalMedicine, 8*, 57-71. https://doi.org/10.1016/j.eclinm.2019.01.012

Rosa, C., Lassonde, M., Pinard, C., Keenan, J. P., & Belin, P. (2008). Investigations of Hemispheric Specialization of Self-Voice Recognition. *Brain and Cognition, 68*(2), 204-214. https://doi.org/10.1016/j.bandc.2008.04.007

Rosburg, T., Boutros, N. N., & Ford, J. M. (2008). Reduced auditory evoked potential component N100 in schizophrenia—a critical review. *Psychiatry research, 161*(3), 259-274. https://doi.org/10.1016/j.psychres.2008.03.017

Rossell, S. L., & **Boundy, C. L.** (2005). Are auditory–verbal hallucinations associated with auditory affective processing deficits?. *Schizophrenia research, 78*(1), 95-106. https://doi.org/10.1016/j.schres.2005.06.002

Rosenblatt, F. (1958). The perceptron: A probabilistic model for information storage and organization in the brain. *Psychological Review, 65*(6), 386–408. https://doi.org/10.1037/h0042519

Roswandowitz, C., Mathias, S. R., Hintz, F., Kreitewolf, J., Schelinski, S., & von Kriegstein, K. (2014). Two cases of selective developmental voice-recognition impairments. *Current Biology, 24*(19), 2348-2353. https://doi.org/10.1016/j.cub.2014.08.048

Rotarska-Jagiela, A., Oertel-Knoechel, V., DeMartino, F., van de Ven, V., Formisano, E., Roebroeck, A., ... & Linden, D. E. (2009). Anatomical brain connectivity and positive symptoms of schizophrenia: a diffusion tensor imaging study. *Psychiatry Research: Neuroimaging, 174*(1), 9-16. https://doi.org/10.1016/j.pscychresns.2009.03.002

Rousseeuw, P. J., & **Hubert, M.** (2011). Robust statistics for outlier detection. *Wiley interdisciplinary reviews: Data mining and knowledge discovery, 1*(1), 73-79. https://doi.org/10.1002/widm.2

Rubinov, M., & **Bullmore, E.** (2022). Schizophrenia and abnormal brain network hubs. *Dialogues in clinical neuroscience*. https://doi.org/10.31887/DCNS.2013.15.3/mrubinov

Rubio, J. M., Sanjuán, J., Flórez-Salamanca, L., & Cuesta, M. J. (2012). Examining the course of hallucinatory experiences in children and adolescents: a systematic review. *Schizophrenia research, 138*(2-3), 248-254. https://doi.org/10.1016/j.schres.2012.03.012

Ruby, P., & **Decety, J.** (2001). Effect of subjective perspective taking during simulation of action: A PET investigation of agency. *Nature Neuroscience, 4*(5), 546–550. https://doi.org/10.1038/87510

Rumelhart, D. E., Hinton, G. E., & Williams, R. J. (1986a). Learning internal representations by back-propagating errors. *Nature, 323*(99), 533-536. https://doi.org/10.1038/323533a0

Rumelhart, D. E., Hinton, G. E., & Williams, R. J. (1986b). Learning internal representations by error propagation. In D. E. Rumelhart & J. L. McClelland (Eds.), Parallel distributed processing: Explorations in the microstructure of cognition. Vol. 1: Foundations (pp. 318–364). Cambridge, MA: Bradford Books/MIT Press. https://apps.dtic.mil/sti/citations/ADA164453

Sakai, N., Masuda, S., Shimotomai, T., & Mori, K. (2009). Brain activation in adults who stutter under delayed auditory feedback: An fMRI study. *International Journal of Speech-Language Pathology, 11*(1), 2–11. https://doi.org/10.1080/17549500802588161

Saltzman, E., Löfqvist, A., Kay, B., Kinsella-Shaw, J., & Rubin, P. (1998). Dynamics of intergestural timing: A perturbation study of lip-larynx coordination. *Experimental Brain Research, 123*(4), 412–424. https://doi.org/10.1007/s002210050586

Samuel, A. G. (2011). Speech perception. *Annual review of psychology, 62*, 49-72. https://doi.org/10.1146/annurev.psych.121208.131643

Sang, L., Qin, W., Liu, Y., Han, W., Zhang, Y., Jiang, T., & Yu, C. (2012). Resting-state functional connectivity of the vermal and hemispheric subregions of the cerebellum with both the cerebral cortical networks and subcortical structures. *NeuroImage, 61*(4), 1213–1225. https://doi.org/10.1016/j.neuroimage.2012.04.011

Sapara, A., Cooke, M. A., Williams, S. C., & Kumari, V. (2015). Is it me? Verbal self-monitoring neural network and clinical insight in schizophrenia. *Psychiatry Research: Neuroimaging, 234*(3), 328-335. https://doi.org/10.1016/j.pscychresns.2015.10.007

Schilling, K., Oberdick, J., Rossi, F., & Baader, S. L. (2008). Besides Purkinje cells and granule neurons: an appraisal of the cell biology of the interneurons of the cerebellar cortex. *Histochemistry and cell biology, 130*(4), 601-615. https://doi.org/10.1007/s00418-008-0483-y

Schirmer, A., & **Kotz, S. A.** (2006). Beyond the right hemisphere: brain mechanisms mediating vocal emotional processing. *Trends in cognitive sciences, 10*(1), 24-30. https://doi.org/10.1016/j.tics.2005.11.009

Schirmer, A. (2018). Is the voice an auditory face? An ALE meta-analysis comparing vocal and facial emotion processing. *Social Cognitive and Affective Neuroscience, 13*(1), 1-13. https://doi.org/10.1093/scan/nsx142

Schelinski, S., Borowiak, K., & von Kriegstein, K. (2016). Temporal voice areas exist in autism spectrum disorder but are dysfunctional for voice identity recognition. *Social Cognitive and Affective Neuroscience, 11*(11), 1812-1822. https://doi.org/10.1093/scan/nsw089

Schlerf, J. E., Verstynen, T. D., Ivry, R. B., & Spencer, R. M. (2010). Evidence of a novel somatopic map in the human neocerebellum during complex actions. *Journal of neurophysiology, 103*(6), 3330-3336. https://doi.org/10.1152/jn.01117.2009

Schmahmann, J. D. (2000). The role of the cerebellum in affect and psychosis. *Journal of Neurolinguistics, 13*(2-3), 189-214. https://doi.org/10.1016/S0911-6044(00)00011-7

Schmahmann, J. D., Smith, E. E., Eichler, F. S., & Filley, C. M. (2008). Cerebral white matter: neuroanatomy, clinical neurology, and neurobehavioral correlates. *Annals of the New York Academy of Sciences, 1142*, 266-309. http://doi.org/10.1196/annals.1444.017

Schmidt, A., Diwadkar, V. A., Smieskova, R., Harrisberger, F., Lang, U. E., McGuire, P., ... & Borgwardt, S. (2015). Approaching a network connectivity-driven classification of the psychosis continuum: a selective review and suggestions for future research. *Frontiers in human neuroscience, 8*, 1047. https://doi.org/10.3389/fnhum.2014.01047

Shmuelof, L., & **Krakauer, J. W.** (2011). Are we ready for a natural history of motor learning?. *Neuron, 72*(3), 469-476. https://doi.org/10.1016/j.neuron.2011.10.017

Schnell, K., Heekeren, K., Schnitker, R., Daumann, J., Weber, J., Heßelmann, V., … Gouzoulis-Mayfrank, E. (2007). An fMRI approach to particularize the frontoparietal network for visuomotor action monitoring: Detection of incongruence between test subjects' actions and resulting perceptions. *NeuroImage, 34*(1), 332–341. https://doi.org/10.1016/j.neuroimage.2006.08.027

Schwartz, J. L., Basirat, A., Ménard, L., & Sato, M. (2012). The Perception-for-Action-Control Theory (PACT): A perceptuo-motor theory of speech perception. *Journal of Neurolinguistics, 25*(5), 336-354. https://doi.org/10.1016/j.jneuroling.2009.12.004

Schwartz, T. L., Sachdeva, S., & Stahl, S. M. (2012). Glutamate neurocircuitry: theoretical underpinnings in schizophrenia. *Frontiers in pharmacology, 3*, 195. https://doi.org/10.3389/fphar.2012.00195

Schwartze, M., & **Kotz, S. A.** (2013). A dual-pathway neural architecture for specific temporal prediction. *Neuroscience & Biobehavioral Reviews, 37*(10), 2587–2596. https://doi.org/10.1016/j.neubiorev.2013.08.005

Schwartze, M., & **Kotz, S. A.** (2016). Contributions of cerebellar event-based temporal processing and preparatory function to speech perception. *Brain and language, 161*, 28-32. https://doi.org/10.1016/j.bandl.2015.08.005

Schweinberger, S. R., Herholz, A., & Sommer, W. (1997). Recognizing famous voices: Influence of stimulus duration and different types of retrieval cues. *Journal of Speech, Language, and Hearing Research, 40*(2), 453-463. https://doi.org/10.1044/jslhr.4002.453

Schweinberger, S. R., Walther, C., Zäske, R., & Kovács, G. (2011). Neural correlates of adaptation to voice identity. *British Journal of Psychology, 102*(4), 748-764. https://doi.org/10.1111/j.2044-8295.2011.02048.x

Schweinberger, S. R., Kawahara, H., Simpson, A. P., Skuk, V. G., & Zäske, R. (2014). Speaker perception. *Wiley Interdisciplinary Reviews: Cognitive Science, 5*(1), 15-25. https://doi.org/10.1002/wcs.1261

Scott, S. K., & **Johnsrude, I. S.** (2003). The neuroanatomical and functional organization of speech perception. *Trends in neurosciences, 26*(2), 100-107. https://doi.org/10.1016/S0166-2236(02)00037-1

Scott, S. K., McGettigan, C., & Eisner, F. (2009). A little more conversation, a little less action—candidate roles for the motor cortex in speech perception. *Nature Reviews Neuroscience, 10*(4), 295-302. https://doi.org/10.1038/nrn2603

Sebanz, N., Bekkering, H., & Knoblich, G. (2006). Joint action: bodies and minds moving together. *Trends in cognitive sciences, 10*(2), 70-76. https://doi.org/10.1016/j.tics.2005.12.009

Sebeok, T. A. (2001). Signs: An introduction to semiotics. University of Toronto Press. https://monoskop.org/images/0/07/Sebeok_Thomas_Signs_An_Introduction_to_Semiocs_2nd_ed_2001.pdf

Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., ... & Greicius, M. D. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *Journal of Neuroscience, 27*(9), 2349-2356. https://doi.org/10.1523/JNEUROSCI.5587-06.2007

Seeley, W. W. (2010). Anterior insula degeneration in frontotemporal dementia. *Brain Structure and Function, 214*(5-6), 465-475. https://doi.org/10.1007/s00429-010-0263-z

Seeley, W. W. (2019). The salience network: a neural system for perceiving and responding to homeostatic demands. *Journal of Neuroscience, 39*(50), 9878-9882. https://doi.org/10.1523/JNEUROSCI.1138-17.2019

Seidler, R. D., Noll, D. C., & Chintalapati, P. (2006). Bilateral basal ganglia activation associated with sensorimotor adaptation. *Experimental Brain Research, 175*(3), 544–555. https://doi.org/10.1007/s00221-006-0571-y

Seth, A. K., Suzuki, K., & Critchley, H. D. (2012). An interoceptive predictive coding model of conscious presence. *Frontiers in psychology, 2*, 395. https://doi.org/10.3389/fpsyg.2011.00395

Sengupta, B., Tozzi, A., Cooray, G. K., Douglas, P. K., & Friston, K. J. (2016). Towards a neuronal gauge theory. *PLoS Biology, 14*(3), e1002400. https://doi.org/10.1371/journal.pbio.1002400

Seok, J. H., Park, H. J., Chun, J. W., Lee, S. K., Cho, H. S., Kwon, J. S., & Kim, J. J. (2007). White matter abnormalities associated with auditory hallucinations in schizophrenia: a combined study of voxel-based analyses of diffusion tensor imaging and structural magnetic resonance imaging. *Psychiatry Research: Neuroimaging, 156*(2), 93-104. https://doi.org/10.1016/j.pscychresns.2007.02.002

Shafer, A. (2005). Meta-analysis of the brief psychiatric rating scale factor structure. *Psychological assessment, 17*(3), 324. https://doi.org/10.1037/1040-3590.17.3.324

Shafer, A., & **Dazzi, F.** (2019). Meta-analysis of the positive and Negative Syndrome Scale (PANSS) factor structure. *Journal of psychiatric research, 115*, 113-120. https://doi.org/10.1016/j.jpsychires.2019.05.008

Shaiman, S., & **Gracco, V. L.** (2002). Task-specific sensorimotor interactions in speech production. *Experimental Brain Research, 146*(4), 411–418. https://doi.org/10.1007/s00221-002-1195-5

Shea, T. L., Sergejew, A. A., Burnham, D., Jones, C., Rossell, S. L., Copolov, D. L., & Egan, G. F. (2007). Emotional prosodic processing in auditory hallucinations. *Schizophrenia research, 90*(1-3), 214-220. https://doi.org/10.1016/j.schres.2006.09.021

Shergill, S. S., Kanaan, R. A., Chitnis, X. A., O'Daly, O., Jones, D. K., Frangou, S., ... & McGuire, P. (2007). A diffusion tensor imaging study of fasciculi in schizophrenia. *American Journal of Psychiatry, 164*(3), 467-473. http://doi.org/10.1176/ajp.2007.164.3.467

Shimada, S., Hiraki, K., & Oda, I. (2005). The parietal role in the sense of self-ownership with temporal discrepancy between visual and proprioceptive feedbacks. *NeuroImage, 24*(4), 1225–1232. https://doi.org/10.1016/j.neuroimage.2004.10.039

Shipp, S. (2016). Neural elements for predictive coding. *Frontiers in psychology, 7*, 1792. https://doi.org/10.3389/fpsyg.2016.01792

Siddi, S., Ochoa, S., Laroi, F., Cella, M., Raballo, A., Saldivia, S., ... & Preti, A. (2019). A cross-national investigation of hallucination-like experiences in 10 countries: the E-CLECTIC study. *Schizophrenia Bulletin, 45*(Supplement_1), S43-S55. https://doi.org/10.1093/schbul/sby156

Siegel, G. M., Schork, E. J., Pick, H. L., & Garber, S. R. (1982). Parameters of auditory feedback. *Journal of Speech, Language, and Hearing Research, 25*(3), 473–475. https://doi.org/10.1044/jshr.2503.473

Silverstein, B. H., Bressler, S. L., & Diwadkar, V. A. (2016). Inferring the dysconnection syndrome in schizophrenia: Interpretational considerations on methods for the network analyses of fMRI data. Frontiers in psychiatry, 7, 132. https://doi.org/10.3389/fpsyt.2016.00132

Siman-Tov, T., Granot, R. Y., Shany, O., Singer, N., Hendler, T., & Gordon, C. R. (2019). Is there a prediction network? Meta-analytic evidence for a cortical-subcortical network likely subserving prediction. *Neuroscience & Biobehavioral Reviews, 105*, 262-275. https://doi.org/10.1016/j.neubiorev.2019.08.012

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

Singmann, H., Bolker, B., Westfall, J., & Aust, F. (2015). afex: Analysis of factorial experiments. R package version 0.13–145. Retrieved from https://cran.r-project.org/web/packages/afex/index.html

Sirigu, A., Duhamel, J. R., Cohen, L., Pillon, B., Dubois, B., & Agid, Y. (1996). The mental representation of hand movements after parietal cortex damage. *Science, 273*(5281), 1564–1568. https://doi.org/10.1126/science.273.5281.1564

Sitek, K. R., Mathalon, D. H., Roach, B. J., Houde, J. F., Niziolek, C. A., & Ford, J. M. (2013). Auditory cortex processes variation in our own speech. *PloS one, 8*(12), e82925. https://doi.org/10.1371/journal.pone.0082925

Skudlarski, P., Jagannathan, K., Anderson, K., Stevens, M. C., Calhoun, V. D., Skudlarska, B. A., & Pearlson, G. (2010). Brain connectivity is not only lower but different in schizophrenia: a combined anatomical and functional approach. *Biological psychiatry, 68*(1), 61-69. https://doi.org/10.1016/j.biopsych.2010.03.035

Smith, D. A., Mar, C. M., & Turoff, B. K. (1998). The structure of schizophrenic symptoms: a meta-analytic confirmatory factor analysis. *Schizophrenia research, 31*(1), 57-70. https://doi.org/10.1016/S0920-9964(98)00009-7

Smith, M. A., Brandt, J., & Shadmehr, R. (2000). Motor disorder in Huntington's disease begins as adysfunction in error feedback control. *Nature, 403*(6769), 544. https://doi.org/10.1038/35000576

Smith, S. M. (2002). Fast robust automated brain extraction. *Human brain mapping, 17*(3), 143-155. https://doi.org/10.1002/hbm.10062

Smith, S. M., Jenkinson, M., Woolrich, M. W., Beckmann, C. F., Behrens, T. E., Johansen-Berg, H., ... & Niazy, R. K. (2004). Advances in functional and structural MR image analysis and implementation as FSL. *Neuroimage, 23*, S208-S219. https://doi.org/10.1016/j.neuroimage.2004.07.051

Smith, M. A., & **Shadmehr, R.** (2005). Intact ability to learn internal models of arm dynamics in Huntington's disease but not cerebellar degeneration. *Journal of Neurophysiology, 93*(5), 2809–2821. https://doi.org/10.1152/jn.00943.2004

Smith, D. R., & **Patterson, R. D.** (2005). The interaction of glottal-pulse rate and vocal-tract length in judgements of speaker size, sex, and age. *The Journal of the Acoustical Society of America, 118*(5), 3177-3186. https://doi.org/10.1121/1.2047107

Smith, S. M., Jenkinson, M., Johansen-Berg, H., Rueckert, D., Nichols, T. E., Mackay, C. E., ... & Behrens, T. E. (2006). Tract-based spatial statistics: voxelwise analysis of multi-subject diffusion data. *Neuroimage, 31*(4), 1487-1505. https://doi.org/10.1016/j.neuroimage.2006.02.024

Smith, D. R., Walters, T. C., & Patterson, R. D. (2007). Discrimination of speaker sex and size when glottal-pulse rate and vocal-tract length are controlled. *The Journal of the Acoustical Society of America, 122*(6), 3628-3639. https://doi.org/10.1121/1.2799507

Smith, S. M., Jenkinson, M., Woolrich, M. W., Beckmann, C. F., Behrens, T. E., Johansen-Berg, H., ... & Matthews, P. M. (2004). Advances in functional and structural MR image analysis and implementation as FSL. *Neuroimage, 23*, S208-S219. https://doi.org/10.1016/j.neuroimage.2004.07.051

Smith, R., Badcock, P., & Friston, K. J. (2021). Recent advances in the application of predictive coding and active inference models within clinical neuroscience. *Psychiatry and Clinical Neurosciences, 75*(1), 3-13. https://doi.org/10.1111/pcn.13138

Soares, J. M., Marques, P., Alves, V., & Sousa, N. (2013). A hitchhiker's guide to diffusion tensor imaging. *Frontiers in neuroscience, 7*, 31. https://doi.org/10.3389/fnins.2013.00031

Sommer, I. E., Diederen, K. M., Blom, J. D., Willems, A., Kushan, L., Slotema, K., ... & Kahn, R. S. (2008). Auditory verbal hallucinations predominantly activate the right inferior frontal area. *Brain, 131*(12), 3169-3177. https://doi.org/10.1093/brain/awn251

Sommer, I. E., Daalman, K., Rietkerk, T., Diederen, K. M., Bakker, S., Wijkstra, J., & Boks, M. P. (2010). Healthy individuals with auditory verbal hallucinations; who are they? Psychiatric assessments of a selected sample of 103 subjects. *Schizophrenia bulletin, 36*(3), 633-641. https://doi.org/10.1093/schbul/sbn130

Sommer, I. E., Kleijer, H., & Hugdahl, K. (2018). Toward personalized treatment of hallucinations. *Current opinion in psychiatry, 31*(3), 237-245. https://doi.org/10.1097/YCO.0000000000000416

Song, Y., Lukasiewicz, T., Xu, Z., & Bogacz, R. (2020). Can the brain do backpropagation?—exact implementation of backpropagation in predictive coding networks. *Advances in neural information processing systems, 33*, 22566. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC7610561/

Spaniel, F., Tintera, J., Rydlo, J., Ibrahim, I., Kasparek, T., Horacek, J., … Mikolas, P. (2015). Altered neural correlate of the self-agency experience in first-episode schizophrenia-spectrum patients: An fMRI study. *Schizophrenia Bulletin, 42*(4), 916–925. https://doi.org/10.1093/schbul/sbv188

Sridharan, D., Levitin, D. J., & Menon, V. (2008). A critical role for the right fronto-insular cortex in switching between central-executive and default-mode networks. *Proceedings of the National Academy of Sciences, 105*(34), 12569-12574. https://doi.org/10.1073/pnas.0800005105

Stahl, S. M. (2007). Beyond the dopamine hypothesis to the NMDA glutamate receptor hypofunction hypothesis of schizophrenia. *CNS spectrums, 12*(4), 265-268. https://doi.org/10.1017/S1092852900021015

Stahl, S. M. (2018). Beyond the dopamine hypothesis of schizophrenia to three neural networks of psychosis: dopamine, serotonin, and glutamate. *CNS spectrums, 23*(3), 187-191. https://doi.org/10.1017/S1092852918001013

Stephan, K. E., Baldeweg, T., & Friston, K. J. (2006). Synaptic plasticity and dysconnection in schizophrenia. *Biological psychiatry, 59*(10), 929-939. https://doi.org/10.1016/j.biopsych.2005.10.005

Stephan, K. E., Friston, K. J., & Frith, C. D. (2009). Dysconnection in schizophrenia: from abnormal synaptic plasticity to failures of self-monitoring. *Schizophrenia bulletin, 35*(3), 509-527. https://doi.org/10.1093/schbul/sbn176

Stephane, M., Thuras, P., Nasrallah, H., & Georgopoulos, A. P. (2003). The internal structure of the phenomenology of auditory verbal hallucinations. *Schizophrenia research, 61*(2-3), 185-193. https://doi.org/10.1016/S0920-9964(03)00013-6

Stevenage, S. V., & **Neil, G. J.** (2014). Hearing faces and seeing voices: The integration and interaction of face and voice processing. *Psychologica Belgica, 54*(3), 266-281. http://dx.doi.org/10.5334/pb.ar

Stejskal, E. O., & **Tanner, J. E.** (1965). Spin diffusion measurements: spin echoes in the presence of a time-dependent field gradient. *The journal of chemical physics*, 42(1), 288-292. https://doi.org/10.1063/1.1695690

Steinmann, S., Leicht, G., & Mulert, C. (2014). Interhemispheric auditory connectivity: structure and function related to auditory verbal hallucinations. *Frontiers in human neuroscience, 8*, 55. https://doi.org/10.3389/fnhum.2014.00055

Steinmann, S., Leicht, G., & Mulert, C. (2019). The interhemispheric miscommunication theory of auditory verbal hallucinations in schizophrenia. *International Journal of Psychophysiology, 145*, 83-90. http://doi.org/10.1016/j.ijpsycho.2019.02.002

Sterzer, P., Adams, R. A., Fletcher, P., Frith, C., Lawrie, S. M., Muckli, L., ... & Corlett, P. R. (2018). The predictive coding account of psychosis. *Biological psychiatry, 84*(9), 634-643. https://doi.org/10.1016/j.biopsych.2018.05.015

Stip, E., & **Letourneau, G.** (2009). Psychotic symptoms as a continuum between normality and pathology. *The Canadian Journal of Psychiatry, 54*(3), 140-151. https://doi.org/10.1177/070674370905400302

Stirling, J., Barkus, E., & Lewis, S. (2007). Hallucination proneness, schizotypy and meta-cognition. *Behaviour Research and Therapy, 45*(6), 1401-1408. https://doi.org/10.1016/j.brat.2006.06.003

Stokes, R. C., Venezia, J. H., & Hickok, G. (2019). The motor system's [modest] contribution to speech perception. *Psychonomic bulletin & review, 26*(4), 1354-1366. https://doi.org/10.3758/s13423-019-01580-2

Stoodley, C. J., & **Schmahmann, J. D.** (2010). Evidence for topographic organization in the cerebellum of motor control versus cognitive and affective processing. *Cortex, 46*(7), 831-844. https://doi.org/10.1016/j.cortex.2009.11.008

Stoodley, C. J., & **Schmahmann, J. D.** (2018). Functional topography of the human cerebellum. *Handbook of clinical neurology, 154*, 59-70. https://doi.org/10.1016/B978-0-444-63956-1.00004-7

Strauss, J. S. (1969). Hallucinations and delusions as points on continua function: Rating scale evidence. *Archives of general psychiatry, 21*(5), 581-586. http://doi.org/10.1001/archpsyc.1969.01740230069010

Stuart, A., Kalinowski, J., Rastatter, M. P., & Lynch, K. (2002). Effect of delayed auditory feedback on normal speakers at two speech rates. *The Journal of the Acoustical Society of America, 111*(5), 2237–2241. https://doi.org/10.1121/1.1466868

Stys, P. K., & **Lipton, S. A.** (2007). White matter NMDA receptors: an unexpected new therapeutic target?. *Trends in Pharmacological Sciences, 28*(11), 561-566. https://doi.org/10.1016/j.tips.2007.10.003

Suárez, L. E., Markello, R. D., Betzel, R. F., & Misic, B. (2020). Linking structure and function in macroscale brain networks. *Trends in Cognitive Sciences, 24(*4), 302-315. https://doi.org/10.1016/j.tics.2020.01.008

Suárez-Pozos, E., Thomason, E. J., & Fuss, B. (2020). Glutamate Transporters: expression and function in oligodendrocytes. *Neurochemical research, 45*(3), 551-560. http://doi.org/10.1007/s11064-018-02708-x

Subramaniam, M., Abdin, E., Vaingankar, J., Picco, L., Shahwan, S., Jeyagurunathan, A., ... & Chong, S. A. (2016). Prevalence of psychotic symptoms among older adults in an Asian population. *International Psychogeriatrics, 28*(7), 1211-1220. https://doi.org/10.1017/S1041610216000399

Subramanian, D., Alers, A., & Sommer, M. A. (2019). Corollary discharge for action and cognition. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 4*(9), 782-790. https://doi.org/10.1016/j.bpsc.2019.05.010

Sun, H., Lui, S., Yao, L., Deng, W., Xiao, Y., Zhang, W., ... & Gong, Q. (2015). Two patterns of white matter abnormalities in medication-naive patients with first-episode schizophrenia revealed by diffusion tensor imaging and cluster analysis. JAMA psychiatry, 72(7), 678-686. https://doi.org/10.1001/jamapsychiatry.2015.0505

Swiney, L., & **Sousa, P.** (2014). A new comparator account of auditory verbal hallucinations: how motor prediction can plausibly contribute to the sense of agency for inner speech. *Frontiers in Human Neuroscience, 8*, 675. https://doi.org/10.3389/fnhum.2014.00675

Szeszko, P. R., Robinson, D. G., Ashtari, M., Vogel, J., Betensky, J., Sevy, S., ... & Bilder, R. M. (2008). Clinical and neuropsychological correlates of white matter abnormalities in recent onset schizophrenia. *Neuropsychopharmacology, 33*(5), 976-984. https://doi.org/10.1038/sj.npp.1301480

Takahashi, N., Sakurai, T., Davis, K. L., & Buxbaum, J. D. (2011). Linking oligodendrocyte and myelin dysfunction to neurocircuitry abnormalities in schizophrenia. *Progress in neurobiology, 93*(1), 13-24. https://doi.org/10.1016/j.pneurobio.2010.09.004

Takaso, H., Eisner, F., Wise, R. J., & Scott, S. K. (2010). The effect of delayed auditory feedback on activity in the temporal lobe while speaking: A positron emission tomography study. *Journal of Speech, Language, and Hearing Research, 53*(2), 226–236. https://doi.org/10.1044/1092-4388(2009/09-0009)

Talairarch, J., & **Tournoux, P.** (1988). Co-planar stereotaxic atlas of the human brain: 3-dimensional proportional system–An approach to cerebral imaging. New York, NY: Theme Medical.

Tanaka, H., Ishikawa, T., Lee, J., & Kakei, S. (2020). The cerebro-cerebellum as a locus of forward model: a review. *Frontiers in systems neuroscience, 14*, 19. https://doi.org/10.3389/fnsys.2020.00019

Tamminga, C. A. (1998). Schizophrenia and glutamatergic transmission. *Critical Reviews™ in Neurobiology, 12*(1-2). http://doi.org/10.1615/CritRevNeurobiol.v12.i1-2.20

Tamnes, C. K., & **Agartz, I.** (2016). White matter microstructure in early-onset schizophrenia: a systematic review of diffusion tensor imaging studies. *Journal of the American Academy of Child & Adolescent Psychiatry, 55*(4), 269-279. https://doi.org/10.1016/j.jaac.2016.01.004

Teufel, C., Fletcher, P. C., & Davis, G. (2010). Seeing other minds: attributed mental states influence perception. *Trends in cognitive sciences, 14*(8), 376-382. https://doi.org/10.1016/j.tics.2010.05.005

Thach, W. T. (1998). A role for the cerebellum in learning movement coordination. *Neurobiology of Learning and Memory, 70*(1–2), 177–188. https://doi.org/10.1006/nlme.1998.3846

Thakkar, K. N., Mathalon, D. H., & Ford, J. M. (2021). Reconciling competing mechanisms posited to underlie auditory verbal hallucinations. *Philosophical Transactions of the Royal Society B, 376*(1817), 20190702. https://doi.org/10.1098/rstb.2019.0702

Therrien, A. S., & **Bastian, A. J.** (2015). Cerebellar damage impairs internal predictions for sensory and motor function. *Current opinion in neurobiology, 33*, 127-133. https://doi.org/10.1016/j.conb.2015.03.013

Thompson, R., Hallas, L., Moseley, P., & Alderson-Day, B. (2021). Cognitive and phenomenological characteristics of hallucination-proneness across the lifespan. Cognitive Neuropsychiatry, 26(1), 18-34. https://doi.org/10.1080/13546805.2020.1850435

Tian, X., & **Poeppel, D.** (2012). Mental imagery of speech: linking motor and perceptual systems through internal simulation and estimation. *Frontiers in human neuroscience, 6*, 314. https://doi.org/10.3389/fnhum.2012.00314

Tien, A. Y. (1991). Distribution of hallucinations in the population. *Social psychiatry and psychiatric epidemiology, 26*(6), 287-292. https://doi.org/10.1007/BF00789221

Tison, R., & **Poirier, P.** (2021a). Communication as Socially Extended Active Inference: An Ecological Approach to Communicative Behavior. *Ecological Psychology*, 1-39. https://doi.org/10.1080/10407413.2021.1965480

Tison, R., & **Poirier, P.** (2021b). Active inference and cooperative communication: an ecological alternative to the alignment view. *Frontiers in Psychology, 12*, 708780. https://doi.org/10.3389/fpsyg.2021.708780

Toh, W. L., Thomas, N., Hollander, Y., & Rossell, S. L. (2020a). On the phenomenology of auditory verbal hallucinations in affective and non-affective psychosis. *Psychiatry Research, 290*, 113147. https://doi.org/10.1016/j.psychres.2020.113147

Toh, W. L., Thomas, N., Robertson, M., & Rossell, S. L. (2020b). Characteristics of non-clinical hallucinations: A mixed-methods analysis of auditory, visual, tactile and olfactory hallucinations in a primary voice-hearing cohort. *Psychiatry Research, 289*, 112987. https://doi.org/10.1016/j.psychres.2020.112987

Tomasello, M., Melis, A. P., Tennie, C., Wyman, E., & Herrmann, E. (2012). Two key steps in the evolution of human cooperation: The interdependence hypothesis. *Current anthropology, 53*(6), 673-692. https://doi.org/10.1086/668207

Tourville, J. A., Reilly, K. J., & Guenther, F. H. (2008). Neural mechanisms underlying auditory feedback control of speech. *NeuroImage, 39*(3), 1429–1443. https://doi.org/10.1016/j.neuroimage.2007.09.054

Tourville, J. A., & **Guenther, F. H.** (2011). The DIVA model: A neural theory of speech acquisition and production. *Language and Cognitive Processes, 26*(7), 952-981. https://doi.org/10.1080/01690960903498424

Toyomura, A., Koyama, S., Miyamaoto, T., Terao, A., Omori, T., Murohashi, H., & Kuriki, S. (2007). Neural correlates of auditory feedback control in human. *Neuroscience, 146*(2), 499–503. https://doi.org/10.1016/j.neuroscience.2007.02.023

Tracy, D. K., & **Shergill, S. S.** (2006). Imaging auditory hallucinations in schizophrenia. *Acta Neuropsychiatrica, 18*(2), 71-78. https://doi.org/10.1111/j.1601-5215.2006.00129.x

Tseng, H. H., Chen, S. H., Liu, C. M., Howes, O., Huang, Y. L., Hsieh, M. H., ... & Hwu, H. G. (2013). Facial and prosodic emotion recognition deficits associate with specific clusters of psychotic symptoms in schizophrenia. *PLoS One, 8*(6), e66571. https://doi.org/10.1371/journal.pone.0066571

Tunik, E., Saleh, S., & Adamovich, S. V. (2013). Visuomotor discordance during visually-guided hand movement in virtual reality modulates sensorimotor cortical activity in healthy and hemiparetic subjects. *IEEE Transactions on Neural Systems and Rehabilitation Engineering, 21*(2), 198–207. https://doi.org/10.1109/TNSRE.2013.2238250

Turkeltaub, P. E., Eickhoff, S. B., Laird, A. R., Fox, M., Wiener, M., & Fox, P. (2012). Minimizing within-experiment and within-group effects in activation likelihood estimation meta-analyses. *Human Brain Mapping, 33* (1), 1–13. https://doi.org/10.1002/hbm.21186

Uddin, L. Q. (2015). Salience processing and insular cortical function and dysfunction. *Nature reviews neuroscience, 16*(1), 55-61. https://doi.org/10.1038/nrn3857

Uhlhaas, P. J. (2013). Dysconnectivity, large-scale networks and neuronal dynamics in schizophrenia. *Current opinion in neurobiology, 23*(2), 283-290. https://doi.org/10.1016/j.conb.2012.11.004

Uno, Y., & **Coyle, J. T.** (2019). Glutamate hypothesis in schizophrenia. *Psychiatry and clinical neurosciences, 73*(5), 204-215. https://doi.org/10.1111/pcn.12823

Upthegrove, R., Broome, M. R., Caldwell, K., Ives, J., Oyebode, F., & Wood, S. J. (2016). Understanding auditory verbal hallucinations: a systematic review of current evidence. *Acta Psychiatrica Scandinavica, 133*(5), 352-367. https://doi.org/10.1111/acps.12531

Urbain, G., Barasuol, V., Semini, C., & Dambre, J. (2020, May). Stance control inspired by cerebellum stabilizes reflex-based locomotion on HyQ robot. In 2020 IEEE International Conference on Robotics and Automation (ICRA) (pp. 6127-6133). IEEE. http://doi.org/10.1109/ICRA40945.2020.9196523

Vallortigara, G. (2021). The Efference Copy Signal as a Key Mechanism for Consciousness. *Frontiers in Systems Neuroscience, 145*. https://doi.org/10.3389/fnsys.2021.765646

Valton, V., Romaniuk, L., Steele, J. D., Lawrie, S., & Seriès, P. (2017). Comprehensive review: Computational modelling of schizophrenia. *Neuroscience & Biobehavioral Reviews, 83*, 631-646. https://doi.org/10.1016/j.neubiorev.2017.08.022

van Berkum, J. J., Van den Brink, D., Tesink, C. M., Kos, M., & Hagoort, P. (2008). The neural integration of speaker and message. *Journal of Cognitive Neuroscience, 20*(4), 580-591. https://doi.org/10.1162/jocn.2008.20054

Van Den Heuvel, M. P., & **Pol, H. E. H.** (2010). Exploring the brain network: a review on resting-state fMRI functional connectivity. *European neuropsychopharmacology, 20*(8), 519-534. https://doi.org/10.1016/j.euroneuro.2010.03.008

Vandervert, L. (2020). The prominent role of the cerebellum in the social learning of the phonological loop in working memory: How language was adaptively built from cerebellar inner speech required during stone-tool making. *AIMS neuroscience, 7*(3), 333. http://doi.org/10.3934/Neuroscience.2020020

van der Weiden, A., Prikken, M., & van Haren, N. E. (2015). Self–other integration and distinction in schizophrenia: A theoretical analysis and a review of the evidence. *Neuroscience & Biobehavioral Reviews, 57*, 220-237. https://doi.org/10.1016/j.neubiorev.2015.09.004

van der Zwaag, W., Kusters, R., Magill, A., Gruetter, R., Martuzzi, R., Blanke, O., & Marques, J. P. (2013). Digit somatotopy in the human cerebellum: a 7 T fMRI study. *Neuroimage, 67*, 354-362. https://doi.org/10.1016/j.neuroimage.2012.11.041

Van Elk, M., & **Aleman, A.** (2017). Brain mechanisms in religion and spirituality: An integrative predictive processing framework. Neuroscience & Biobehavioral Reviews, 73, 359-378. https://doi.org/10.1016/j.neubiorev.2016.12.031

Van Lancker, D. R., & **Canter, G. J.** (1982). Impairment of voice and face recognition in patients with hemispheric damage. *Brain and Cognition, 1*(2), 185-195. https://doi.org/10.1016/0278-2626(82)90016-1

Van Lancker, D. R., & **Kreiman, J.** (1987). Voice discrimination and recognition are separate abilities. *Neuropsychologia, 25*(5), 829-834. https://doi.org/10.1016/0028-3932(87)90120-5

Van Lancker, D. R., Cummings, J. L., Kreiman, J., & Dobkin, B. H. (1988). Phonagnosia: A dissociation between familiar and unfamiliar voices. *Cortex, 24*(2), 195-209. https://doi.org/10.1016/S0010-9452(88)80029-7

van Lutterveld, R., Sommer, I. E., & Ford, J. M. (2011). The neurophysiology of auditory hallucinations–a historical and contemporary review. *Frontiers in Psychiatry, 2*, 28. https://doi.org/10.3389/fpsyt.2011.00028

van Lutterveld, R., Diederen, K. M., Koops, S., Begemann, M. J., & Sommer, I. E. (2013). The influence of stimulus detection on activation patterns during auditory hallucinations. *Schizophrenia research, 145*(1-3), 27-32. https://doi.org/10.1016/j.schres.2013.01.004

van Os, J., Hanssen, M., Bijl, R. V., & Ravelli, A. (2000). Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophrenia research, 45*(1-2), 11-20. https://doi.org/10.1016/S0920-9964(99)00224-8

van Os, J. (2003). Is there a continuum of psychotic experiences in the general population? *Epidemiology and Psychiatric Sciences, 12*(4), 242-252. https://doi.org/10.1017/S1121189X00003067

van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. J. P. M. (2009). A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychological medicine, 39*(2), 179. http://doi.org/doi:10.1017/S0033291708003814

Varese, F., Barkus, E., & Bentall, R. P. (2011). Dissociative and metacognitive factors in hallucination-proneness when controlling for comorbid symptoms. *Cognitive neuropsychiatry, 16*(3), 193-217. https://doi.org/10.1080/13546805.2010.495244

Varese, F., Barkus, E., & Bentall, R. P. (2012). Dissociation mediates the relationship between childhood trauma and hallucination-proneness. *Psychological medicine, 42(5)*, 1025-1036. https://doi.org/10.1017/S0033291711001826

Vasil, J., Badcock, P. B., Constant, A., Friston, K., & Ramstead, M. J. (2020). A world unto itself: Human communication as active inference. *Frontiers in psychology, 11*, 417. https://doi.org/10.3389/fpsyg.2020.00417

Ventura, M. I., Nagarajan, S. S., & Houde, J. F. (2009). Speech target modulates speaking induced suppression in auditory cortex. *BMC Neuroscience, 10*(1), 58. https://doi.org/10.1186/1471-2202-10-58

Vercammen, A., & **Aleman, A.** (2010). Semantic expectations can induce false perceptions in hallucination-prone individuals. *Schizophrenia Bulletin, 36*(1), 151-156. https://doi.org/10.1093/schbul/sbn063

Verdoux, H., & **van Os, J.** (2002). Psychotic symptoms in non-clinical populations and the continuum of psychosis. *Schizophrenia research, 54*(1-2), 59-65. https://doi.org/10.1016/S0920-9964(01)00352-8

Versmissen, D., Janssen, I., Johns, L., McGuire, P. H. I. L. I. P., Drukker, M., Campo, J. A., ... & Krabbendam, L. (2007). Verbal self-monitoring in psychosis: a non-replication. *Psychological Medicine, 37*(4), 569-576. https://doi.org/10.1017/S0033291706009780

Vesper, C., Butterfill, S., Knoblich, G., & Sebanz, N. (2010). A minimal architecture for joint action. *Neural Networks, 23*(8-9), 998-1003. https://doi.org/10.1016/j.neunet.2010.06.002

Viard, A., Eustache, F., & Segobin, S. (2021). History of magnetic resonance imaging: a trip down memory lane. *Neuroscience, 474*, 3-13. https://doi.org/10.1016/j.neuroscience.2021.06.038

Vilas, M. G., Auksztulewicz, R., & Melloni, L. (2021). Active inference as a computational framework for consciousness. *Review of Philosophy and Psychology*, 1-20. https://doi.org/10.1007/s13164-021-00579-w

Venezia, J. H., & **Hickok, G.** (2009). Mirror neurons, the motor system and language: from the motor theory to embodied cognition and beyond. *Language and Linguistics Compass, 3*(6), 1403-1416. https://doi-org.mu.idm.oclc.org/10.1111/j.1749-818X.2009.00169.x

Voineskos, A. N., Foussias, G., Lerch, J., Felsky, D., Remington, G., Rajji, T. K., ... & Mulsant, B. H. (2013). Neuroimaging evidence for the deficit subtype of schizophrenia. *JAMA psychiatry, 70*(5), 472-480. http://doi.org/10.1001/jamapsychiatry.2013.786

Von Bartheld, C. S., Bahney, J., & Herculano-Houzel, S. (2016). The search for true numbers of neurons and glial cells in the human brain: A review of 150 years of cell counting. *Journal of Comparative Neurology, 524*(18), 3865-3895. https://doi-org.mu.idm.oclc.org/10.1002/cne.24040

Von Der Heide, R. J., Skipper, L. M., Klobusicky, E., & Olson, I. R. (2013). Dissecting the uncinate fasciculus: disorders, controversies, and a hypothesis. *Brain, 136*(6), 1692-1707. https://doi.org/10.1093/brain/awt094

von Holst, E., & **Mittelstaedt, H.** (1950). Das reafferenzprinzip. *Naturwissenschaften, 37*(20), 464–476. https://doi.org/10.1007/BF00622503

von Kriegstein, K., Eger, E., Kleinschmidt, A., & Giraud, A. L. (2003). Modulation of neural responses to speech by directing attention to voices or verbal content. *Cognitive Brain Research, 17*(1), 48-55. https://doi.org/10.1016/S0926-6410(03)00079-X

von Kriegstein, K., & **Giraud, A. L.** (2004). Distinct functional substrates along the right superior temporal sulcus for the processing of voices. *Neuroimage, 22*(2), 948-955. https://doi.org/10.1016/j.neuroimage.2004.02.020

von Kriegstein, K., Kleinschmidt, A., Sterzer, P., & Giraud, A. L. (2005). Interaction of face and voice areas during speaker recognition. *Journal of cognitive neuroscience, 17*(3), 367-376. https://doi.org/10.1162/0898929053279577

Wallwork, R. S., Fortgang, R., Hashimoto, R., Weinberger, D. R., & Dickinson, D. (2012). Searching for a consensus five-factor model of the Positive and Negative Syndrome Scale for schizophrenia. *Schizophrenia research, 137*(1-3), 246-250. https://doi.org/10.1016/j.schres.2012.01.031

Walterfang, M., Wood, S. J., Velakoulis, D., & Pantelis, C. (2006). Neuropathological, neurogenetic and neuroimaging evidence for white matter pathology in schizophrenia. *Neuroscience & Biobehavioral Reviews, 30*(7), 918-948. https://doi.org/10.1016/j.neubiorev.2006.02.001

Walterfang, M., Velakoulis, D., Whitford, T. J., & Pantelis, C. (2011). Understanding aberrant white matter development in schizophrenia: an avenue for therapy?. *Expert review of neurotherapeutics, 11*(7), 971-987. https://doi.org/10.1586/ern.11.76

Wang, J., Mathalon, D. H., Roach, B. J., Reilly, J., Keedy, S. K., Sweeney, J. A., & Ford, J. M. (2014). Action planning and predictive coding when speaking. *Neuroimage, 91*, 91-98. https://doi.org/10.1016/j.neuroimage.2014.01.003

Warren, J. D., Jennings, A. R., & Griffiths, T. D. (2005a). Analysis of the spectral envelope of sounds by the human brain. *Neuroimage, 24*(4), 1052-1057. https://doi.org/10.1016/j.neuroimage.2004.10.031

Warren, J. E., Wise, R. J., & Warren, J. D. (2005b). Sounds do-able: auditory–motor transformations and the posterior temporal plane. *Trends in neurosciences, 28*(12), 636-643. https://doi.org/10.1016/j.tins.2005.09.010

Warren, J. D., Scott, S. K., Price, C. J., & Griffiths, T. D. (2006). Human brain mechanisms for the early analysis of voices. *Neuroimage, 31*(3), 1389-1397. https://doi.org/10.1016/j.neuroimage.2006.01.034

Waters, F., Badcock, J. C., & Maybery, M. T. (2003). Revision of the factor structure of the Launay–Slade Hallucination Scale (LSHS-R). *Personality and Individual Differences, 35*(6), 1351-1357. https://doi.org/10.1016/S0191-8869(02)00354-9

Waters, F., Allen, P., Aleman, A., Fernyhough, C., Woodward, T. S., Badcock, J. C., ... & Larøi, F. (2012a). Auditory hallucinations in schizophrenia and nonschizophrenia populations: a review and integrated model of cognitive mechanisms. *Schizophrenia bulletin, 38*(4), 683-693. https://doi.org/10.1093/schbul/sbs045

Waters, F., Woodward, T., Allen, P., Aleman, A., & Sommer, I. (2012b). Self-recognition deficits in schizophrenia patients with auditory hallucinations: a meta-analysis of the literature. *Schizophrenia bulletin, 38*(4), 741-750. https://doi.org/10.1093/schbul/sbq144

Waters, F., Collerton, D., Ffytche, D. H., Jardri, R., Pins, D., Dudley, R., ... & Larøi, F. (2014). Visual hallucinations in the psychosis spectrum and comparative information from neurodegenerative disorders and eye disease. *Schizophrenia bulletin, 40*(Suppl_4), S233-S245. https://doi.org/10.1093/schbul/sbu036

Waters, F., & **Fernyhough, C.** (2017). Hallucinations: a systematic review of points of similarity and difference across diagnostic classes. *Schizophrenia bulletin, 43*(1), 32-43. https://doi.org/10.1093/schbul/sbw132

Watkins, K., Patel, N., Davis, S., & Howell, P. (2005). Brain activity during altered auditory feedback: an FMRI study in healthy adolescents. *Neuroimage, 26*(Supp 1), 304. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2801072/

Watson, R., Latinus, M., Charest, I., Crabbe, F., & Belin, P. (2014a). People-selectivity, audiovisual integration and heteromodality in the superior temporal sulcus. *cortex, 50*, 125-136. https://doi.org/10.1016/j.cortex.2013.07.011

Watson, R., Latinus, M., Noguchi, T., Garrod, O., Crabbe, F., & Belin, P. (2014b). Crossmodal adaptation in right posterior superior temporal sulcus during face–voice emotional integration. *Journal of Neuroscience, 34*(20), 6813-6821. https://doi.org/10.1523/JNEUROSCI.4478-13.2014

Watson, T. C., Becker, N., Apps, R., & Jones, M. W. (2014c). Back to front: Cerebellar connections and interactions with the prefrontal cortex. *Frontiers in Systems Neuroscience, 8*(4), 1–4. https://doi.org/10.3389/fnsys.2014.00004

Weiss, A. P., & **Heckers, S.** (1999). Neuroimaging of hallucinations: a review of the literature. *Psychiatry Research: Neuroimaging, 92*(2-3), 61-74. https://doi.org/10.1016/S0925-4927(99)00041-4

Welniarz, Q., Worbe, Y., & Gallea, C. (2021). The forward model: a unifying theory for the role of the cerebellum in motor control and sense of agency. *Frontiers in Systems Neuroscience, 15*. http://doi.org/10.3389/fnsys.2021.644059

Williams, D. (2021). Imaginative constraints and generative models. *Australasian Journal of Philosophy, 99*(1), 68-82. https://doi.org/10.1080/00048402.2020.1719523

Winklewski, P. J., Sabisz, A., Naumczyk, P., Jodzio, K., Szurowska, E., & Szarmach, A. (2018). Understanding the physiopathology behind axial and radial diffusivity changes—what do we know?. *Frontiers in neurology, 9*, 92. https://doi.org/10.3389/fneur.2018.00092

Winton-Brown, T. T., Fusar-Poli, P., Ungless, M. A., & Howes, O. D. (2014). Dopaminergic basis of salience dysregulation in psychosis. *Trends in neurosciences, 37*(2), 85-94. https://doi.org/10.1016/j.tins.2013.11.003

Wilson, S. M., Saygin, A. P., Sereno, M. I., & Iacoboni, M. (2004). Listening to speech activates motor areas involved in speech production. Nature neuroscience, 7(7), 701-702. https://doi.org/10.1038/nn1263

Wise, S. P., & **Strick, P. L.** (1984). Anatomical and physiological organization of the non-primary motor cortex. *Trends in Neurosciences, 7*(11), 442–446. https://doi.org/10.1016/S0166-2236(84)80150-2

Wheeler-Kingshott, C. A., & **Cercignani, M.** (2009). About "axial" and "radial" diffusivities. *Magnetic Resonance in Medicine: An Official Journal of the International Society for Magnetic Resonance in Medicine, 61*(5), 1255-1260. https://doi.org/10.1002/mrm.21965

Whitford, T. J., Kubicki, M., Schneiderman, J. S., O'Donnell, L. J., King, R., Alvarado, J. L., ... & Shenton, M. E. (2010). Corpus callosum abnormalities and their association with psychotic symptoms in patients with schizophrenia. *Biological psychiatry, 68*(1), 70-77. https://doi.org/10.1016/j.biopsych.2010.03.025

Whitford, T. J., Mathalon, D. H., Shenton, M. E., Roach, B. J., Bammer, R., Adcock, R. A., ... & Schneiderman, J. S. (2011). Electrophysiological and diffusion tensor imaging evidence of delayed corollary discharges in patients with schizophrenia. *Psychological Medicine, 41*(5), 959-969. https://doi.org/10.1017/S0033291710001376

Whitford, T. J., Ford, J. M., Mathalon, D. H., Kubicki, M., & Shenton, M. E. (2012). Schizophrenia, myelination, and delayed corollary discharges: a hypothesis. *Schizophrenia bulletin, 38*(3), 486-494. https://doi.org/10.1093/schbul/sbq105

Whitford, T. J. (2019). Speaking-induced suppression of the auditory cortex in humans and its relevance to schizophrenia. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 4*(9), 791-804. https://doi.org/10.1016/j.bpsc.2019.05.011

Whittington, J. C., & **Bogacz, R.** (2019). Theories of error back-propagation in the brain. *Trends in cognitive sciences, 23*(3), 235-250. https://doi.org/10.1016/j.tics.2018.12.005

Wickham, H. (2017). tidyverse: Easily Install and Load the 'Tidyverse'. R package version 1.2. 1. Retrieved from https://cran.r-project.org/web/packages/tidyverse/index.html

Wiestler, T., McGonigle, D. J., & Diedrichsen, J. (2011). Integration of sensory and motor representations of single fingers in the human cerebellum. *Journal of neurophysiology, 105*(6), 3042-3053. https://doi.org/10.1152/jn.00106.2011

Wiethoff, S., Wildgruber, D., Kreifelts, B., Becker, H., Herbert, C., Grodd, W., & Ethofer, T. (2008). Cerebral processing of emotional prosody—influence of acoustic parameters and arousal. *Neuroimage, 39*(2), 885-893. https://doi.org/10.1016/j.neuroimage.2007.09.028

Wigand, M., Kubicki, M., Clemm von Hohenberg, C., Leicht, G., Karch, S., Eckbo, R., ... & Mulert, C. (2015). Auditory verbal hallucinations and the interhemispheric auditory pathway in chronic schizophrenia. *The World Journal of Biological Psychiatry, 16*(1), 31-44. https://doi.org/10.3109/15622975.2014.948063

Williamson, P. C., & **Allman, J. M.** (2012). A framework for interpreting functional networks in schizophrenia. Frontiers in human neuroscience, 6, 184. https://doi.org/10.3389/fnhum.2012.00184

Wilson, M., & **Knoblich, G.** (2005). The case for motor involvement in perceiving conspecifics. *Psychological bulletin, 131*(3), 460. http://doi.org/10.1037/0033-2909.131.3.460.

Wilson, S. M. (2009). Speech perception when the motor system is compromised. *Trends in cognitive sciences, 13*(8), 329. http://doi.org/10.1016/j.tics.2009.06.001

Witter, L., & **De Zeeuw, C. I.** (2015). Regional functionality of the cerebellum. *Current Opinion in Neurobiology, 33*, 150–155. https://doi.org/10.1016/j.conb.2015.03.017

Wolpert, D. M., Ghahramani, Z., & Jordan, M. I. (1995). An internal model for sensorimotor integration. *Science, 269*(5232), 1880-1882. http://doi.org/10.1126/science.7569931

Wolpert, D. M. (1997). Computational approaches to motor control. *Trends in Cognitive Sciences, 1*(6), 209–216. https://doi.org/10.1016/S1364-6613(97)01070-X

Wolpert, D. M., Miall, R. C., & Kawato, M. (1998). Internal models in the cerebellum. *Trends in Cognitive Sciences, 2*(9), 338–347. https://doi.org/10.1016/S1364-6613(98)01221-2

Wolpert, D. M., & **Kawato, M.** (1998). Multiple paired forward and inverse models for motor control. *Neural networks, 11*(7-8), 1317-1329. https://doi.org/10.1016/S0893-6080(98)00066-5

Wolpert, D. M., & **Flanagan, J. R.** (2001). Motor prediction. *Current biology, 11*(18), R729-R732. https://doi.org/10.1016/S0960-9822(01)00432-8

Wolpert, D. M., Ghahramani, Z., & Flanagan, J. R. (2001). Perspectives and problems in motor learning. *Trends in cognitive sciences, 5*(11), 487-494. https://doi.org/10.1016/S1364-6613(00)01773-3

Wolpert, D. M., Doya, K., & Kawato, M. (2003). A unifying computational framework for motor control and social interaction. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 358*(1431), 593-602. https://doi.org/10.1098/rstb.2002.1238

Wolpert, D. M., Diedrichsen, J., & Flanagan, J. R. (2011). Principles of sensorimotor learning. *Nature reviews neuroscience, 12*(12), 739-751. https://doi.org/10.1038/nrn3112

Woodruff, P. W., Wright, I. C., Bullmore, E. T., Brammer, M., Howard, R. J., Williams, S. C., ... & Murray, R. M. (1997). Auditory hallucinations and the temporal cortical response to speech in schizophrenia: a functional magnetic resonance imaging study. *American Journal of Psychiatry, 154*(12), 1676-1682. https://doi.org/10.1176/ajp.154.12.1676

Woods, A., Jones, N., Alderson-Day, B., Callard, F., & Fernyhough, C. (2015). Experiences of hearing voices: analysis of a novel phenomenological survey. *The Lancet Psychiatry, 2*(4), 323-331. https://doi.org/10.1016/S2215-0366(15)00006-1

Wotruba, D., Michels, L., Buechler, R., Metzler, S., Theodoridou, A., Gerstenberg, M., ... & Heekeren, K. (2014). Aberrant coupling within and across the default mode, task-positive, and salience network in subjects at risk for psychosis. *Schizophrenia bulletin, 40*(5), 1095-1104. https://doi.org/10.1093/schbul/sbt161

Wu, Z. M., Chen, M. L., Wu, X. H., & Li, L. (2014). Interaction between auditory and motor systems in speech perception. *Neuroscience Bulletin, 30*(3), 490-496. https://doi.org/10.1007/s12264-013-1428-6

Xu, Y., Larson, C. R., Bauer, J. J., & Hain, T. C. (2004). Compensation for pitch-shifted auditory feedback during the production of mandarin tone sequences. *The Journal of the Acoustical Society of America, 116*(2), 1168–1178. https://doi.org/10.1121/1.1763952

Xi, Y. B., Guo, F., Li, H., Chang, X., Sun, J. B., Zhu, Y. Q., ... & Yin, H. (2016). The structural connectivity pathology of first-episode schizophrenia based on the cardinal symptom of auditory verbal hallucinations. *Psychiatry Research: Neuroimaging, 257*, 25-30. https://doi.org/10.1016/j.pscychresns.2016.09.011

Xu, M., Homae, F., Hashimoto, R. I., & Hagiwara, H. (2013). Acoustic cues for the recognition of self-voice and other-voice. *Frontiers in Psychology, 4*, 735. https://doi.org/10.3389/fpsyg.2013.00735

Xue, G., Aron, A. R., & Poldrack, R. A. (2008). Common neural substrates for inhibition of spoken and manual responses. *Cerebral Cortex, 18*(8), 1923-1932. https://doi.org/10.1093/cercor/bhm220

Yamazaki, T., & **Lennon, W.** (2019). Revisiting a theory of cerebellar cortex. *Neuroscience research, 148*, 1-8. https://doi.org/10.1016/j.neures.2019.03.001

Yang, F., Fang, X., Tang, W., Hui, L., Chen, Y., Zhang, C., & Tian, X. (2019). Effects and potential mechanisms of transcranial direct current stimulation (tDCS) on auditory hallucinations: A meta-analysis. Psychiatry research, 273, 343-349. https://doi.org/10.1016/j.psychres.2019.01.059

Yang, G. R., & **Wang, X. J.** (2020). Artificial neural networks for neuroscientists: A primer. *Neuron, 107*(6), 1048-1070. https://doi.org/10.1016/j.neuron.2020.09.005

Yates, K., Lång, U., Peters, E. M., Wigman, J. T., McNicholas, F., Cannon, M., ... & Kelleher, I. (2021). Hallucinations in the general population across the adult lifespan: prevalence and psychopathologic significance. *The British Journal of Psychiatry, 219*(6), 652-658. https://doi.org/10.1192/bjp.2021.100

Yomogida, Y., Sugiura, M., Sassa, Y., Wakusawa, K., Sekiguchi, A., Fukushima, A., … Kawashima, R. (2010). The neural basis of agency: An fMRI study. *NeuroImage, 50*(1), 198–207. https://doi.org/10.1016/j.neuroimage.2009.12.054

Young, I. R., Szeverenyi, N. M., Du, J., & Bydder, G. M. (2020). Pulse sequences as tissue property filters (TP-filters): a way of understanding the signal, contrast and weighting of magnetic resonance images. *Quantitative Imaging in Medicine and Surgery, 10*(5), 1080. http://doi.org/10.21037/qims.2020.04.07

Yovel, G., & **Belin, P.** (2013). A unified coding strategy for processing faces and voices. *Trends in cognitive sciences, 17*(6), 263-271. https://doi.org/10.1016/j.tics.2013.04.004

Zaitsev, M., Maclaren, J., & Herbst, M. (2015). Motion artifacts in MRI: A complex problem with many partial solutions. *Journal of Magnetic Resonance Imaging, 42*(4), 887-901. https://doi.org/10.1002/jmri.24850

Zang, Y., & **De Schutter, E.** (2019). Climbing fibers provide graded error signals in cerebellar learning. *Frontiers in Systems Neuroscience, 46*. https://doi.org/10.3389/fnsys.2019.00046

Zapparoli, L., Paulesu, E., Mariano, M., Ravani, A., & Sacheli, L. M. (2022). The sense of agency in joint actions: a theory-driven meta-analysis. *Cortex*. https://doi.org/10.1016/j.cortex.2022.01.002

Zarate, J. M., & **Zatorre, R. J.** (2005). Neural substrates governing audiovocal integration for vocal pitch regulation in singing. *Annals of the New York Academy of Sciences, 1060*, 404-408. https://doi.org/10.1196/annals.1360.058

Zarate, J. M., Wood, S., & Zatorre, R. J. (2010). Neural networks involved in voluntary and involuntary vocal pitch regulation in experienced singers. *Neuropsychologia, 48*(2), 607–618. https://doi.org/10.1016/j.neuropsychologia.2009.10.025

Zarate, J. M., & **Zatorre, R. J.** (2008). Experience-dependent neural substrates involved in vocal pitch regulation during singing. *NeuroImage, 40(4)*, 1871–1887. https://doi.org/10.1016/j.neuroimage.2008.01.026

Zatorre, R. J., Belin, P., & Penhune, V. B. (2002). Structure and function of auditory cortex: music and speech. *Trends in cognitive sciences, 6*(1), 37-46. https://doi.org/10.1016/S1364-6613(00)01816-7

Zatorre, R. J., **& Gandour, J. T.** (2008). Neural specializations for speech and pitch: moving beyond the dichotomies. *Philosophical Transactions of the Royal Society B: Biological Sciences, 363*(1493), 1087-1104. http://doi.org/10.1098/rstb.2007.2161

Zhang, X. Y., Fan, F. M., Chen, D. C., Tan, Y. L., Tan, S. P., Hu, K., ... & Soares, J. C. (2016). Extensive white matter abnormalities and clinical symptoms in drug-naive patients with first-episode schizophrenia: a voxel-based diffusion tensor imaging study. *The Journal of clinical psychiatry, 77*(2), 205-211. http://doi.org/10.4088/JCP.14m09374

Zhang, X., Gao, J., Zhu, F., Wang, W., Fan, Y., Ma, Q., ... & Yang, J. (2018). Reduced white matter connectivity associated with auditory verbal hallucinations in first episode and chronic schizophrenia: a diffusion tensor imaging study. *Psychiatry Research: Neuroimaging, 273*, 63-70. https://doi.org/10.1016/j.pscychresns.2018.01.002

Zhang, Y., Ding, Y., Huang, J., Zhou, W., Ling, Z., Hong, B., & Wang, X. (2021). Hierarchical cortical networks of "voice patches" for processing voices in human brain. *Proceedings of the National Academy of Sciences, 118*(52), e2113887118. https://doi.org/10.1073/pnas.2113887118

Zheng, Z. Z., Munhall, K. G., & Johnsrude, I. S. (2010). Functional overlap between regions involved in speech perception and in monitoring one's own voice during speech production. *Journal of Cognitive Neuroscience, 22*(8), 1770–1781. https://doi.org/10.1162/jocn.2009.21324

Zheng, Z. Z., MacDonald, E. N., Munhall, K. G., & Johnsrude, I. S. (2011). Perceiving a stranger's voice as being one's own: A 'rubber voice' illusion?. *PloS One, 6*(4). https://doi.org/10.1371/journal.pone.0018655

Zheng, Z. Z., Vicente-Grabovetsky, A., MacDonald, E. N., Munhall, K. G., Cusack, R., & Johnsrude, I. S. (2013). Multivoxel patterns reveal functionally differentiated networks underlying auditory feedback processing of speech. *Journal of Neuroscience, 33*(10), 4339–4348. https://doi.org/10.1523/JNEUROSCI.6319-11.2013

Zhuo, C., Jiang, D., Liu, C., Lin, X., Li, J., Chen, G., ... & Zhu, J. (2019). Understanding auditory verbal hallucinations in healthy individuals and individuals with psychiatric disorders. *Psychiatry research, 274*, 213-219. https://doi.org/10.1016/j.psychres.2019.02.040

Zmigrod, L., Garrison, J. R., Carr, J., & Simons, J. S. (2016). The neural mechanisms of hallucinations: a quantitative meta-analysis of neuroimaging studies. *Neuroscience & Biobehavioral Reviews, 69*, 113-123. https://doi.org/10.1016/j.neubiorev.2016.05.037